“Between a Rock and a Hard Place”

Visceral Rupture Caused by Enterolith Formation in the Equine Patient

Melody G Sharp

Mississippi State University College of Veterinary Medicine

Class of 2019

Clinicopathological Conference

Presented on July 20th, 2018

CPC Advisor:

Cathleen Mochal-King, DVM, MS, Diplomate ACVS-LA

Associate Professor, Equine Surgery Department of Clinical Sciences

Introduction:

A 9-year-old Tennessee Walking Horse gelding presented to the Mississippi State University College of Veterinary Medicine's Equine Service on May 19th, 2017 for severe abdominal pain. The gelding had previously been examined by a referring veterinarian for an approximately 1 week history of intermittent colic. His colic signs were suspected to be due to gastric ulcerations.

History and Presentation:

The gelding present to the Emergency Equine Service on May 19th, 2017. Having been treated for approximately 1 week's duration with Gastroguard by the referring veterinarian for suspected gastric ulcerations. At approximately 5 pm on May 19th, the gelding resumed having colic related symptoms and was given 10 mls (500 mg or 1 mg/kg) of flunixin meglumine intravenously. At 6 pm, he was examined by the referring veterinarian for worsening severity of colic signs. His heart rate was 70 bpm with severe abdominal distention giving abloated appearance. Upon rectal examination demonstratedcolonic distension. He was described to have "muddy" mucous membranes. Nasogastric intubation yielded 0 liters of net gastric reflux. ½ gallon of mineral oil and 2 gallons of water was administered via the nasogastric tubing. He was then referred to the MSU-CVM Equine Emergency Service due to severity of pain, in conjunction with a surgical option, and was transported with an NG tube in place. He received 0.5 mL intravenous detomidine just prior to transport. Upon presentation, the gelding had removed his nasogastric tube in the trailer. He had generalized ataxia causing ambulation to be

difficult. He appeared to have stranguria but was anuric during these episodes. He was attempting to lie down constantly. Physical examination revealed a heart rate of 68 bpm, and his temperature was too hypothermic for the thermometer to register (<96 °F). Mucous membranes demonstrated a toxic line and were dark purple in color along with his tongue. Upon auscultation of the abdomen, gastrointestinal borborygmi were decreased in all four quadrants.

Diagnostic Approach:

Upon initial rectal examination, a moderate amount of gas was felt in the dorsal colon. The examination had to be discontinued due to the gelding's discomfort level. Subsequent rectal examination revealed "empty" colonic structures and was not within normal palpable limits.

Blood was collected from the jugular vein to be used to measure a systemic lactate, total protein, and a packed cell volume. The gelding's systemic lactate was revealed to be 14.1 mmol/L(reference range 1-2 mmol/L). A packed cell volume was measured to be 47% (normal reference 30-40%). A serum total protein was measured to be 5.7 mg/dL.

A nasogastric tube was reinserted to determine if the gelding was actively refluxing. No spontaneous reflux was obtained during the horse's time in clinic. Following intubation, an abdominal ultrasound was performed. Abdominal ultrasound revealed various dilated loops of small intestine and a large volumeof free fluid in the abdominal cavity.

An abdominocentesis was performed. Approximately 1 mL of green-brown colored, malodorous fluid and sediment were obtained on abdominocentesis, suggestive of gastrointestinal perforation. Lactate reading was unmeasurably low; it was suspected that this low reading was due to dilution occurring from a gastrointestinal rupture.

Pathophysiology: Visceral Ruptures/ Peritonitis

One of the major causes of peritonitis is visceral rupture. One of the main causes of visceral rupture is intestinal obstruction. Intestinal obstructions result from movement of gastrointestinal contents being blocked. Once the gastrointestinal contents are non-motile, the intestine distends. As the intestine distends, the venous outflow from the area is compromised causing mucosal injury and ischemia which weakens the wall of the intestine allowing for a rupture to occur. This leads to gastrointestinal contents and bacteria being released into the peritoneal cavity leading to peritonitis. (2)

Peritonitis in the horse is most commonly presented as septic peritonitis involving pathogens from the gastrointestinal tract. Some of the most common causes of septic peritonitis include trauma to the gastrointestinal tract including surgical trauma, diagnostic testing induced trauma, or visceral perforation as seen in this case. Visceral perforation results in a mixed bacterial population polluting the peritoneal cavity. Due to this contamination, catecholamines, histamine, and serotonin are released from the peritoneal mast cells resulting in vasodilation and increased permeability of the peritoneal vasculature. Due to this increased permeability, protein-rich fluid along with macrophages and other inflammatory cells are released into the peritoneal cavity. This mechanism conversely allows for bacteria and endotoxin to be absorbed into the blood stream. The body reacts to these effects by decreasing the fibrinolytic activity of the peritoneum and allowing fibrin to deposit in an attempt to confine the insult. The macrophages release inflammatory mediators to react to and control the bacterial LPS. As a result of all of these mechanisms, endotoxic shock due to septic peritonitis ensues. (1)

Septic peritonitis, in particular due to visceral rupture, carries a grave prognosis. Septic peritonitis resulting from abdominal surgery without visceral rupture carries a 56% fatality rate. In non-complicated cases of peritonitis, prognosis can be up to a 78% survival rate. (1) If surgery

and lavage is attempted after a colon rupture and after septic peritonitis has developed, chances of recovery have still been shown to be minimal. (3)

Case Outcome/ Necropsy Findings:

Due to the severity of disease and the poor prognosis of intestinal rupture, the owners elected to humanely euthanize the gelding. He was then sent for a necropsy to determine the extent of his disease.

