Traumatic Diaphragmatic Hernia in the Canine

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Introduction:

A diaphragmatic hernia, also known as pleuroperitoneal hernia, occurs when there is a disruption of the continuity of the diaphragm such that the abdominal organs can shift into the thoracic cavity. Diaphragmatic hernias are caused by trauma in up to 85% of cases.[3,17,18] Traumatic diaphragmatic hernias are most commonly seen in male dogs ranging from one to three years of age; however, they can occur in both dogs and cats.[3,9,14] Congenital pleuroperitoneal hernias are rarely reported and many affected animals die soon after birth from severe respiratory deficiencies.[8] The clinical course of traumatic diaphragmatic hernias can either be acute or chronic and dyspnea is the single most common clinical sign seen in 38% of affected animals.[8,9,14] Acute traumatic hernias usually present with a history of trauma and the patient may be in shock, in respiratory distress, or have pale cyanotic mucous membranes. The patient who presents with a chronic diaphragmatic hernia may have no clinical signs and the hernia might be an incidental finding, typically radiographic. However, some chronic diaphragmatic hernia patients present with a history of dyspnea, tachypnea, exercise intolerance, anorexia, depression, vomiting, dysphagia, diarrhea, constipation, weight loss, difficulty in lying down, or abdominal distention. Physical exam findings include: signs of hypovolemic shock (including dyspnea and/or tachypnea, pale or cyanotic membranes, weak pulses, tachycardia), cardiac arrhythmias, muffled heart and lung sounds, borborygmi on thoracic auscultation, hyperesonance on chest wall percussion (indicating gastric tympany), hyporesonance on chest wall percussion (indicating pleural effusion), tucked-up or empty appearance of the abdomen or abdominal distention with fluid wave if ascites is present.[9]

A diaphragmatic hernia is verified based on the loss of diaphragmatic integrity by thoracic or abdominal imaging. Thoracic radiographs may show: loss of the diaphragmatic line,

loss of the cardiac silhouette, dorsal displacement of the lungs, pleural effusion, or presence of gas, indicating the stomach or intestines are within in the thoracic cavity.[9] Abdominal radiographs may show the absence or cranial displacement of normal abdominal viscera.[9]

When treating a patient with a diaphragmatic hernia, there are four main goals: stabilize the patient, resolve respiratory distress, return the abdominal organs to the abdominal cavity, and repair the diaphragmatic defect.[9] Surgery is the most common treatment utilized to repair diaphragmatic hernias. Generally, prognosis is good if the animal survives the early postoperative period of 12 to 24 hours with survival to discharge rates ranging from 82% to 89%; but, 15% of animals will die prior to presentation and surgical correction of traumatic diaphragmatic hernias.[7,8] The purpose of this case report is to describe the management of one case of a traumatic diaphragmatic hernia in a one-year old mixed (Schnauzer/Poodle) breed canine using surgical intervention.

History and presentation:

Jolly, a one-year old, intact male mixed breed canine, presented to his primary veterinarian on Sunday, January 21th, 2018 for a 1-day history of increased lethargy and tachypnea. Saturday, January 20th, Jolly's owner noticed Jolly was becoming increasingly lethargic, so she decided to monitor him throughout the night. The next morning, Jolly's owners believed he had not improve and seemed to have an increased respiratory effort as well and elected to take him into his primary veterinarian. During the physical examination, Jolly's primary veterinarian elicited slight pain on abdominal palpation. Thoracic radiographs were performed which revealed abdominal contents within the thoracic cavity as evidenced by loops of bowel seen on the lateral view with an indistinct diaphragm and abdominal organs taking up the majority of the left hemithorax on the ventrodorsal view. At this time, Jolly was diagnosed

with a diaphragmatic hernia, possibly traumatic in origin due to his previously known history of enjoying chasing horses on his farm of which he has free roam, and referred to MSU-CVM Small Animal Surgery for repair.

