When You Hear Hoofbeats

A Case Report

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Abstract

There are many possible causes of colic in equine patients, ranging from transient issues that can be managed medically to those that require surgical correction. This case report discusses a warmblood mare that was presented for a suspected strangulating gastrointestinal lesion, but died while being prepared for anesthesia. Upon necropsy, a traumatic diaphragmatic herniation of the spleen and stomach was found. Herniations of gastrointestinal tract through the diaphragm are uncommon, but can occur due to congenital malformations or trauma. Prognosis is considered to be guarded, although case reports have shown surgical repair and subsequent return to performance is possible.

Introduction

Contrary to small and companion animal medicine reports, diaphragmatic hernias in the equine species are uncommon, although not exceedingly rare. Patients often present with clinical signs of colic, with a markedly elevated respiratory rate and decreased gastrointestinal sounds or gastrointestinal sounds within the thoracic cavity (cranioventral borborygmi). Patients may present with acute signs of abdominal pain that is not responsive to analgesic therapy (Cheramie & Moll, 1999). Cyanotic mucous membranes may also help to identify a herniation of gastrointestinal viscera into the thoracic cavity. The presence of GI contents within the thorax prevents the lungs from fully expanding, therefore preventing normal ventilation and resulting in decreased oxygen consumption(Hart & Brown, 2009). Simultaneous disease processes, such as PPID, may also predispose patients to an increased risk of diaphragmatic hernia (Shepard, Lee, & Eggleston, 2015).

Diaphragmatic hernias generally have a poor prognosis. In a case study that looked at over forty horses admitted for surgical repair of diaphragmatic hernias, prognosis was improved by age (<2 years) and the amount of damage to the viscera. Generally, a more favorable prognosis was given if the horse had a normal peritoneal and systemic lactate (Hart & Brown, 2009). Diaphragmatic herniations in adult horses are generally due to trauma, or an increase in repeated microtrauma (Rocken, Mosel, Barske, & Witte, 2013).

Case Summary

On the evening of April 10th 2019 , a 19 year old warmblood mare, presented to her regular veterinarian after exhibiting signs of colic. This mare had a history of colic episodes, and was seen by MSU-CVM Equine Department on June 4th, 2016 for a small colon impaction and nephrosplenic entrapment. She underwent surgical correction for both and recovered without complication. Upon

arrival to her referring DVM (rDVM), the mare was reported to have pale mucous membranes, an elevated heart rate, and no gastrointestinal sounds could be auscultated. A nasogastric tube was placed at that time, and no net reflux was obtained. The rDVM performed a rectal palpation and suspected a right dorsal displacement of the large colon was noted. Bloodwork revealed a packed cell volume (PCV) of 32%, a total protein value of 6.4 g/dL, and a blood lactate value of 1.2 mmol/dL. Peritoneal fluid obtained via abdominocentesis resulted in a total protein value of 3.9 g/dL, and a blood lactate value of 1.2 mmol/L. The mare was then transferred to Mississippi State College of Veterinary Medicine Equine Department for further evaluation.

Upon arrival to MSU-CVM Equine Department, the mare's condition had become significantly more severe. She was noted to have generalized muscle fasciculations, and a heart rate and respiratory rate exceeding 80 beats per minute. A full physical exam was not able to be performed due to her temperament and level of discomfort. Xylazine and buscopan were administered and a rectal exam was attempted, but not completed due to patient discomfort. The large colon was markedly distended. Ultrasound examination was also abbreviated due to her severe discomfort. The left kidney was not visualized and an abnormal tenial band was located ventrally on the left side with subjectively dilated vessels. Blood work was rechecked, and a systemic lactate value of 8.1mmol/dL(rr <2 mmol/dL) was measured. 2 liters of hypertonic saline(4ml/kg) were administered intravenously as a bolus, and surgery was elected. While preparing for surgery, the mare suddenly became belligerent and began to hyperextend her forelimbs and thrash in her stall before falling over backwards and hitting her head and limbs against the walls repeatedly. She died shortly after, and was submitted for necropsy and cremation on April 11th, 2019.

Necropsy Findings

On gross exam, hemorrhage was present from both nostrils as well as the left ear. Additionally, the abdomen was distended. Once the skin on the thorax and abdomen was reflected back, there were areas of red to dark red discoloration of the muscle and subcutaneous tissues over the cranioventral right thorax (4cm by 4cm), lateral to midline on the right side of the abdomen, and cranial to the coxofemoral joint (10cm by 12cm). On examination of the thoracic cavity, the lungs were collapsed and diffusely mottled dark red to pink. Fibrinous tags were present on the surface of the lungs, and dark fluid oozed on cut surface in the most craniodorsal lung fields. Within the trachea there was red tinged foam, as well as 1cm diameter paintbrush hemorrhages located on each trachea ring from the proximal third extending to the tracheal bifurcation. Dilated vessels were present along the septum of the epicardium, in addition to several petechial hemorrhages.

