# The One Where Leo Goes to the Hospital

A Case Report of Pyothorax in the Canine

**Presented by:** 

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### Introduction

Pyothorax, also referred to as thoracic empyema, is a potentially fatal disease process involving a septic pleural effusion of the thoracic cavity. The pleural space, made up of the parietal and visceral pleura, normally contains a small amount of fluid to lubricate the pleura, allowing for passive ventilation without friction. Pathology due to several disease processes leads to an abnormal fluid accumulation in the pleural space, referred to as pleural effusion.

While the etiology is often undetermined, the current thoughts on the source of infection include penetrating plant material or other foreign object that migrates from the upper respiratory tract and through the lung parenchyma or through the thoracic wall into the pleural space, ultimately causing an inflammatory reaction within the pleura. This disease process can affect any dog, but medium to large breed hunting dogs tend to be overrepresented. (1,2,7)

Pyothorax is an uncommonly diagnosed condition in dogs that is characterized by a slowly progressive respiratory disease that manifests itself the form of weight loss, marked exercise intolerance, and increased respiratory effort. (1) A presumptive diagnosis is made based on patient signalment, history and clinical signs; definitive diagnosis requires thoracic imaging to demonstrate the presence of pleural fluid and cytologic examination of the fluid. Many patients with pyothorax present on an emergency basis in acute respiratory distress due to pleural effusion and can decompensate quickly. Thus, it is imperative to appropriately diagnose and initiate aggressive therapy in a timely manner. Both medical and surgical treatment options are available, but surgical exploration of the thoracic cavity to identify and remove a nidus combined with thoracostomy tube drainage and long-term antimicrobial therapy has been associated with a better long-term outcome. (1,4,6) This report will outline a common presentation of canine

pyothorax and illustrate the possibility of a positive outcome if diagnosed appropriately and treated promptly.

## **History and Presentation**

Leo is a 7-year-old, neutered male, Labrador Retriever that was presented on emergency to the Animal Emergency and Referral Center (AERC) in Flowood, MS, the night of July 8, 2017 following an acute episode of coughing and increased respiratory effort that ultimately led to what the owners referred to as vomiting. At that time, his owner reported a 2-3-week history of a dry cough, lethargy, and exercise intolerance that was most obvious when he began to refuse climbing stairs, not wanting to go outside as much, and when he needed assistance getting into the car. On physical examination, Leo was bright, alert, and responsive but dyspneic with an increased resting respiratory rate. He had a rectal temperature of 104.5°F, a heart rate of 104 beats per minute and he was panting. Oxygen saturation declined from 99% to 93% within 4 hours of arrival. Thoracic radiographs revealed findings consistent with marked lung consolidation and pleural effusion. Two separate attempts to evacuate the thoracic fluid overnight were unsuccessful. An intravenous cephalic catheter was placed, and Leo was started on an isotonic fluid infusion (Normosol-R) at 70 mL/hour. A dose of methadone (0.1 mg/kg IV q6h) was administered for analgesia, a dose of maropitant (1 mg/kg IV q24h) was given as an antiemetic, and he was started on ampicillin/sulbactam (30 mg/kg IV q8h) to initiate broad spectrum antibiotic coverage. The following morning, Leo was referred to the Mississippi State University Animal Health Center for further diagnostics and treatment for pyothorax. Upon presentation to the MSU-CVM Emergency Service, Leo was stable, alert and responsive, and was panting with abdominal effort at rest. There was no evidence of external wounds or lesions. He was still febrile at 104.1°F. Cardiac auscultation was unsuccessful due to muffled

