Lung Lobe Torsion in a Pug: A Case Report

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Introduction

Lung lobe torsion is a rare, life-threatening pulmonary disorder in small animals and in humans that is described as an axial rotation of the lung lobe around the bronchiovascular pedicle at the hilus.[10] This results in congestion of the lung lobe, which can lead to edema, hemorrhage, and necrosis.[5] Historically, a predisposition to lung lobe torsion has been reported in large, deep-chested dogs, with the Afghan hound being the most represented; however, lung lobe torsion has also been documented in pugs, yorkshire terriers, miniature poodles, beagles, and other small, mixed-breed dogs.[4,11,12] Lung lobe torsion has also been known to affect cats, though the prevalence is much lower than in dogs.[2,4] Documented cases can be spontaneous, but are generally due to a predisposing condition, such as thoracic trauma, pleural space disease, thoracic surgery, pulmonary parenchyma disease, and diaphragmatic hernia repair.[8]

History and Presentation

A 14 year old, spayed, female pug presented to Mississippi State University College of Veterinary Medicine on September 6th for coughing, respiratory distress, and pleural effusion. The patient had an intermittent cough for 2 months and a recent decline in clinical condition characterized by an increased frequency of respiratory distress episodes and progressive coughing. The patient initially presented to the primary veterinarian and her clinical signs were reportedly treated with a single steroid injection and promethazine with codeine (unknown dose and frequency), which seemed to resolve the clinical signs for a period of about one month. The coughing episodes reoccurred within the previous 24-48 hours prior to presentation and dyspnea was detected by the owner. The patient was re-evaluated by the primary veterinarian and a single ventrodorsal radiograph of the thorax revealed widening of the cranial mediastinum. The patient

was referred to Mississippi State University College of Veterinary Medicine (MSU-CVM) for further evaluation and treatment.

The patient presented on September, 6th 2016 to MSU-CVM in respiratory distress. She maintained a normal respiratory rate but had increased abdominal effort. A thoracic FAST scan (Focused Assessment with Sonography for Trauma) revealed mild pleural effusion. Her vital parameters were within normal limits (Temperature: 101.6 F, Heart rate: 96 beats per minute, Respiratory rate: 20 breaths per minute) with a body condition score of 8/9. No stertor or stridor was noted and no crackles or wheezes were auscultated. Ceruminous debris was noted in the right ear canal and a small amount of frank blood was noted in her stool. Her right axillary lymph node was palpably enlarged. The rest of the physical exam was unremarkable.

Immediately following presentation a diagnostic thoracocentesis was performed and fluid analysis was consistent with a hemorrhagic, non-septic exudate (protein 4.2 g/dL, nucleated cells 22,000 /uL). The inflammatory cells present consisted of 73% nondegenerate neutrophils, 23% large mononuclear cells, and 3% small lymphocytes. Erythrophagia was noted consistent with recent hemorrhage. No neoplastic or etiologic agents were identified. An aerobic and anaerobic culture of the pleural effusion revealed no bacterial growth. Due to the evidence of hemorrhagic pleural effusion, a coagulation profile was performed. The prothrombin time (PT) and partial thromboplastin time (PTT) were within normal limits (7.9 and 13.0 sec, respectively). A Complete Blood Count (CBC) revealed a mild neutrophilia (19260 /ul; reference range 3500-14200 /ul) and a chemistry panel revealed a mild hyperphosphatemia (6.4 mg/dl; reference range 2.5-5.0 mg/dl) and mildly increased alkaline phosphatase (150 U/L; reference range 11-140 U/L). A urinalysis revealed minimally concentrated urine (USG 1.019; reference range >1.030). A urine culture was negative for bacterial growth.

Thoracic radiographs showed a diffuse unstructured pulmonary pattern, retraction of the caudodorsal lung lobe on the left projection, and multiple pleural fissures lines. Additionally, there was a severe widening of the cranial mediastinum spanning the width of the thoracic cavity with a mottled soft tissue and fat opacity within the cranial mediastinum. The left mainstem bronchus was medially deviated. A thoracic ultrasound was performed to further evaluate the mediastinum. The cranial lung lobes appeared hepatized likely due to atelectasis; however, no additional information was provided and the entire mediastinum could not be evaluated. Abdominal radiographs and ultrasound showed evidence of hepatosplenomegaly, bilateral nephroliths, a nodule within the cranial pole of the left adrenal gland, diffusely dilated loops of bowel, and mucosal speckling within the small intestines. The following day, a thoracic CT scan revealed a large, triangular, smoothly marginated, minimally contrast enhancing soft tissue opacity mass measuring 69 x 27 x 68 mm at the location of the cranial subsegment of the left cranial lung lobe. These changes were suspected to be secondary to a lung lobe torsion. There was also an increased soft tissue opacity within the cranial and middle lung lobes. The bronchi were diffusely thickened and severe compression of the mainstem bronchi was present. Within the cranial mediastinum, there was an irregularly shaped, irregularly marginated, mildly contrast enhancing soft tissue opacity mass measuring 23 x 28 x 10 mm. An ultrasound guided needle aspirate of the left cranial lung lobe revealed a mixed population of inflammatory cells that consisted of neutrophils, macrophages, and small lymphocytes. Numerous mesothelial cells exhibited mild to significant amounts of atypia. No infectious agents or overt evidence of neoplasia could be seen. Treatments included continuous oxygen supplementation, Lactated Ringer's Solution (40 ml/kg/day), albuterol (1-2 puffs aerosolized as needed), butorphanol

