Colic Ain't Cheap

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Introduction

Equine colic describes a wide range of functional, anatomical, and physiologic derangements that cause abdominal pain. In many cases, these abnormalities lead to systemic dysfunction in both the cardiovascular and metabolic systems and can lead to fatal endotoxemia and sepsis[6]. Colic signs range from mild to severe, and may include pawing at the ground, rolling, laying down and rising repeatedly, straining to urinate or defecate, and kicking at the flank. Anterior enteritis in the horse is characterized by inflammation of the small intestine—most frequently the duodenum or the proximal jejunum[6]. Although the exact cause is unknown, several infectious etiologies have been proposed. Anterior enteritis may also be incited or complicated by abrupt changes in the patient's diet[3]. Regardless of etiology, anterior enteritis results in functional ileus and small intestinal distention coupled with large amounts of gastric reflux. Diagnosis is multifactorial and varied, and anterior enteritis may present a unique diagnostic "rule-out" challenge. Hallmarks of anterior enteritis include mild to moderate colic with considerable relief after analgesic medications and gastric decompression via nasogastric intubation. Once a diagnosis is made, treatment is largely supportive unless the intestine becomes necrotic, necessitating exploratory surgery. Prognosis is variable but in many cases fair to excellent, with horses that are managed medically more successful[6]. Prevention is aimed at altering daily management strategies for the patient.

History and Presentation

Wheeling Nu Cash, "Cash", is a 10-year-old chestnut quarter horse mare who is used as a teenager's reining and 4-H horse. She presented to MSU CVM once previously for colic in 2018, was diagnosed with flatulent colic, and was successfully managed with medical therapy. On July 28, 2019, she was fed her normal grain ration around 6pm and ate normally. She is turned out

during the day on 7 acres of pasture with herd mates and is stalled at night. When checked later that evening around 11pm, Cash was exhibiting moderate pain. She presented to MSU CVM Equine Emergency service at 11:30 pm that same night. On presentation, Cash was depressed but responsive. She weighed 1140 pounds and was a body condition score of a 6/9. She was loaded into the stocks and a thorough physical exam was conducted. On physical exam, Cash had a normal heart rate at 44 beats per minute, had a normal respiratory rate of 14 breaths per minute, and had a slightly low temperature of 98.3 degrees Fahrenheit. No crackles or wheezes were appreciated when auscultating the lungs, and no murmurs or arrhythmias were noted on cardiac auscultation. She appeared mildly dehydrated with pink, moist mucous membranes, and a capillary refill time of 3 seconds. Her gut sounds were hypomotile in all four quadrants and her abdomen was tense on palpation. She had none-to-slight digital pulses in all four limbs. The remainder of her physical examination was within normal limits.

Diagnostic Approach

Most patients experiencing colic present on emergency, and an initial diagnostic workup is often standardized for these patients. This should include a thorough physical examination on arrival unless the patient is unable to be handled safely, sedation if the patient is fractious and to assess its effects on pain level, rectal examination with extreme care taken for patient safety, nasogastric tube placement or evaluation if the patient arrives with one in place, and ultrasound examination of the abdomen. Additional diagnostics should also include a minimum database of a complete blood count and a chemistry panel, making sure to include a blood lactate.

Once the patient has been sedated, nasogastric intubation is a critical first step to facilitate gastric reflux and decompression, as gastric rupture is possible with enough fluid accumulation. The quantity and quality of this reflux also presents some diagnostic differentiation between a

large intestinal and small intestinal lesion. Commonly, patients with anterior enteritis will produce large amounts of red- to orange-brown liquid. The act of gastric decompression also frequently decreases or abolishes colic signs, but patients remain depressed or quiet[5]. Manual rectal palpation is a mainstay of the colic diagnostic workup, but care should be taken to avoid rectal tears. This gives the clinician a better idea of the possibility of large intestinal displacement or an impaction at the cecum or pelvic flexure.

Ultrasonographic examination is useful for determining small intestinal wall thickness, assessment for motility, or ileus, evaluating the nephrosplenic space for possible intestinal entrapment and evaluating the stomach for reflux accumulation[7]. It is also useful for identifying a site that is ideal for abdominocentesis.