The stomach and spleen were also present in the thorax, along with feed material, most likely due to post mortem rupture of the stomach wall. Paint brush hemorrhage was seen along the greater curvature of the stomach, where there was separation and stretching of the serosal layer. The stomach wall contained a large amount of clear to yellow gelatinous material within the squamous portion. All vessels along the stomach were dilated, and most severely at the junction with the diaphragm. Within the stomach, multifocal erosions were present along the margo plicatus, within the non-glandular portion of the stomach. These erosions were tinged dark red to black, indicating hemorrhage. The diaphragm itself contained diffuse dark red linear discoloration, as well as hemorrhage throughout the musculature and tendinous portion where a rent of approximately 10cm by 10cm was found at the fibromuscular junction, allowing herniation of the stomach and spleen into the thorax. There was apparent splenomegaly due to severe venous congestion with petechial hemorrhages present over the entire splenic capsule. Within the abdomen, there was abundant red tinged fluid. The cecum was dorsocranially displaced with a 180 degrees torsion of the colon. The small bowel was caudally

displaced, and there was petechia evident throughout. The mesenteric and omental vessels were markedly dilated. A segment of the small bowel was entrapped behind the spleen. Dark red to black coalescing areas were present throughout segments of the small bowel, varying in size with the largest measuring 6cm by 1.5cm. Throughout the jejunum there was ecchymosis along the mucosal surface. Both kidneys had notable multifocal to coalescing tan to white discolorations throughout their capsules. The left kidney contains are 1.5cm by 1.5cm firm tan nodule on the surface of the cortex.

The nasal septum was diffusely dark red to purple, with diffusely dilated vessels. The nasal turbinates' and nasopharynx contained multifocal to coalescing paintbrush hemorrhages. Within the brain there were bilaterally located soft brown masses within the lateral ventricles, approximately 1cm by 0.5cm each.

Pathophysiology

The occurrence of diaphragmatic hernias can be due to either congenital defect or trauma. Congenital abnormalities are often diagnosed early in life. Surgical correction in neonates is more successful due to improved visualization and access to the diaphragm. Congenital diaphragmatic hernias are thought to be the result of incomplete fusion of the pleuroperitoneal folds or failure of one of the folds to fuse with the septum transversum (Hart & Brown, 2009). Despite the presence of a congenital lesion, horses may escape adverse effects for many years, rather than being identified at the neonatal stage (Palmer, 2011). Congenital diaphragm defects can be differentiated from traumatic lesions because their edges are smooth, and lack of hemorrhage or fibrosis. Edges of traumatic hernias tend to be thin with roughened edges. Causes can include increased intrabdominal pressure due to parturition, external trauma, or heavy exercise (Hart & Brown, 2009). Previous studies report numerous configurations of one or more abdominal viscera that can herniate through a diaphragm rent, including liver, stomach, small intestine, large intestine, and spleen. Small defects are associated with herniation

and subsequent strangulation of small intestine, with patients presenting for signs of colic consistent with such. Larger defects that permit herniation of organs are likely to lead to signs of respiratory distress as well as abdominal pain (Romero & Rodgerson, 2010).

Although the defect may be severe, diagnosis of a diaphragmatic herniation in equine patients is often challenging and requires multiple modalities for confirmation. Patients can present with mild to severe signs of colic (abdominal pain). An initial physical examination, followed by use of imaging modalities such as ultrasound or radiographs may confirm suspected herniations. Auscultation of gastrointestinal sounds within the thorax may trigger the need for additional diagnostics. although ultrasonography is generally considered to be a common part of an initial workup when a horse is presented for colic, especially at referral institutions (Clark, Arighi, Jamison, Pascoe, & Johnson, 1987). Even with a complete colic workup, it may not be possible to identify a defect within the diaphragm due to the location of the rent and herniated viscera. For example, aerated lungs may prevent visualization of abdominal organs within the thorax. Because clinical signs associated with diaphragmatic herniation are often similar or identical to that of a gastrointestinal strangulation, patients are often taken to surgery without extensive imaging, as pain control cannot be achieved. An exploratory laparotomy that includes evaluation of the diaphragm is often critical in diagnosing and repairing diaphragm lesions (Hart & Brown, 2009) (Hartzband, Kerr, & Morris, 1990).