Upon presentation MSU-CVM Emergency Service on Sunday, January 22nd, Jolly was bright, alert and responsive and weighed 10.4kg with an ideal body condition score of 5/9. He was normothermic with a temperature of 101.7 degrees Fahrenheit, normocardic with pulse of 96 beats per minute and slightly tachypneic with a respiratory rate of 36 breaths per minute. His hydration status was normal. Heart sounds were decreased and muffled bilaterally, more so on the left than right hemithorax, but normal heart sounds and no murmurs or arrhythmias were auscultated. Increased bronchovesicular sounds were noted on the right craniodorsal aspect of the lung fields, with decreased lung sounds heard in the caudodorsal left lung fields. The rest of the physical exam was within normal limits. On thoracic FAST scan, an intact spleen, stomach, loops of intestine and some of the liver were suspected to be in the thoracic cavity. Multiple lung rockets were also observed on thoracic FAST scan, ruling out a pneumothorax, but indicating the presence of interstitial lung fluid, either edema or hemorrhage, due to pulmonary contusions or parenchymal injuries.[4] Abdominal FAST scan revealed the intestines to be more cranial than normal, a relatively empty abdominal cavity with an intact small urinary bladder and no free fluid. ECG, SpO2 and blood pressures were within normal limits and no external wounds or abrasions could be appreciated.

Based on the history, presentation and further examination of the primary veterinarian's radiographs, Jolly was confirmed to have a diaphragmatic hernia as previously diagnosed by his primary veterinarian was scheduled for herniorrhaphy on January 23rd. An intravenous catheter was placed in Jolly's left cephalic vein and he was given methadone intravenously (IV) every 6

hours throughout the night while remaining in the Intensive Care Unit to manage pain and discomfort, in addition to monitoring for signs of sudden respiratory decompensation. Throughout the night, Jolly was quiet and calm in his kennel.

Pathophysiology:

Various etiologies can result in a diaphragmatic hernia and are divided into two categories: congenital or traumatic. A defect in the dorsolateral part of the diaphragm or a portion of the left lumbar muscle of the crus may be absent, or both crura may be deficient in addition to central parts of the tendon leading to congenital diaphragmatic hernias; however, these are not the focus of this work due to the presumptive traumatic nature of Jolly's injuries.[9] Traumatic injuries to the diaphragm are either direct or indirect. Direct injuries might involve a thoracoabdominal stab or gunshot wound, although rarely seen in animals.[2] Iatrogenic injury can occur during thoracocentesis, motor vehicle accidents, blunt trauma to the abdomen, or an abrupt increase in intra-abdominal pressure with an open glottis resulting in a large pleuroperitoneal pressure gradient.[3,9,17]

Normally, during quiet inspiration, the pleuroperitoneal pressure varies from 7 to 20 cm H_2O. On maximal inspiration, however, it increases beyond 100 cm H_2O.[8,9] The high pleural pressure provides some stabilization to the diaphragm. Application of force to the abdominal cavity with the glottis open increases the peritoneal-to-pleural gradient, and herniation of the viscera is usually immediate after the diaphragm tears.[5,8] The diaphragm usually tears at its weakest points, which are the muscular portions such as the diaphragmatic costal muscles. The location and size of the tear in the diaphragm is dependent on the position of the animal at the time of impact and location of the viscera. In dogs, radial and circumferential tears of the

diaphragmatic costal muscles are both equally common with each tear occurring 40% of the time; however, in 20% of the cases, a combination of the two tears occurs.[6]

Due to the nature of automobile or other traumatic events, multisystem injury and shock are potential complications in patients with traumatic diaphragmatic hernias. Viscera malposition in the thoracic cavity can suffer from ischemic injury from alterations in blood flow, which may lead to incarceration, obstruction and strangulation.[3,9] The consequences of obstruction of the gastrointestinal tract depend on the level at which the blockage occurs.[8] Herniation of the liver may result in hepatic venous stasis, hepatic necrosis, biliary tract obstruction, and jaundice.[8] Venous congestion of entrapped liver lobes within the thoracic cavity may then lead to pleural or abdominal effusion.[9] With liver herniation, compression or collapse of the portal vein, intrahepatic sinusoids or hepatic veins results in occlusion to hepatic venous outflow leading to hepatic congestion.[8] As a result, hepatic lymphatic vessels distend, and copious quantities of hepatic lymph are extravasated through the liver capsule.[8] Depending on location of the lobe, rapid accumulation of pleural fluid, pericardial fluid, ascites or a combination occurs.[8,12]