Cash was sedated with xylazine at 0.3 mg/kg, which made her more comfortable and allowed for further diagnostics. A nasogastric tube was placed, and a net of approximately 31 liters of brown, putrid gastric reflux containing feed material was recovered. A jugular catheter was placed, and blood was drawn for a complete blood count and a chemistry panel. The CBC revealed a mild neutrophilia of 8,208/ul (2,500-6,000). The chemistry panel revealed a severely elevated AST of 1061 U/L (134-406), a mild hyperglobulinemia of 4.2 g/dl (2.5-4.0), and a blood lactate of 0.9 mg/dl (<2). Cash's ventral abdomen was sterilely prepped with chlorhexidine scrub and alcohol and a bitch catheter was used to facilitate abdominocentesis. The fluid analysis revealed a red blood cell count of 245/ul, a nucleated cell count of 300/ul, a protein of <2.0 g/dl, and a lactate of 1.9 mg/dl. Buscopan was administered at 0.2 mg/kg before rectal palpation. The rectal exam revealed large colon distention which limited thorough palpation. A large, viscous-like structure was palpated on the right side of the caudal abdomen. The left kidney and nephrosplenic space were not able to be palpated as the left caudal abdomen was taken up largely

by gas filled large colon. No loops of small intestine were palpated. Due to the inability to feel the kidney or nephrosplenic space, a left dorsal displacement was a differential diagnosis. Abdominal ultrasound showed loops of distended small intestine between the spleen and body wall and decreased duodenal motility with a thickened wall.

Repeated diagnostics later that morning and early afternoon included a complete blood count and chemistry panel, rectal examination, and abdominal ultrasound. The second rectal exam, performed after initial fluid replacement and reflux, revealed minimal large colon gas distention, a palpable nephrosplenic space, and no large viscous in the right caudal abdomen like in the first exam. A repeat abdominal ultrasound showed that the left kidney and spleen were able to be pictured in the same frame, with no small intestine noted between the spleen and body wall. There was increased motility compared to the previous ultrasound, but the duodenal wall was thickened at 11.8mm. Cecal and colon wall thickness were within normal limits. The CBC showed an increased but still mild neutrophilia of 9592/ul (2500-6000) and a mild lymphopenia of 1199/ul (1250-5000). The chemistry panel showed mild hyperchloremia of 108.1 mmol/L (98.0-106.0), a moderately increased AST of 949 U/L (134-406), mild hypophosphatemia of 2.1 mg/dl (2.4-4.0), and moderate hypomagnesemia of 0.9 mg/dl (1.6-2.5).

Differential Diagnoses

Colic cases may be differentiated into two categories: surgical and medical. Determining what category into which a patient falls can be a considerable diagnostic challenge. Using the diagnostics above and evaluating each helps paint a clinical picture. There are several parameters which may be evaluated to determine if surgery is necessary: pain level, including colic signs, heart rate, and patient attitude, reflux quality and quantity over several sessions, minimum

database results, ratio between systemic and fluid lactate, abdominal fluid analysis, serial rectal exam results, and ultrasound findings[7].

Initially, Cash presented in moderate pain with sweating and a tense abdomen. She had a mildly increased heart rate in response to pain, but much of this was able to be mitigated with analgesia and sedation. Upon nasogastric intubation, she had a large amount of putrid reflux, and this gastric decompression further decreased her pain level. Though her bloodwork did reveal some abnormalities, most notable were her elevated AST, a non-specific enzyme in muscle and liver tissues that is often elevated in anterior enteritis due to ascending cholangiohepatitis from duodenitis[2]. Her initial fluid analysis was unremarkable, with low concentrations of RBCs and nucleated cells, and a protein level within normal limits. Her blood lactate was normal at 0.9 mg/dl, but her fluid lactate of 1.9 mg/dl was suggestive of a non-obstructive small intestinal lesion, peritonitis, anterior enteritis, or colitis[4]. Her first rectal exam gave suspicion of nephrosplenic entrapment or other left dorsal displacement due to the inability to palpate the nephrosplenic space or left kidney. Her first abdominal ultrasound also furthered that suspicion.

Repeat diagnostics after anti-inflammatory medication and bolus fluid therapy painted a different picture. Cash's pain had greatly diminished, but her depression remained. Repeat bloodwork showed components of a stress leukogram, mild electrolyte abnormalities, and a decreased AST from the first chemistry panel. Her second rectal exam showed improvement in large colon distention and a palpable nephrosplenic space, and her repeat ultrasound supported that as well. It also revealed increased small intestinal motility and a moderately thickened duodenum. As a result of serial diagnostics and an evolving clinical picture, Cash was diagnosed with proximal enteritis.

Pathophysiology

Proximal enteritis, also known as anterior enteritis and duodenitis-proximal jejunitis (DPJ), is an inflammatory condition of the small intestines that causes mild to moderate colic signs, significant gastric reflux, and ileus[2]. Though a clear etiology has not been established, there are many proposed, including *Salmonella spp.,* mycotoxins, *Clostridium spp.*—notably *C. difficile*—with a possible link to certain feeding practices[3]. This inflammatory process leads to increased small intestinal mucosal permeability, allowing bacteria to enter the bloodstream and cause toxemia[2]. Inflammation also causes small intestinal distention, fluid buildup, and ileus, which leads to fluid accumulation in the gastric lumen. Ileus described in DPJ may be attributed to *Clostridium spp.* toxins which alter small intestinal motility[2]. This gastric and intestinal distention leads to colic signs, and, without gastric decompression, can cause acute rupture and peritonitis. Electrolytes are lost in this fluid, leading to electrolyte derangements such as decreases in sodium, chloride, potassium, and calcium[2]. If allowed to continue without intervention, hemorrhagic, fibrinonecrotic duodenitis-proximal jejunitis can result[6].

