“Chronic Diaphragmatic Hernia in the Cat”

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Class of 2018

Clinicopathologic Conference

Presented on July 28, 2017

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Introduction

A hernia is described as an internal organ or structure that pushes through a weakness or defect in the surrounding muscle or tissue into an area where it normally would not inhabit.[1] To be considered a "true hernia", the components must include the hernial ring, contents, and sac, where as a "false hernia" does not include a sac.[1] While there are many different types of hernias, the most common types include protrusion of an organ through the abdominal wall, thoracic wall, or between the muscles of the pelvic diaphragm.[1] Thoracic and abdominal hernias can be either acquired (traumatic in origin, including, but not limited to: hit by car, kicking, bite wound, etc.) or congenital and should be further distinguished, by site, as hernias are described based on their location.[2,3,4,5,11,12]

One of the more frequently diagnosed hernias in small animal surgery is the diaphragmatic hernia, with one article suggesting as many as 80% of hernia cases being diaphragmatic hernias.[2,3] This may include: pleuroperitoneal, peritoneopericardial, or hiatal (acquired or congenital) diaphragmatic hernias. [1,3,7] The treatment of choice for any of the above mentioned hernias is surgical repair, or herniorrhapy, but the immediacy of the surgery is most often determined by overall stability of the patient at the time of diagnosis and financial capability.[3,4,5,12] Another factor to consider when determining if surgery is in the best interest of the patient at the time of diagnosis is determining the nature of the hernia, whether it be acute or chronic (>1-year duration), as some studies have suggested poorer prognosis in chronic cases.[3,4,5,7] In most cases, acute hernias are acquired, or traumatic in origin, and patients often present with other concurrent injuries separate from the hernia, but chronicity of a hernia does not necessarily rule out other associated co-morbidities.[3,4,5] Acute or chronic hernia patients can present with a wide variety of clinical signs including, but not limited to: respiratory distress,

tachypnea, tachycardia, abnormal posture, vomiting, and anorexia.[3,4,5,11,12] Overall, the extensiveness of the associated defect, associated co-morbidities (if present), and financial capabilities of the owner all play a major role in survival of the patient as one study suggested 15% of traumatic diaphragmatic hernia cases die before/during presentation.[4,5,11]

History and Presentation

Elsa is an approximately 6-month-old, intact female, domestic short hair kitten that was presented to the Mississippi State University College of Veterinary Medicine Community Veterinary Services for assessment of blepharospasm and epiphora of her left eye. Elsa was adopted by her owner around 6 weeks old, after an individual found her in respiratory distress as a kitten and had her treated accordingly. Records of this treatment could not be obtained prior to admittance to MSU-CVM. She had been healthy since adoption, with the exception of an historical mild increase in respiratory rate. Elsa presented with a moderately elevated respiratory rate (80 breaths per minute), and upon auscultation of Elsa's heart and lungs, her right thorax auscultated normally, but her left thorax revealed muffled heart and lung sounds. Abdominal palpation revealed a soft, small abdomen, with minimal contents being palpable.

Diagnosis of a diaphragmatic hernia in small animals is generally discovered through a combination of history, clinical signs, and radiographic evaluation of the thorax and abdomen.[4,5,6,8,11,12] Although cases of acquired hernias can be easy to diagnose when they are associated with a severe traumatic accident and patients present with respiratory distress or shock, cases can be more difficult to diagnose when there are minimal clinical signs.[5,11] One study found a diagnosis of less than 57% of cases within 30 days of initial occurrence, as the animals presented with minimal respiratory distress.[11] With regards to congenital diaphragmatic hernia, patient history often includes: failure to thrive, regurgitation, cyanosis, dyspnea, and

death soon after birth.[9] Clinical signs are very generalized and do not always indicate that an animal has a diaphragmatic hernia, and other differentials should be included to rule out other or concurrent diseases.[4,5,11] Most studies have found that respiratory distress is the number one associated clinical sign associated with this disease, but commonly occurring clinical signs include tucked abdomen, muffled heart sounds, absent lung sounds, regurgitation, anorexia, weight loss, lethargy, and abdominal discomfort.[4,5,11]

Pathophysiology

True congenital and traumatic diaphragmatic hernia cases generally have a clinically similar presentation, despite the difference in how the hernia was acquired.[3,4,7] The etiology of congenital diaphragmatic hernia cases in small animals is not well understood, as only 5-10% of case presentations are congenital in nature.[7] One study pertaining to congenital diaphragmatic hernias in the dog suggested a genetic component associated with autosomal recessive inheritance.[9] Valentine, et al. bred a historically diagnosed Golden Retriever sire to three unrelated female dogs, and then back bred two F1 generation offspring (all clinically normal) to the sire.[9] Of the 27 puppies sired in the F2 generation, 18.5% were diagnosed with congenital diaphragmatic hernia with positive contrast radiography, and the defect in the diaphragm was noted in the dorsolateral portion of the left crus.[9] The defect is proposed to occur during embryogenesis due to incomplete fusion of the left pleuroperitoneal fold, which later becomes part of the left crus of the diaphragm.[2,9]