The official necropsy diagnosis was enterolithiasis with resulting large colon obstruction and rupture with septic peritonitis. The oral mucous membranes were pale with a slightly pink rim around the incisors. Abundant red tinged fluid exuded from the nares. When the abdominal cavity was opened, approximately 5-10 liters of fetid dark brown green fluid mixed with ingesta was within the abdominal cavity. The abdominal cavity was also distended with air. The serosal surface of the abdominal organs was covered by a thick mat of feed material mixed with fibrin. The serosa was discolored dark red over most of the intestinal tract. A 4 cm in diameter, full thickness tear with jagged, hemorrhagic edges, was present in the right dorsal colon. The right dorsal colon in the dorsal colon proximal to the diaphragmatic flexure contained two large irregularly round mineralized enteroliths that 13 and 14 cm in diameter and weighed 1.82 and 2.18 kg, respectively that obstructed the movement of ingesta from the large colon to the small colon, resulting in rupture of the right dorsal colon. The lungs showed evidence of pulmonary edema. All other organs were within normal limits.

Pathophysiology: Enteroliths

Enteroliths are formed in the intestines when a nidus, such as hair, pebbles, or other foreign material, have concentric rings of minerals laid around it forming a tetrahedral or

spherical shape. They range in size from a few ounces to multiple pounds. The majority of enteroliths are formed of struvite, or magnesium ammonium phosphate. They most commonly are formed in the transverse or dorsal colons. (4) Enteroliths often result in frequent, mild episodes of colic due to the typical presentation of only partial blockage of the intestines. (6)

Research has found that a few factors predispose horses to enterolith formation. Alfalfa hay has been associated with higher incidence of these intestinal stones. Feeding alfalfa allows for an excess of magnesium, nitrogen, and calcium in the diet. The magnesium content of alfalfa hay appears to be higher in alfalfa hay grown in California than the rest of the continental US. The excess dietary calcium in the alfalfa fed diet allows for a more alkaline environment of the colon. In an alkaline environment, struvite more readily crystallizes forming the concentric rings around the inciting nidus. (4) It has also been suggested that Arabians, Morgans, American Saddlebreds, and miniature horses have a higher risk of developing enterolithiasis, though the reasoning is unknown. (5,6)

The prognosis for non-perforating cases has been shown to be approximately 92%. Colonic rupture has been estimated to appear in approximately 15% of cases. If this happens, septic peritonitis ensues, and a grave prognosis is given. (5)

Treatment/Management:

The majority of septic peritonitis and intestinal perforation cases result in euthanasia. Though in some cases, owners may elect to attempt treatment. Due to the severity of clinical signs and the condition of the horse, euthanasia was elected in this case.

Treatment for uncomplicated peritonitis cases includes NSAID therapy, intravenous broad spectrum antibiotic therapy, and peritoneal lavage with a drain placed. The most

commonly used antibiotic therapy includes 22,000 units/kg IV every 6 hours of potassium penicillin and gentamicin at 6.6 mg/kg every 24 hours IV. Lavage can be completed with a balanced electrolyte solution such as Lactate Ringer's Solution in a volume of around 10 to 20 liters. This lavage is usually repeated multiple times daily for approximately 5 days. If visceral perforation is suspected and treatment is a reasonable option, surgical exploration must be used to correct the primary cause of the peritonitis. (1)

The majority of enterolith cases require surgical removal, though some have been reported to be passed in feces or have been removed rectally. (5) It has also been suggested that non-obstructing enteroliths can be attempted to be treated by dissolution. A study at Cornell indicated that feeding apple cider vinegar and feeding a higher proportion of grain can lower the colon pH to lower than 6.5 to allow for dissolution of the struvite crystals forming the intestinal stone. (7) If the horse is experiencing mild to severe colic symptoms and enteroliths are suspected, surgery is indicated to prevent perforation. Surgery usually includes a ventral celiotomy and enterotomy at the right dorsal colon. (5) If a tetrahedral enterolith is found, multiple enteroliths are typically indicated and further surgical exploration is necessary. (4)

References:

1. Reed, S. M., Bayly, W. M., & Sellon, D. C. (2018). *Equine Internal Medicine*(4th ed., p. 762-765). St. Louis, MO: Elsevier.
2. Orsini, J. A., & Divers, T. J. (2014). *Equine emergencies: Treatment and procedures*(4th ed., p. 186). St. Louis: Elsevier.
3. Sprayberry, K. A., & Robinson, N. E. (2015). *Current Therapy in Equine Medicine*(6th ed., p. 410). St. Louis, MO: Elsevier/Saunders.
4. Bray, R. E. (1995). Enteroliths: Feeding and management recommendations. *Journal of Equine Veterinary Science,15*(11), 474-478. doi:10.1016/s0737-0806(06)81820-4
5. Reed, S. M., Bayly, W. M., & Sellon, D. C. (2018). *Equine Internal Medicine*(4th ed., p. 889). St. Louis, MO: Elsevier.
6. Constable, P. D., Done, S., Grüenberg, W., Hinchcliff, K. W., & Radostis, O. M. (2017). *Veterinary medicine: A textbook of the diseases of cattle, horses, sheep, pigs, and goats* (11th ed., Vol. 1, p. 265-266).
7. Hassel, D. M., Rakestraw, P. C., Gardner, I. A., Spier, S. J., & Snyder, J. R. (2004). Dietary Risk Factors and Colonic pH and Mineral Concentrations in Horses with Enterolithiasis. *Journal of Veterinary Internal Medicine,18*(3), 346-349. doi:10.1111/j.1939-1676.2004.tb02556.x