A Case Report: Grass Awn Migration and Bronchiolar Fistula in a Labrador

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INTRODUCTION

Plant material, especially grass awns, are a major contributor to foreign body disease in dogs. The pyogranulomatous inflammation or abscessation which occurs as a result of grass awn migration is commonly referred to as "grass awn syndrome".[9] This disease most frequently affects hunting and sporting breeds of dogs, likely because these animals have increased exposure to the offending plants.[6,11]

The grass awn is particularly adept at migration as it has a sharp anterior end which easily traverses orifices or skin.[1,4] The most common offending genera of grass awns in the Southeastern US are *Stipa ,* also known as Buzzard grass, and *Setaria*.[1,8] The barbed shape of the grass awn's posterior end prevents retrograde migration, and facilitates forward progressive migration of the foreign body as the animal moves. As a result, the grass awn can migrate to a variety of sites within an animal. Awns are capable of lodging in the external ear canal, which can lead to the destruction of the tympanic membrane.[1,4] Grass awns also commonly penetrate the subcutaneous tissue of the interdigital web, head, face, flank, neck, and costochondral regions where they can lead to the development of subcutaneous abscesses.[4] Grass awn migration in the sublumbar region has been reported to cause development of lumbar diskospondylitis.[1] A case report in 2012 demonstrated that intrathoracic grass awn migration can result in hypertrophic osteopathy (HO).[9] If a grass awn is aspirated or ingested it can migrate through the bronchiolar and esophageal mucosa causing bronchiolar fistula, chronic pleuritis, lobar pneumonia, pyothorax, or pneumothorax.[1,4,12]

HISTORY AND PRESENTATION

"Hey U" is a 5-year-old neutered, male Labrador retriever who presented to the Internal Medicine service at Mississippi State University College of Veterinary Medicine on February 29th, 2016 for a chronic fever of unknown origin. His owner reported that the patient had been suffering recurrent bouts of fever and lethargy since 2014. He was originally taken to his primary veterinarian in November of 2014 for respiratory difficulty. At that time, he was treated for pneumothorax, exudative pleural effusion, and a possible lung abscess or foreign body. Since his initial presentation to his referring veterinarian in November of 2014, he had suffered from cyclical episodes of lethargy and fever which had been treated with a variety of antibiotics including enrofloxacin, doxycycline, and sulfamethoxazole trimethoprim (SMZ). These episodes seemed to initially respond to antibiotic treatment, only to flare up again a few weeks later. During these episodes, the patient exhibited signs of lethargy, decreased performance, fever, and gagging. Throughout the 2015-2016 winter period he continued to show signs of lethargy and fever despite being treated with cefpodoxime by his primary veterinarian. Therefore, in late February he was referred to MSU-CVM Internal Medicine service for a more extensive work up.

On presentation the patient was bright, alert, and responsive. He weighed 29.5kgs (64.9lbs) and was assigned a body condition score of 3 out of 5. His respiratory rate, heart rate, and temperature were all within normal limits (temperature= 101.8F, heart rate= 94 beats per minute, and respiratory rate=48 breaths per minute). His mucous membranes were pink and his capillary refill time was less than two seconds. A rectal exam was performed and findings were within normal limits. Respiratory auscultation revealed normal respiratory sounds.

IMAGING AND LABORATORY RESULTS

On 2/29/2016 a complete blood count panel (CBC), serum chemistry panel, urinalysis (UA), urine culture and sensitivity, thoracic radiographs, abdominal radiographs, abdominal ultrasound, and thoracic computed tomography (CT) with contrast were performed. The CBC revealed an eosinophilia (1960 cells/uL, reference range 120-1300 cells/uL) and the serum chemistry revealed a hyperglobulinemia (4.5g/dl). The UA was unremarkable. Thoracic radiographs revealed the presence of pleural fissures and partial border effacement of the cardiac silhouette, which was likely due to the small amount of pleural effusion seen in the thoracic cavity. The abdominal radiographs were unremarkable. Abdominal ultrasound revealed a hypoechoic liver and jejunal lymph nodes, and a small amount of peritoneal effusion.