(0.2mg/kg IV as needed), and maropitant (1 mg/kg IV q24 h). Due to high suspicion of a lung lobe torsion, an exploratory thoracotomy and lung lobectomy was scheduled for the next day.

Pathophysiology

Lung lobe torsion occurs when a lung lobe rotates around the bronchus and vasculature, remaining in that position.[4] This results in congestion of the lung lobe, which can lead to edema, hemorrhage, and necrosis.[5] The right middle and the left cranial lung lobes are most commonly affected, but torsion has been reported in all lobes.[4,5,10,11,14] The right middle lobe is predisposed due to its thin and narrow physical structure and lack of a mediastinal, thoracic wall, and adjacent lobe attachment.[6] The left cranial lung lobe also lacks widespread attachments to neighboring structures.[4] The etiology of lung lobe torsion is poorly understood, but any mechanism that results in atelectasis, pleural effusion, pneumothorax, or increases the space between the lungs can potentially result in increased mobility and subsequent torsion.[5,16] Another theory describes a partial collapse of a lung lobe, either spontaneously or as a result of disease, such as chronic respiratory disease or pneumonia; trauma, such as a migrating foreign body; or surgery, such as a diaphragmatic hernia repair.[4,15] Once the lung tissue collapses, the remaining lung lobes have more space and mobility, leading to torsion of a lung lobe.[10] It has also been postulated that bronchial cartilage dysplasia increases the mobility of lung lobes.[4] The torsed lung lobe over time becomes atelectic, resulting in a lower tidal volume and an increased respiratory rate to compensate for inadequate minute ventilation.[4] Other possible predisposing factors include an altered balance between production and absorption of pleural fluid.[16] A cause and effect relationship between pleural effusion and lung lobe torsion is highly debated. Pleural effusion may result in lung lobe torsion by causing an atelectic lobe as described above, or it may

develop due to venous congestion, resulting in leakage of fluid through the lobar pleura into the pleural cavity.[10]

Diagnostic Approach/Consideration

The most common clinical signs in dogs with lung lobe torsion include progressive dyspnea, tachypnea, lethargy, and anorexia, whereas coughing, pale mucous membranes, pyrexia, and vomiting.[10,11, 15, 16] Physical exam findings typically include muffled cardiopulmonary sounds, weakness, signs of shock, pale mucous membranes, and cyanosis.[4,10,11,15,16] History and physical exam alone are non-specific, but usually relate to the respiratory system. [4] Laboratory tests such as a complete blood count and serum chemistry profile generally results in neutrophilic leukocytosis with a neutrophilia and a monocytosis, mild anemia, hypoproteinemia, hyperbilirubinemia, elevated serum alkaline phosphatase and serum alanine aminotransferase, and an elevated serum creatinine level.[4,10] These are non-specific changes, and the diagnosis of a lung lobe torsion requires diagnostic imaging, which is the most important test in confirming the diagnosis.[14] Pleural effusion is generally present on radiographs, so a thoracocentesis should be performed to drain the fluid and radiographs repeated. The type of fluid commonly found with lung lobe torsions are sterile inflammatory, serosanguinous, sanguineous, and chylous effusions.[4,10]