bronchovesicular sounds appreciated in all lung fields, but a femoral pulse rate was interpreted to be 96 beats per minute. Injected sclera could be appreciated in both eyes. Mucus membranes were pink, moist, and capillary refill time was less than 2 seconds, indicating adequate capillary perfusion. Oxygen saturation was 94% on arrival, and three sequential blood pressures had an average mean arterial pressure of 142 mmHg. All other findings of his physical exam were within normal limits. During triage, Leo was supplemented with flow-by oxygen and a chest tube was successfully placed to allow approximately 100-120 ml of a red, serosanguinous fluid to be evacuated from the pleural space. After stabilization, Leo was placed in an oxygen cage for monitoring where he was continued on isotonic fluids at a maintenance rate of 68 ml/hr and intravenous ampicillin/sulbactam (30 mg/kg IV q8hr). Analgesia was maintained with hydromorphone (0.1 mg/kg IV q6hr). Chest tube care (including drainage, quantification of pleural fluid) was indicated every 4 hours and approximately 250 mL of pleural fluid was evacuated overnight. Leo was maintained on this protocol until the following morning when he was transferred to the MSU-CVM Small Animal Internal Medicine Service for further evaluation.

## **Diagnostic Approach/Considerations**

A diagnosis of pyothorax is made based on identification of a septic pleural effusion by means of thoracic imaging, cytological analysis of pleural effusion, hematology and serology, histology, and bacterial culture. A neutrophilic leukocytosis with or without the presence of a left shift is the most common abnormality seen on hematology in patients with thoracic empyema. However, its absence does not rule out the possibility of pyothorax, as decompensating patients in sepsis can present with a neutropenia, along with a normocytic/normochromic anemia of chronic disease. (3) In a normal, healthy patient, the pleura should not be visible on thoracic radiography.

Soft tissue and fluid have the same radiographic opacity and are more radiopaque (grey) than air, which appears radiolucent (black) on radiographs. Classic findings of fluid accumulation in the pleura can be appreciated in pyothorax patients; these findings include widened interlobar fissures and consolidated lung lobes that are retracted from the thoracic wall and border effacement of the cardiac silhouette. Computed tomography (CT) scans involve many radiographs taken in cross-sectional slices to provide a detailed anatomic analysis for evaluating bony lesions and soft tissue abnormalities that may not be appreciated on digital radiographs, especially when an intravenous contrast dye is utilized. Thus, CT is a valuable tool when attempting to image the mediastinum and the surrounding structures in a way that cannot be achieved by digital radiography. (3) Thoracic fluid analysis to determine the type of effusion (transudate versus exudate) is essential for diagnoses. Ideally, samples should be collected prior to starting antimicrobial therapy and sent off for cytological examination as well as culture and sensitivity so that treatment is specific for the pathogens present. (1)

In Leo, a CBC and chemistry profile revealed a marked leukocytosis with left shift, and mild mastocytosis (consistent with acute inflammatory disease), along with decreased BUN and a moderate to severe hypoalbuminemia (indicating a negative acute phase response by the liver). Thoracic radiographs taken prior to thoracocentesis revealed classic findings associated with pleural effusion (border effacement of the cardiac silhouette, pulmonary atelectasis with retraction of the lung lobes from the thoracic wall, pleural fissure lines, and an alveolar pulmonary pattern).

Evaluation of thoracic fluid to determine the type of effusion (transudate versus exudate) is essential for diagnosis. (6) Because of the two unsuccessful attempts at thoracocentesis at AERC, Leo was started on ampicillin/sulbactam before a sample could be analyzed. A sample of thoracic fluid was obtained shortly after presenting to MSU-CVM Emergency service and was diagnosed as an exudate, possibly malignant. A pathologist's report failed to identify an etiologic agent but did find a large amount of basophilic proteinaceous fluid that contained many nondegenerate neutrophils. Results of the culture and sensitivity showed light growth of *Pasteurella multocida*, and moderate growth of both *Fusobacterium nucleatum* and *Collinsella aerofaciens*. After 5 days, there was no growth of *Norcardia* or *Actinomyces*, which are common aerobes found in the oral cavity and upper respiratory tract of dogs that have previously been linked to pyothorax in the form of grass awn migration.