Differential Diagnoses

Horses that are ultimately diagnosed with a diaphragmatic hernia, either through an exploratory laparotomy or necropsy evaluation, generally present with signs of colic. Initial physical examination usually shows an elevated heart rate and respiratory rate, as well as a high level of discomfort that may not be well controlled with analgesia. These clinical signs are also consistent with strangulating lesions

within the gastrointestinal tract, such as a torsion, entrapment of Small Bowel, or a strangulating lipoma (Cheramie & Moll, 1999) (Romero & Rodgerson, 2010).

Absence of normal gastrointestinal sounds within the abdomen may also be due to infectious causes of illeus such as colitis, or enteritis (Abutarbush, Carmalt, & Shoemaker, 2005). An increased respiratory rate an abnormal lung sounds may be due to infectious causes as well, or due to trauma to the thorax leading to poor lung expansion and cyanosis (Boy & Sweeney, 2000).

Diagnostic Approach/Considerations

Evaluation of patient history and clinical picture provide important clues to causes of abdominal discomfort in the equine patient. Length of time that clinical signs have been exhibited, as well as past history of episodes of colic may help to identify cause (Hart & Brown, 2009). Use of ultrasound to view structures within the thorax and abdomen may help to evaluate location of structures and gastric motility. In addition, the use of radiographic techniques such as thoracic and abdominal radiographs and, less commonly, contrast radiography may be helpful in diagnosis of a diaphragmatic defect in patients stable enough for imaging (Hartzband, Kerr, & Morris, 1990). Ultimately, a majority of diaphragmatic defects are identified during exploratory laparotomy, as patients present with severe signs of colic similar to strangulating gastrointestinal lesions, or during post mortem examination (Hart & Brown, 2009).

Treatment and Management Options

Although medical and surgical management are both possible methods for management of diaphragmatic hernias, surgical approaches to repair the defect is preferred when possible. Case reports and small-scale retrospective studies have suggested that surgical correction of the defect is possible,

and can result in a favorable outcome depending on the signalment and concurrent disease processes for each particular patient (Clark, Arighi, Jamison, Pascoe, & Johnson, 1987).

Congenital defects found in foals tend to lend themselves to surgical correction and recovery due to the size of the patient and a lack of concurrent defects. Surgical correction within adult horses is challenging due to decreased to no visibility of the defect (Palmer, 2011). Access can be improved using a laparoscopic approach and rib resection in adult horses (Malone, Farnsworth, Lennox, Tomlinson, & Sage, 2004) . The size of the defect can also impact repair, and mesh is often required to repair defects due to a lack of available tissue for closure. Diaphragmatic rents that are dorsally located may be repaired using blind stapling of polypropylene mesh, while ventral rents may be repaired by suturing the defect using a simple continuous pattern, or suturing a mesh over the defect (Hart & Brown, 2009).

Laparoscopic approach can be utilized to locate and repair a rent within the diaphragm. In chronic cases where the herniation of abdominal contents has occurred due to a congenital abnormality, polypropylene mesh can be used to close the rent (Dabareiner & White, 1999). Laparoscopic approaches have been utilized in colic surgery as well, making this favorable when performing an exploratory when a definitive cause of abdominal pain cannot be determined, and a diaphragmatic hernia cannot be ruled out (Mason, Johnston, Wallace, & Christie, 1970).

Conservative management consisting of administration of flunixin meglumine, nasogastric intubation and administration of mineral oil, and a dietary modification to entirely pelleted feed, can resolve clinical signs in some cases (Pauwels, et al., 2007).

Expected Outcome and Prognosis

As mentioned before, due to involvement of abdominal structures, patients with diaphragmatic defects are generally presented for colic. Clinical signs associated with horses ultimately diagnosed with a diaphragmatic hernia include tachypnea, dyspnea, exercise intolerance and lethargy (Hart & Brown, 2009). Rectal palpation, imaging, and auscultation (revealing cranioventral borborygmi) may assist with ruling out impaction, torsion, or displacement. Patient pain level must be considered when reviewing options, as an abdominal exploratory via ventral midline celiotomy may be the most reliable method of determining the cause of clinical signs.

Overall, diagnosis of a diaphragmatic hernia in an equine patient carries a poor prognosis, with on overall survival rate of 23%, and a surgical success rate of 46%(Romero & Rodgerson, 2010). In cases where diaphragmatic hernia is identified, the evaluation of peritoneal fluid obtained prior to surgery appears to be the most informative with regard to prognosis. Abnormal peritoneal fluid has been associated with a guarded prognosis. Improved survival rates have also been associated with an age of less than 2 years, the amount of compromised viscera, and the size of the defect (less than 10 cm). Prognosis for horses with a diaphragmatic hernia is poor, but lesions that can be surgically repaired do have a fair prognosis for long term survival (Hart & Brown, 2009).

Sources

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