The thoracic CT with contrast revealed a large, 3.8cm by 5.1cm by 7.1 cm, multilobulated, ovoid, sharply marginated, contrast enhancing mass of soft tissue density to the left of midline at the level of the diaphragm. The mass was present on both the cranial and caudal side of the diaphragm and made its course just to the left of the caudal esophagus. The caudal most extent of the esophagus was obscured due to compression by the mass. The mass was also seen causing a ventral and leftward deviation of the lesser curvature of the stomach. The CT revealed thickened bronchiolar walls in the area adjacent to the thoracic component of the mass. The CT with contrast also revealed a large amount of heterogenous, ill-defined, contrast enhancing material of soft tissue density beginning at the level of the second sternebra and coursing caudally to the diaphragm, more to the left side of midline. Within this ill-defined material, was a more clearly defined, ovoid, sharply marginated, 1.7 cm by 1.4 cm by 1.1 cm nodule present in the right ventral thorax near the cardiac apex.

On March 1, 2016 the patient was placed under general anesthesia to have an ultrasound guided fine needle aspirate (FNA) sample taken from the ill-defined material which coursed along the ventral aspect of the thorax from the level of the second sternebra caudally to the diaphragm. The cytology of these slides revealed a mixed population of inflammatory cells including neutrophils, macrophages, and small lymphocytes. No infectious agents or overt evidence of neoplasia were noted in the slide cytology. The population of cells present in the material aspirated suggested a process of mild suppurative inflammation. The sample of material taken during the FNA was cultured and revealed a light growth of *Escherichia coli*. Differentials that were considered at this stage of the clinical investigation included: infection, trauma, or foreign body. The patient was later transferred to MSU CVM surgery service for an exploratory ceilotomy.

INITIAL TREATMENT

For presurgical supportive care, the patient was placed on intravenous Plasmalyte fluids at a rate of 1.8mls/kg/hr for the 24 hours preceding his surgery. Hey U developed a fever of 103˚ F on March 2, 2016 and was given cefpodoxime PO at 10mg/kg. On the morning of March 3, 2016 Hey U underwent an exploratory ceilotomy. Upon opening the abdomen, a large, approximately 10cm long and 5cm wide, linear shaped mass was visualized adhering to the lesser curvature of the stomach. The mass traversed the diaphragm as it coursed cranially into the thorax where is was also adhered to the left caudal lung lobe. A portion of the diaphragm was removed while dissecting around the mass. A left caudal lung lobectomy was performed as the most cranial portion of the pass was intimately adhered to a diseased left caudal lung lobe. The mass and the left caudal lung lobe were cultured and submitted for histopathology. The

diaphragm was reconstructed and a chest tube was placed to reestablish negative pressure within the thorax.

PATHOPHYSIOLOGY

Upon inhalation, the grass awn is thought to migrate distally down the trachea and subsequently along the bronchi. Due to its fusiform shape and backward pointing barbs, the grass awn undergoes progressive, forward migration which is spurred along by normal respiratory movements. Also thanks to this characteristic shape, the grass awn is unlikely to be expelled by coughing or expectoration.[2,8] The migration of a grass awn through the lung does not usually cause acute clinical signs unless pulmonary vasculature are disrupted. Therefore, acute inhalation of grass awns often goes unnoticed by the owner. Consequently, the grass awn has opportunity to continue its migration through the lungs and potentially into the pleural space, pericardium, retroperitoneal cavity, sublumbar muscles, or even through the thoracic wall.[3] Migrating grass awns cause severe inflammatory tissue reactions and can lead to sepsis.[3]

In areas with endemic grass awn disease practitioners often note encapsulated foreign material, which resembles grass awns, as a common and incidental finding in the lungs of dogs at necropsy.[8] However, in some instances, grass awns can cause extensive lung lobe abscessation with secondary bacterial involvement. In one study looking at 182 cases of suspected grass awn disease in dogs and cats, the bacteria *Actinomyces* sp. was the most common bacteria isolate from deep visceral locations, such as the thoracic and lumbar regions.[1] When the grass awn migrates into pleural space, a fulminating pleuritis typically results. This type of pleuritis usually becomes chronic and proliferative. Exacerbations in clinical signs can usually be correlated with periods during which the awn is progressively migrating.[8]

The clinical signs which occur with thoracic syndrome of grass awn disease can vary a great deal. In the early stages of thoracic syndrome common clinical signs are decreased performance and lethargy. Auscultation of respiratory sounds is usually normal in the early stages of disease. The animal may exhibit a fluctuating temperature during the early disease period.[8] As the disease progresses, dogs can show a variety of clinical signs including: depression, anorexia, dyspnea, abdominal breathing, decreased body condition, and fluctuating body temperature. Auscultation of animals in later disease stages may reveal sounds resulting from pleuritic friction, and/or moist or dry rales.[8] The thoracic syndrome of grass awn disease is typically a very chronic condition that results in clinical signs which may last for months at a time, interspersed with short periods of more acute flare-ups, along with periods of near normal physical performance.