Of the two most common hernias, traumatic diaphragmatic hernia pathophysiology is much more understood than its congenital counterpart.[3,4,5,7,12] The most common cause of traumatic diaphragmatic hernia in small animals is due to automobile trauma, but in other instances, bite wounds, forceful kicks, and excessive fall height have been shown to cause an

acquired hernia.[2,3,4,5,11,12] The rapid increase in intraabdominal pressure combined with an open glottis causes an acute, rapid rise in the pressure gradient between the thoracic and peritoneal cavities, causing the abdominal viscera to herniate through the weakest point of protection in the diaphragm.[2,11,12] Generally, the diaphragmatic costal muscles are the most likely area to form a rent, as the central tendon and crural muscles of the diaphragm are more tendinous in nature.[2,11,12] Traumatic hernias should be critically evaluated at the time of presentation, as studies have suggested a varying rate of 52-88% survivability; with those animals presenting with other co-morbidities such as fractures, hypovolemia, and shock being less likely to survive.[4,5,11] In both congenital and acquired hernia presentations, there can be varying levels of abdominal herniation, with the liver, stomach, small intestines, and spleen being the most common organs to be found in the thoracic cavity.[1,2,10,11] Patients should be examined thoroughly as entrapment and strangulation of abdominal viscera are also associated with a poorer prognosis.[10,11] In both cases, differential diagnoses for pleuroperitoneal diaphragmatic hernia should include: peritoneopericardial hernia, hiatal hernia, lung atelectasis, pleural effusion, megaesophagus, gastroesophageal intussuception, pleural mass, and pulmonary mass.[3,5,7]

Diagnostic Approach/Considerations

When combining associated clinical signs with diagnostic tools, radiographs are usually the most useful tool in diagnosing this disease as displacement of the abdominal viscera into the thoracic cavity is indicative of a defect in the diaphragm.[2,6,8,11] If a clinician is suspicious of a diaphragmatic hernia, and further diagnostics are warranted due to increased thoracic density, the lateral projection is generally the most useful projection combined with positive contrast administration if necessary.[2,8] In one study, clinical radiographic signs most reliably associated

with diagnosis of a diaphragmatic hernia beyond that of loss of the diaphragmatic line, included: changes related to cardiac shadow (70% of cases), increased intra-thoracic density (87%), and presence of abdominal gas in the thorax (73%).[8] In addition to these signs, a displaced trachea, a cranioventrally displaced stomach (50% of cases), and dorsally displaced bronchi (50% of cases) were also useful in diagnosing a diaphragmatic hernia.[8] If radiography is not found to be useful, ultrasound has also been used to diagnose diaphragmatic hernias, and one article found an accuracy of 93% with regards to diagnosis.[13]

Elsa's thoracic radiographs revealed a large amount of soft tissue to fluid opaque material in the left thoracic region, displacing the lungs, pulmonary vasculature, and cardiac silhouette dorsally and to the right. Numerous loops of small intestine and colon were also present within the thoracic cavity. Abdominal radiographs revealed an indistinct diaphragmatic margin, with the colon seen passing through the diaphragm and into the thoracic cavity. Minimal abdominal contents could be appreciated in the abdominal cavity, with the exception of a portion of the liver, portion of the stomach, kidneys, and distal portion of the colon.

Based on the radiographic findings, it was determined that Elsa had a diaphragmatic hernia causing herniation of most of her gastrointestinal contents into her left thorax region of her chest. Due to Elsa being a young animal, with an unknown historical respiratory illness, the differential diagnoses for her included: congenital pleuroperitoneal diaphragmatic hernia and traumatic pleuroperitoneal hernia. Taking into account the etiologies of both diseases, individuals with the congenital form rarely survive through birth, while acquired hernias are generally caused by some form of trauma.

Surgery was scheduled for the following day to repair the diaphragmatic hernia and to assess the vitality of her abdominal organs. A Minimum Database, including a complete blood

count and serum chemistry, was performed prior to surgery to assess Elsa for any unknown health complications, which revealed no significant abnormalities**.**

Treatment and Management

In cases of diaphragmatic hernias, there are very few options for treatment of this disease and associated disease complications.[12] Medical management is not warranted, as the disease will generally progress over time, with increasing amounts of abdominal viscera decreasing space within the chest cavity.[1,4,5,12] As the disease becomes more chronic, adhesions can occur, causing the hernia to become less reducible and the lungs to become more atalectic.[3,4,5,12] Due to these reasons, no treatment or medical management is not something that is generally suggested, as it reduces the quality of the life of the animal.[11,12] Euthanasia should be considered if surgery is not an option.[3,4,12]