Thoracic CT scans of Leo revealed a bilateral pleural effusion that was most evident in the ventral thorax, extensive pneumothorax with atelectasis bilaterally (likely iatrogenic due to chest tube placement), a soft tissue mass in the ventral mediastinum, and a suspect mass on the dorsal aspect the right cranial lung lobe. Subcutaneous edema was also present along the right dorsal thorax and was also likely iatrogenic due to chest tube placement. After interpretation of CT scans, a presumptive diagnosis of pyothorax was made, with consideration given to the mediastinal mass seen on CT and radiographs.

#### Pathophysiology

In a normal, healthy patient, a small amount of pleural fluid exists to prevent friction during ventilation. A function of Starling's Forces, the formation of pleural effusion occurs when the normal mechanisms of pleural fluid turnover are overwhelmed, in this case by a foreign bacterial insult, causing the fluid to accumulate secondary to an inflammatory response. (5) The pyrexia associated with infection coupled with the release of inflammatory mediators and cytokines cause a change in vessel wall permeability, allowing larger cells and protein to enter the pleural space. Lymphatic drainage is obstructed as the pleural wall thickens from chronic inflammation;

as the fluid accumulates, albumin is lost in the exudate and the colloid osmotic pressure of the pleural cavity continues to increase. (5) As the oncotic pressure gradient between the pleural fluid and the vessels decreases, it is more difficult to retain fluid within the vessels. These changes ultimately lead to accumulation of a septic, purulent exudate within the pleural space. There are several proposed causes for pyothorax in dogs, but most of these cases never identify the causative agent. The current theory is that the organisms most likely to cause pyothorax normally inhabit the oral cavity and upper respiratory cavity (*E. coli, Pasteurella spp., Actinomyces spp., Norcardia spp., Streptococcus, Staphylococcus spp., Fusobacterium spp., and Bacteroides spp*). (7)

### **Treatment and Management**

Both medical and surgical treatment options exist for pyothorax, but controversy exits on how treatment is approached and whether surgery should be reserved after medical management has failed. Medical management has been reported to include initial thoracocentesis and long term antibiotic therapy, with or without the placement of a thoracostomy tube for continuous or intermittent thoracic drainage. (1,4,6,7) Patients treated only with medical management have been reported to have higher rates of recurrence, likely due to incomplete removal of the cause. For surgical treatment, both median sternotomy and intercostal thoracotomy (right or left lateral) have been described in the literature, the latter being used more for focal infections of the thorax or those with an identifiable cause. A retrospective study from 2002 surgical exploration of the thoracic cavity to identify the inciting cause, combined with thoracostomy tube placement for post-operative drainage and long-term antimicrobial therapy according to culture and sensitivity results has been associated with a better long-term outcome. (1) Complications of surgical treatment are reported in approximately 40% of all surgical cases and of those, less than 10% are

due to recurrent pyothorax. The majority of post-operative complications presented in the form of complications with the incision or the drain, regardless of technique during closure. (6) Due to the severity of Leo's dyspnea along with the possibility of a malignancy and uncertain findings from cytology, it was recommended for the thoracic cavity to be surgically explored to rule out a mass. Leo's owners elected surgical treatment and median sternotomy was scheduled to explore the thorax, due to the difficulty locating the cause and the bilateral nature of his effusion. Upon entry to the thoracic cavity, the mediastinum was markedly thickened and was deep red to dark purple in color. Occasional small, pinpoint, raised white plaques could be appreciated throughout the mediastinal tissue. The ventral part of the right cranial lung lobe was adhered to the mediastinal tissue. The abnormal mediastinum was excised and a partial right cranial lung lobectomy was performed. Both tissues were submitted for histopathologic evaluation and pieces of the mediastinum were submitted for culture and susceptibility. Prior to closure, the thoracic cavity was copiously lavaged with warmed sterile saline and a thoracostomy tube was placed to allow drainage post-operatively. Leo's sternebrae were reapposed using 18-guage stainless steel figure-of-8 cerclage wires. A wound-soaker catheter was placed through a stab incision caudal to the sternum and sutured to the skin to provide local analgesia through intermittent bupivacaine infusions to the incision site. Leo's recovery from anesthesia was uneventful, but in the days following surgery he developed a progressive dependent edema at his ventral neck, both thoracic limbs and caudal abdomen. Because Leo was hypoalbuminemic prior to surgery, he was placed on colloid therapy for 1 day (Hetastarch 20 mL/kg/day) and was treated with intermittent warm-packs and massages to disperse the edema. His edema subsided over the next two days and Leo's fluids were discontinued on Friday July 14, 2017. He was started on oral acetaminophen/codeine (1 mg/kg PO q8h), carprofen (2.2