Clinical signs reflect respiratory dysfunction secondary to loss of diaphragmatic integrity, pleural effusion, or displacement of pulmonary parenchyma or abdominal viscera. Lack of a functioning diaphragm due to a tear, lung compression by abdominal viscera and shock in addition to respiratory insufficiency may result from dysfunction of the chest wall, pleural space, lungs, airway and cardiovascular system.[3] With rupture of the diaphragm, parietal pleural contact with the lungs is lost causing pleural and peritoneal pressures to equalize.[8] When this occurs, abdominal and thoracic wall muscles take over the job of the diaphragm. Thoracic expansion is then limited by pain and mechanical factors associated with concurrent chest wall contusions, rib fractions and flail chest.[8] Compression and atelectasis of the lung lobes by herniated organs, fluid

or air in the pleural space lead to hypoventilation, ventilation-perfusion mismatch, and hypoxia.[8] Pulmonary function further deteriorates in patients in shock with an increase in pulmonary vasculature permeability, pulmonary edema, and hypoventilation, which causes further ventilation-perfusion mismatch.[8]

Diagnostic approach:

A complete blood count (CBC), biochemistry panel urinalysis provides variable results depending on time of presentation, severity of clinical signs, and organs displaced in the thorax. Thoracic radiographs should be taken of all patients that have a known history of severe trauma, particularly in patients presenting with dyspnea or fractures.[13,16] Thoracic radiographs show a loss of diaphragmatic visualization, loss of cardiac silhouette, dorsal displacement of the lungs, pleural effusion, or presence of gas (stomach or intestines) in the thoracic cavity.[9] The single most useful view for diagnosing a diaphragmatic hernia is the lateral projection, which will reveal partial loss of the normal line of the diaphragm in 66 to 97% of patients.[15] In a patient with a large volume of pleural effusion or to alleviate hypoventilation, thoracocentesis should be performed prior to thoracic radiographs to aid in diagnosis. Abdominal radiographs show absence or cranial displacement of normal abdominal viscera.

Ultrasonography and contrast radiography are used as advanced or confirmatory diagnostic testing. Ultrasonography demonstrates a rent in the diaphragm, the organs herniating through it, or abnormally positioned viscera; however, this is not always true in every case. Ultrasonography is more useful in making the diagnosis in patients with pleural effusion without the need to drain off the pleural fluid. Contrast radiography of the intestinal tract shows barium-filled stomach or intestine in the thoracic cavity definitively diagnosing the condition.[8] In one study, contrast radiographs were helpful in making a diagnosis in 11 of 14 dogs in which initial

plain radiographs were nondiagnostic.[8,15] However, contrast studies in some cases are nondiagnostic if the animal has partial gastrointestinal obstruction and thus, delayed transit of the contrast media.[8] Diaphragmatic hernias are also incidentally diagnosed during exploratory surgery.

Treatment and management:

Oxygen administration via face mask, nasal cannula, or oxygen cage should be provided to the patient who presents dyspneic or hypoxic. Also, fluid therapy may be necessary to stabilize patients who have cardiovascular compromise, especially in patients with severe acute trauma and blood loss. If not already done during the diagnostic period, thoracocentesis is necessary in unstable patients with severe pleural effusion and in respiratory distress. Positioning the patient in sternal recumbency with the head elevated above the rear limbs (forelimbs elevated) using towels or soft blankets, if tolerated, may also help to elevate some of the pressure of the abdominal viscera on the lungs and heart to aid in adequate ventilation of the patient.[9] These acute treatments may help aid in patient stabilization prior to surgery; however, if the stomach has herniated into the thoracic cavity, surgery should be performed as soon as possible. Gastric herniation that causes compression of the caudal vena cava and lungs as it distends is rapidly fatal.[6] Acute gastric gaseous distention effectively can produce a tension pneumothorax that can be managed by emergency decompression with a hypodermic needle or passage of an orogastric tube.[8]

Surgical repair is the most common course of treatment for diaphragmatic hernias and should be performed at the earliest opportunity in a stable patient and prior to fracture repair. A midline celiotomy is the most common approach used to repair diaphragmatic hernias; however, a median sternotomy of the caudal two or three sternebrae extending the midline celiotomy may

also be required for additional exposure and to break down adhesions.[1,6,11,17] Rarely, a ninth intercostal lateral thoracotomy is used in cases in which the side of herniation is known that also provides exposure to the organs and diaphragmatic tears.[14] After making the initial incision appropriate for each patient, excising the falciform ligament may aid in visualization of the abdominal cavity, but is not mandatory, Finochetto or Balfour self-retaining retractors are inserted to provide better exposure of the cranial abdominal cavity, and peritoneal and pleural fluid are aspirated.[8] Then, abdominal organs are returned to the abdominal cavity. The liver is the most common organ herniated, being found in the thoracic cavity in 88% of patients, followed by the small intestine, stomach, spleen, omentum, pancreas, colon, cecum and uterus.[3,6,17] The side in which the diaphragmatic defect is present plays a role in which organs will have herniated. In right-sided tears, the liver, small intestine, and pancreas tend to herniate, but on the left, herniation of the stomach, spleen and small intestine is more common.[6]