Ventral midline celiotomy with diaphragmatic herniorraphy is the surgical treatment of choice in managing animals with this disease, but other diagnostic approaches such as a median sternotomy or ninth intercostal lateral thoracotomy have also been used to correct a defect in the diaphragm.[2,3] A ventral midline celiotomy allows for evaluation of all abdominal organs, and the incision can be extended cranially to include a median sternotomy if excessive adhesions or an irreducible hernia is present.[2,3] In contrast, a ninth intercostal lateral thoracotomy is very useful when looking at the severity of the hernia and adhesions present, but exposure only includes one side of the thorax.[2,3] Other diagnostics must be performed before surgical intervention, as a contralateral thoracotomy must be performed if the incision is made on the opposite side of the herniation.[2,3]

With surgery being the treatment of choice in managing animals with this disease, there have also been historically high associations with death occurring during or after surgical

intervention .[4,5,11] A retrospective study performed by Legallet, et al. on diagnosed traumatic diaphragmatic hernia cases from 2001-2013 found that in acute cases, 83.3% of cats survived and 79.2% of dogs survived through surgical intervention. This same study also compared chronic case survival rate, and 100% of cats survived and 80.6% of dogs survived.[4] This data suggests a better prognosis than a previously published research article that found a 62.5% survival rate, but this paper combined congenital, acquired, acute, and chronic diaphragmatic hernias.[4] This increase in survival rate could also be attributed to better pre-surgical stabilization and correction of cardiovascular shock. [4,5]

Because patients often have other concurrent injuries, they should be stabilized prior to surgery due to shock or cardiovascular compromise.[4,5] Legallet, et al. suggested that there was a significant increase in mortality rate if animals had an increased surgical time, had concurrent orthopedic/soft tissue injuries (7.3 times greater odds of mortality), and were perioperatively dependent on oxygen (5 times greater odds of mortality).[4] Another retrospective study performed by Schmiedt, et al. found an association between age and respiratory effort, with older cats, cats with dyspnea or lower respiratory rate, and cats with concurrent injuries being more likely to die after repair of a hernia.[5] At the time of surgical intervention, organs should be assessed for viability, as returning under perfused abdominal viscera to the abdomen allows for reperfusion and subsequent release of lethal by-products produced by anaerobic metabolism, lessening chances of survival.[10,11] This phenomenon is known as ischemia-reperfusion (IR).[10,11]

In addition to the previously mentioned complications, cats specifically have been shown to have decreased survival rates due to a phenomenon called re-expansion pulmonary edema (RPE).[10] RPE pathogenesis is not fully understood, but one theory is that there is a mechanical interruption in the alveolar vessels in the lungs due to the acute increase in negative pressure in a

previously atalectic lung.[10] This causes an increased hydrostatic pressure in the alveolar capillaries, and subsequent release of fluid, causing pulmonary edema in the lung tissue, with pleural effusion sometimes being noted as well.[10] Recommendations at this time suggest that previously atalectic lungs should gradually be re-expanded, and animals should be recuperated with a pneumothorax, that is slowly relieved over 8-12 hours.[10] Patients should be closely monitored for dyspnea, poor ventilation, and frothy, serosanguinous fluid in the mouth.[10]

Upon entering of Elsa's abdomen, a 5cm tear was noted in the tendinous portion of the diaphragm, with intestines, liver lobes, gallbladder, stomach, and pancreas being noted to have herniated through the defect into the thoracic cavity. Adhesions were present, causing the liver to become attached to portions of the diaphragm and mediastinum. All fibrous adhesions were broken down to further facilitate proper placement of all herniated viscera back into the abdominal cavity. After removal of visceral contents from the thoracic cavity, Elsa's heart rate and respiratory rate slowed significantly, with atelectasis and decreased tidal volume being noted on the left and right sides of her chest, respectively. All anesthetic drugs were discontinued or reversed as needed, and Elsa's tidal volume was increased as necessary. A chest tube was placed in the left thoracic cavity wall, and the diaphragmatic rent was sutured closed. The cavity was then emptied of all air until negative pressure was re-established, and 30mls of air were added back to reduce the likelihood of re-expansion pulmonary edema after refilling the lungs. The incision was then closed in a normal fashion, and Elsa was transferred to ICU into the oxygen cage until she was stabilized.

Case Outcome

Although the prognosis of diaphragmatic hernias has a good survival rate, it is very specific to individuals on a case by case basis.[4,5,11] Elsa underwent a planned surgical repair for

her chronic diaphragmatic hernia. She was very stable prior to surgery, but she had respiratory complications that led to her lungs being filled back up quickly rather than a gradual re-expansion. While she was not diagnosed with REP, Elsa was given post-operative medications such as mannitol to reduce the likelihood of acquiring pulmonary edema post surgery. Elsa was also pre-operatively and post-operatively placed back in the oxygen cage, as she had difficulty waking up for a few hours after her surgery ended. While this has been associated with increased mortality, Elsa returned back to MSU-CVM two weeks later for her re-check, and she was eupnic, with a normal respiratory rate. At this time, Elsa has not had any further complications, and she has been healthy per her owner.

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