mg/kg PO q12h), and amoxicillin/clavulanic acid (17 mg/kg PO q12h) while awaiting the culture and susceptibility results from samples taken in surgery. The thoracostomy tube was drained every 4-6 hours so the fluid could be monitored and quantified. After 6 days, character of the fluid became less opaque and amount of fluid drained from the tube had decreased to 2 mL/kg/day, indicating that this was an appropriate time to remove the tube.

#### Leo's Outcome:

Biopsy results from the mediastinum tissue samples revealed a chronic and proliferative pyogranulomatous pleuritis and mediastinitis, consistent with pyothorax. Histopathology of the mediastinal tissue showed marked fibrovascular and mesothelial cell proliferation, and there was tissue expansion due to many macrophages, plasma cells, neutrophils and lymphocytes. The right cranial lung lobe had a grossly visible nodule that was identified as hemorrhagic necrosis. This pattern of marked chronic inflammation and proliferation extended along the pleural surface of the lung. No infectious agents or evidence of neoplasia were identified on routine or special stains. A faint growth of *Prevotella denticola* was cultured from the mediastinal tissue. Tested strains were susceptible to amoxicillin/clavulanic acid; therefore, antibiosis with this antibiotic was continued for a total of 6 weeks.

Leo was sent home 8 days post-operative and was instructed to recover with strict kennel rest, controlled leash walks with a harness, gradual return to activity. At both his 4 and 8-week recheck appointments, thoracic radiographs did not show any evidence of osteolytic changes to the sternebrae or implant failure. Aside from a new rightward mediastinal shift due to the lung lobectomy of the right cranial lung lobe, Leo has been free of respiratory disease since his surgery in July 2017.

#### **Conclusion:**

Pyothorax is an uncommon diagnosis but should be a differential for any patient with the supporting signalment and history presenting with chronic respiratory signs. A minimum database should include CBC, chemistry panel, thoracic radiographs, and thoracocentesis and cytologic evaluation of the fluid is recommended prior to starting antimicrobial therapy. Surgical treatment versus medical treatment is still a topic of discussion, but retrospective studies report that those treated with medical management alone were more likely to develop a fibrosing pleuritis or a recurrence of pyothorax. Prognosis for these patients is generally good if treated appropriately, with an overall reported survival rate of 80%. (6)

## References

- 1. Stillion, J, Legendre, JA. A clinical review of the pathophysiology, diagnosis, and treatment of pyothorax in dogs and cats. J Vet Emerg Crit Care 2015; 25: 113-129.
- Brennan KE, Ihrke PJ. Grass awn migration in dogs and cats: a retrospective study of 182 cases. J Am Vet Med Assoc 1983; 182(11):1201-1204.
- Thrall, D. The Pleural Space. Textbook of Diagnostic Radiology. St. Louis: Elsevier, 2013; 550-557.
- Rooney, MB, Monnet E. Medical and surgical treatment of pyothorax in dogs: 26 cases (1991-2001). J Am Vet Med Assoc 2002, 221(1): 86-92.
- 5. Epstein SE. Exudative pleural diseases in small animals. Vet Clin North Am Small Anim Pract 2014; 55(1): 161-180.
- Fossum, TW. Surgery of the lower respiratory system: pyothorax. Small Animal Surgery 5<sup>th</sup> ed. Philadelphia: Elsevier, 2019; 947-951.
- Cote, E. Pyothorax. Clinical Veterinary Advisor Dogs and Cats 3<sup>rd</sup> ed. St. Louis: Elsevier, 2015; 879-881.