DIAGNOSTIC APPROACH/CONSIDERATIONS

Diagnostic work ups for ill patients commonly include a thorough physical exam, full laboratory blood work, urinalysis, thoracic and abdominal radiography, and, if necessary, advanced imaging such as ultrasound or computed tomography. Animals with grass awn disease can present with a variety of clinical signs due to the many anatomical locations in which a grass awn can lodge. In cases of thoracic syndrome, when a grass awn has migrated into the thoracic cavity, animals commonly present with clinical signs such as depression, short, jerky inspirations, elevated temperature, anorexia, dyspnea, and increased abdominal effort during expiration.[8] A thorough physical exam and auscultation can reveal sounds associated with pleuritic friction and/or dry or moist rales.

A complete blood count (CBC) and serum chemistry are usually non-specific for grass awn syndrome. However, these diagnostics tools may show evidence of an ongoing infection revealing a neutrophilia, lymphocytosis, or hyperglobulinemia. One study showed that 65% of dogs with intrathoracic grass awns showed neutrophilia on CBC.[10] However, diagnosis of grass awn disease with blood values alone is difficult because these animals can present with normal, mildly, or severely elevated levels of leukocytes.[8]

Diagnostic imaging, such as radiography, ultrasound, and computed tomography, are very effective ways to diagnose and, in some cases, to assist in treatment of grass awn migration in dogs. In cases of thoracic syndrome of grass awn disease thoracic radiographs often reveal a diffuse increase in opacity in the dorsocaudal lung fields and the presence of pleural fissures lines.[3] These radiographic findings are consistent with thickened lung pleura and pleural effusion.[5,8]

Ultrasound can be very helpful in the evaluation and identification of foreign bodies, such as grass awns. Grass awns have a characteristic appearance on ultrasound. They usually appear spindle shaped with two to three linear echogenic protuberances projecting outwards. The inflammatory reaction that occurs in the soft-tissue surrounding the grass awn can also be helpful in identification, as the accumulation of inflammatory fluid around the foreign body creates an anechoic halo to allow for better visualization of the interfaces.[6] The treatment of grass awn syndrome typically involves the removal of the foreign material, therefore, ultrasound guided surgical removal of grass awns can be used as a means to treat, as well as diagnose, grass awn disease. A recent study demonstrated the successful removal of a migrating grass awn within the pulmonary paraenchyma of a dog by using transesophageal ultrasonography to visualize and subsequently remove the grass awn with thoracoscopic removal.[2]

Computed tomography (CT) can be a useful method for localizing the site of grass awns and acting as a guide for exploratory surgery and ultrasound.[13] Grass awn migration often causes the development of a bronchopleural fistula, i.e. is a communication between the bronchus and the pleural space. Bronchopleural fistulas can lead to the development of spontaneous pneumothorax, a life threatening condition. CT examination can aid in the diagnosis of spontaneous pneumothorax due to bronchopleural fistulas, which occur secondary to foreign body migration.[12] During a CT examination grass awns commonly appear as elongated gas-containing foci or hyperattenuating foci within soft tissues.[10]

POST-OPERATIVE TREATMENT

Immediately following surgery, the patient was taken to the Intensive Care Unit where his recover from anesthesia was uneventful. Perioperative pain management and medical intervention included intravenous Plasmalyte fluids run at 2.7mls/kg/hr with 20 mEq/L of postassium chloride (KCl), dexmedetomidine and morphine constant rate infusion (CRI), carprofen SQ at 4.4mg/kg q24hrs, and cefazolin IV at 20mg/kg q8h. A few hours after recovery Hey U's chest tube was re-sutured in place because of a leakage. After that time, his chest tube was aspirated every 4 hours and the amount of air drawn back was recorded. In the 12 hours following surgery, he was provided with oxygen supplementation via nasal cannula and constant ECG monitoring.