Organs that have become congested and swollen or developed adhesions need the diaphragmatic defect to be enlarged with a ventrally directed radially incision, avoiding major phrenic vessels, phrenic nerves, and the caudal vena cava.[8] Serosal adhesions less than two weeks old consisting of only fibrin may be easily broken down and then, with gentle traction, the abdominal organs herniated should be easily replaced.[8,9] Mature organized adhesions are broken by sharp separation using scissors, electrocautery, or transection of one of the organs involved using appropriate surgical technique.[8] Before closing the diaphragmatic tear, warm saline should be used to lavage the abdominal and thoracic cavity.[8] The diaphragmatic defect can be closed with 3-0 to 0 USP absorbable synthetic monofilament suture in a simple continuous pattern commencing dorsally ending ventrally. In chronic diaphragmatic hernias, atrophy and fibrotic contracture of the diaphragm may prevent complete closure of the rent with suture. Other closure

options can be used, such as omentum, muscle, liver, fascia, polypropylene mesh, or silicon rubber sheeting.[8] Prior to complete closure of the defect, a mechanism to remove the air and fluid from the pleural cavity should be put into proper positioning to be used after closure is complete. In certain patients, an indwelling thoracostomy tube should be placed in the lateral thoracic cavity into the pleural cavity. Gradual re-expansion and re-inflation of chronic atelectatic lungs should be done because rapid re-expansion with excessive pressures results in reperfusion injury of the collapsed vascular beds and the development of pulmonary edema several hours after surgery.[8] Finally, examining the entire abdominal cavity for associated injuries if the hernia was traumatic in origin is a must.

Severe complications will occur in 50% of diaphragmatic hernia patients during the post-operative period.[8] The first 24 hours after surgery, death is caused by hemothorax, pneumothorax, pulmonary edema, shock, pleural effusion, and cardiac dysrhythmias.[3,6,18] Death occurs later in patients secondary to rupture, obstruction, or strangulation of the GI tract or diseases unrelated to the hernia.[3,6] Re-expansion pulmonary edema occurs in patients in which lung re-expansion takes place rapidly during surgery and should be prevented due to intensive treatment and guarded prognosis. High inspiratory pressures (> 20cm H_2O) should be avoided by the anesthetist to help reduce the risk of re-expansion pulmonary edema and rupture of the pulmonary parenchyma, leading to intrapulmonary hemorrhage and pneumothorax post-operatively.[8] Also, hypoventilation or hypoxia due to pain, pneumothorax, hemothorax or tight bandages may occur and should be treated with oxygen administration, adequate analgesia, and gentle aspiration of the thoracostomy tube if in place. In the case of chronic hernias where patients experience a loss of abdominal domain, abdominal compartment syndrome due to increased intraperitoneal pressures may occur. To prevent or catch such complications early,

monitoring the patient the first 24 to 48 hours out from surgery is recommended. The patient's vital signs, perfusion parameters, respiratory patterns and pain should be assessed at least every four hours until the patient is stable.

Case outcome:

Jolly's vitals were within normal limits and he was bright, alert and responsive when presenting to the MSU-CVM Surgery Department. His temperature was 99.3 degrees Fahrenheit, pulse was 88 beats per minute, respiratory rate was 33 breaths per minute and his mucous membranes were pink with a capillary refill time of less than 2 seconds. Blood collected for a CBC and neuro chemistry panel the night prior was submitted and the results are as follows. Abnormalities noted on CBC were a high MCV of 86.4fL, with normal ranging between 63.0-77.0, a high segmented neutrophil percentage of 93%, with normal ranging between 60-77, a low lymphocyte percentage of 4%, with normal ranging between 12-30, and a lymphocytopenia of 476, with normal ranging between 1200-6500. Platelet estimate appeared adequate with a count of approximately 368, with few platelet clumping. Red blood cell morphology revealed a slight polychromasia, 1+ pokilocytosis, few/2+ codocytes/ target cells. However, none of these abnormalities on the complete blood count were noted to be significant. Abnormalities on the chemistry were an increased ALT of 321 with normal ranging between 10-90 and an increased CK of 389 with normal ranging between 50-300. Urine was collected for urinalysis and was noted to be dark yellow and cloudy in appearance. Several abnormalities were noted: specific gravity of 1.064, pH of 7.0, 4+ protein, 3+ SSA, 0.2 urobilinogen, large bilirubin, large blood, 1-5 WBC, TNTC RBC, occasional epithelial cells seen, amorphous cells occasionally seen, occasional lipids seen and occasional bile stained sediment.