Thoracic radiographs most commonly reveal lung lobe consolidation and air bronchograms may be seen early in the disease process.[8,11,12] Another predominant feature for lung lobe torsion is abnormal bronchial alignment; but mediastinal shift, pneumothorax, and pneumomediastinum have also been reported.[4, 11] However, radiographs are not always reliable to diagnose lung lobe torsion, especially when there is pleural effusion or pneumothorax, so

thoracic ultrasound is an important adjunct.[2,4] On ultrasound, as the lung becomes consolidated, the torsed lung lobe looks like normal liver tissue on ultrasound. This process is called hepatization.[4,10] Doppler is also a useful tool to estimate blood flow compromise to the lung, but the Doppler modality can be hindered by respiratory motion artifact. One study used contrast-enhanced ultrasonography to show reduction of pulmonary vascularization in a torsed lung, as well as distinguish atelectasis from a lung lobe torsion.[3] Computed tomography is another useful diagnostic device to compliment radiographs and ultrasound. The CT findings in one study are as follows: abrupt termination of the bronchus, trapped gas distal to the terminated bronchus, and a vesicular gas pattern in the twisted lobe.[9] Another study suggests using CT to rule out extraluminal masses, pulmonary or pleural disease, and using virtual bronchoscopy to visualize narrow or occluded bronchial lumen in a tapering angle to come to a more definitive diagnosis.[13] A true definitive diagnosis is achieved with direct inspection at surgery or necropsy.

Treatment and Management

If dyspnea is present, the first step to treatment is stabilization. Many patients require thoracocentesis to relieve pleural effusion or pneumothorax. Oxygen therapy is warranted if dyspnea continues to persist following thoracocentesis. In cases of severe respiratory distress that is unresponsive to oxygen therapy, anesthesia and intubation is required for stabilization.[4] Any underlying conditions, such as pneumonia, should be resolved before further treatment.[15] A lung lobectomy of the affected lung is the treatment of choice for lung lobe torsion.[4,5,7,10] Derotation of the lung lobe is not recommended due to the friable, necrotic nature of the lung after torsion. Derotation can cause the release of toxins, cytokines, and oxygen radicals, resulting in reperfusion injury and ischemia to the lung and vasculature.[5,15] A thoracotomy is performed, and before removing the lung lobe, the bronchovascular pedicle must be clamped to prevent the

release of reactive oxygen species into the bloodstream. The lobar artery is ligated, followed by the vein. The bronchus is then clamped and divided, either with suture or a stapling device. A thoracostomy tube is then placed intraoperatively in order to remove air and fluid from the pleural space postoperatively.[15] Postoperative analgesia, oxygen supplementation, antibiotics, and fluid therapy are also recommended postoperatively. Pleural effusion may not stop completely due to irritation of the tube and <2 ml/kg/day is considered an acceptable amount.[4] Once that parameter has been reached and no air has been removed from the tube for 24 hours, the tube can be removed.[4] One study performed a lung lobectomy using thorascopy and 5mm hem-o-lok clips to ligate the hilus instead of a traditional thoracotomy and suture or a stapler device.[7] Spontaneous recurrence is not common; however, in these cases, the second torsion is likely a result of the extra space provided by the lobectomy.[15] Minor regeneration of the surgical site also occurs and the remaining lobes expand to fill the remaining space.[15] Pleural effusion secondary to thoracic surgery can also predispose a second torsion.[15] The prognosis of the patient is guarded to fair and is highly dependent on the underlying condition; however, patients with chylothorax tend to have a poor prognosis.[4,11]

Case Outcome

The patient went to surgery the morning following her suspected diagnosis, on September 9th, 2016. At induction, an endotracheal wash was performed and these samples were submitted for fluid analysis, cytology, aerobic and anaerobic culture, and mycoplasma PCR. A left cranial thoracotomy of the 4th intercostal space was performed, the left cranial lung was evaluated, and it was determined that a complete lobectomy of the left cranial lung lobe would be performed. A TA-30 stapler was used to seal the pedicle to 1 mm thickness, and the lung was removed. Pleural effusion was removed using a Poole suction tip. Following removal of the torsed lung lobe, the

patient underwent cardiac arrest and cardiopulmonary resuscitation was immediately instituted using intrathoracic cardiac compressions and intrathoracic defibrillation was performed thrice. Unfortunately, the patient did not respond to resuscitative efforts. The aerobic and anaerobic culture from the endotracheal wash revealed growth of hemolytic E. coli, but only from an enrichment broth. The mediastinum was not able to be evaluated during surgery, and a necropsy was discussed with the owner. The necropsy was declined; however, the removed lung was submitted for biopsy for the purposes of this report. The histological findings were typical of a venous infarction subsequent to lung lobe torsion and cessation of venous outflow from the lung. The lung lobe had evidence of immature fibrovascular tissue but no collagen, estimating the torsion to be of 3-4 days duration. Though the underlying disease was not definitely diagnosed, it is suspected that chronic pulmonary disease predisposed the patient to lung lobe torsion based on the radiographic and CT findings. Differentials for the diffuse unstructured interstitial pulmonary pattern are chronic bronchitis, chronic irritants, or infection. Differentials for the mediastinal mass include reactive lymphadenopathy, or neoplasia, such as thymoma, lymphoma, adenocarcinoma, or sarcoma.

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