The day after surgery, 3/4/2016, the patient was taken off oxygen supplementation and ECG monitoring. He was also switched to oral medications which included: cefpodoxime at 10mg/kg PO q24h, carprofen at 2.2mg/kg PO q12h, trazodone at 3mg/kg PO q8h, and Tylenol 4

at 2mg/kg PO q8h. The chest tube was removed at this time. On the morning of 3/5/2016, the patient was discharged to his owner with instructions to continue current medications and to return if he began showing signs of illness.

DIAGNOSIS

In many cases, the most concrete way to definitively diagnose grass awn disease is via histopathology of the suspected grass awn foreign body and the surrounding tissues. In some cases, the grass awn shape may be recognizable via advanced imaging like ultrasound and computed tomography.[2, 3] However, in this case the grass awn was surrounded by many layers of fibrous tissue and was no longer recognizable by the characteristic shape of a grass awn.

Histopathology of the mass revealed multiple foci of plant foreign material surrounded by foci of intense purulent exudate with a predominant cell type of neutrophils intermingled with fewer macrophages. These foci of abscessation and plant foreign material were divided by broad bands of fibrous connective tissue. In some of these inflammatory foci, colonies of bacteria were identified. In some sections of the diseased tissue, the plant foreign material was marginated by macrophages and giant cells with peripheral fibrosis and then infiltrated with lymphocytes, eosinophils, plasma cells, and neutrophils. Plant foreign material was also identified in dilated distal bronchi of the left caudal lung lobe which was surgically removed and submitted to histopathology. The histopathologic results suggested a chronic, but very active inflammatory process. A Warthin Starry stain of the diseased tissue showed large numbers of elongated filamentous bacteria suggestive of Nocardia or Actinomyces.

TREATMENT

In the case of thoracic syndrome of grass awn disease, the best hope for a successful treatment is to surgically remove the causative agent.[2, 8] The use of scoping techniques can be helpful to visualize the grass awn and assist in its removal. One study examining the outcomes of 43 cases of suspected migrating intrathoracic grass awns used a thoracoscopic approach to visualize and assist in grass awn removal. The thoracoscope port was placed 2 or 4 intercostal spaces cranial or caudal to the location of the grass awn, which had previously been identified via ultrasonography. When the grass awns were visible protruding through the lung pleura, they were removed using 2.5mm Hartmann forceps through an instrument port.[2] When the foreign body was not visible, a partial lung lobectomy was performed to remove the diseased portion of the lung.[2] In conservative treatment, it is important to provide continuous drainage of the pleural cavity, along with systemic and local antibiotic therapy based on the outcome of fluid cultures.

As in many cases of disease, the best treatment is often prevention. The most practical method of prevention of grass awn disease appears to be a brief cleansing of the mouth and nose after a dog has spent time hunting or in grassy areas. Carefully cleaning these areas with a cloth can be useful to dislodge any foreign plant material which may be attached to the oral or nasal mucosa.[8]

CASE OUTCOME

On March 5, 2016 the patient was discharged from the surgery service of Mississippi State University College of Veterinary Medicine. Medications he was sent home with included cefpodoxime at 10mg/kg q24hrs for 5 days, trazodone at 3mg/kg q8hrs for 14 days, and Tylenol

4 at 2mg/kg q8hrs for 7 days. This choice of antibiotic was based on the culture results from pleural fluid that was aspirated from the patient's thoracic cavity on March 1, 2016.

In late March, he again began showing signs of fever and lethargy. He was taken to his local veterinarian who performed chest radiographs. The thoracic radiographs revealed the presence of pleural effusion. The patient was given cefpodoxime at 10mg/kg by his local veterinarian at that time. This antibiotic regime did not resolve the episode of illness, so he was taken back to his local veterinarian in early April. At this time, the fluid in his thorax was cultured revealing an anaerobic infection. The patient was then placed on clindamycin and furosemide. His owner reports that after finishing the 4 week regime of these two drugs that he has had no more episodes of fever or lethargy. He is now back to performing at agility competitions and has recovered extremely well.

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