Based on his chemistry and urinalysis results, intravenous fluid administration of Lactated Ringers Solution (LRS) was begun due to possible muscle trauma and myoglobinuria. He was started on LRS fluids at a rate of 4 mL/kg/hr (41 mL/hr) and continued to receive fluids until the morning after surgery. In order to continue managing pain while in the surgical wards, Jolly received Tylenol 4 (acetaminophen with codeine) at a dose of 1.5 mg/kg per os every 8 hours until surgery. Over the course of the following night, Jolly's respiratory rate steadily increased, approaching the low 60s by the next morning of January 24th. Despite an increased respiratory rate, the rest of his physical exam remained the same as the day prior, with decreased heart and lung sounds auscultated on the left side of the thorax.

Jolly was taken to surgery and placed under general anesthesia in dorsal recumbency. A midline celiotomy incision was made and the faliciform fat was removed with grounded monopolar electrocautery. The defect in Jolly's diaphragm was visualized and two tears were identified: a radial tear was located just prior to the caudal vena caval hiatus and a circumferential tear was located from the xiphoid to the mid-lateral on the right. Multiple loops of small bowel, all lobes of the liver, the entire spleen and stomach were removed from the thorax using gentle traction breaking down minimal adhesions. To have complete visualization of the diaphragmatic defects, a stomach tube was passed to remove food, fluid and air from the stomach. The defects in the diaphragm were repaired using 3-0 Maxon in simple continuous suture patterns beginning at the most dorsal defects continuing ventrally. Thorough examination of the abdominal organs revealed a bruised right kidney, but no other abnormalities or bruising of any other abdominal organs. A three way stop cock and 30 mL syringe were attached to an 8Fr red rubber catheter and used to evacuate air from the thoracic cavity, reestablishing negative pressure and the normal dome shaped anatomy to the diaphragm. Three 30mL syringes of air

were removed from the thoracic cavity to re-establish negative pressure. The abdomen was flushed with 2L of warm, sterile saline and the incision was closed in a 3-layer fashion. Recovery from surgery and anesthesia was uneventful.

Jolly continued to remain on LRS fluids post-operatively after identifying a bruised kidney during surgery. Hydromorphone (0.05 mg/kg IV q4h) was used to control any post-operative pain. His incision was iced every 6 hours to control inflammation and swelling and was monitored for any discharge. SpO2 measurements were within normal limits every four hours post-operatively. Jolly's vitals remained within normal limits the night after surgery and the following morning with a respiratory rate of 20 while resting in his kennel. No pain was elicited upon palpation of his abdomen the morning after surgery and his surgical incision was dry and uninflamed; thus, he was switch back to oral pain medications, Tylenol 4 (acetaminophen with codeine) at 1.5 mg/kg PO q8h, and was removed from ICU. By the afternoon, Jolly seemed to remain comfortable on oral pain management and respiratory rate and SpO2 were still noted within normal limits. Jolly was discharged and sent home on an additional 10 days of pain control. Incision care, ice and warm packing the incision, activity restriction for the next two weeks within a cage or small room, wearing an E-collar for the next two weeks and how to monitor his respiratory rate were all recommended to the owner at the time of discharge. It was emphasized that should Jolly's respiratory rate increase to over 50 breaths per minute while at rest, or his incision become open, oozing with discharge or painful to return to MSU-CVM. To assist in Jolly remaining calm while on cage rest, Trazadone at 5 mg/kg PO q8-q12 for 14 days was sent home with his owners. Trazadone is a serotonin antagonist reuptake inhibitor (SARI) used for behavior modification of dogs who suffer from separation anxiety and other anxiety

related conditions. Since surgery, Jolly has been a much “calmer, well mannered” dog; however, he has yet to be neutered and continues to enjoy free reign of the farm.

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