Clostridium difficile has been proposed as a likely etiologic agent in horses clinically affected with DPJ. In one study, six healthy horses were inoculated with *C. difficile* toxins, via gastroscopy, that were isolated from a horse with active DPJ. At the conclusion of the study, 2 of 6 horses showed clinical signs consistent with DPJ, with all 6 showing histologic evidence of DPJ[1]. This study also observed that these toxins have a profound effect on gastrointestinal motility, a hallmark change in horses with DPJ.

Horses that are fed large amounts of concentrates daily may be at a greater risk of developing colic and DPJ or similar enteritis, according to one study[3]. Cases of DPJ were also more likely to be horses that grazed pasture in addition to their concentrate intake. However, as

this study was retrospective, there was no attempt to validate owner history answers about diet, and it is unknown if any recent, sudden diet changes were made in these cases.

Treatment and Management Options

Treatment of DPJ aims to control endotoxemia, gastric decompression, replace fluid deficits, improve motility, and control pain with aggressive supportive care[6]. Often, the first steps of this treatment are taken during the diagnostic workup. Serial gastric decompression, often every 2 hours, with documentation of net reflux ensures that the stomach does not become overly distended, and also may serve as a marker for progression or improvement of disease. Because bacterial infiltration is proposed as a causative agent, broad-spectrum antimicrobials may be necessary to mitigate infection.

Many horses that present for colic and are diagnosed with DPJ are mildly to moderately dehydrated. Intravenous fluid replacement is necessary to correct dehydration, maintain fluid intake, and address ongoing losses (i.e. gastric reflux). Isotonic crystalloids are commonly used, such as Lactated Ringer's solution (LRS), but hypertonic saline may be indicated in shock followed by LRS[6]. In horses with severe hypoproteinemia caused by protein-losing enteropathy may be treated with colloid bolus or, ideally, plasma transfusion. Pain may be addressed with alpha-2 agonists used for sedation, anti-inflammatory medication such as flunixin meglumine or phenylbutazone, opioids such as butorphanol, and serial gastric decompression. Supplemental nutrition may also be indicated in horses that are anorectic for more than 4 days, as a negative energy balance can delay healing and worsen hypoproteinemia[6]. Motility may be improved with use of intravenous lidocaine in a constant rate infusion (CRI). Lidocaine acts as an indirect promotility agent, helps to control cardiac arrhythmias if present, and provides analgesia. In one

study, horses that received lidocaine CRI passed feces significantly sooner and resulted in shorter hospitalization as compared to the placebo group[8].

After Cash's initial diagnostic workup, she was given flunixin meglumine at 1.1 mg/kg IV every 12 hours. She was also given a bolus of 10 L of LRS fluids spiked with 120 ml calcium gluconate and 100 ml of potassium chloride to address electrolyte losses. After the initial bolus, she was maintained at 2 L/hr with 120ml calcium gluconate per 10 L and 400 ml magnesium sulfate per 10 L. She was also started on enrofloxacin at 6.5 mg/kg IV to be given every 24 hours. Later that day, after more diagnostics, she was started on ranitidine at 7.5 mg/kg every 8 hours, sucralfate at 25 mg/kg every 8 hours, and Platinum Balance at two scoops every 12 hours, all to be given orally.

Prognosis

Outcomes for DPJ are variable but uncomplicated cases usually result in a full recovery[6]. Refractory cases that continue to reflux large volumes for days to weeks may be intervened with surgery, which has been associated with less favorable outcomes. In a retrospective study of 120 horses, those treated surgically refluxed a greater volume and for a longer duration than those treated medically. However, there was no difference in surgical versus medical cases in duration or volume of reflux in horses that survived[9]. Negative prognostic indicators are the often severe sequelae of the disease, including uncontrollable endotoxemia, laminitis as a result of sepsis, intestinal adhesions from severe inflammation, peritonitis if there is small intestinal or gastric rupture, or cholangiohepatitis from ascending inflammation from the duodenum into the bile ducts.

Case Outcome

Cash's net loss to reflux for 7/28/19, at presentation, was 31 L. Her net loss on 7/29 was 19 L. On 7/30, it was 20 L, with a steady 2 L every 2 hours overnight and into the early morning of 7/31. The nasogastric tube was pulled at 7:50 am on 7/31. Cash had been maintained on 2 L/hr LRS since the early hours of 7/29 and was decreased to 1 L/hr at 6 pm on 7/30. She continued to receive IV flunixin meglumine every 12 hours until 8/1. Enrofloxacin was given intravenously every 24 hours until 8/1 as well. Her jugular catheter was pulled at 3 pm on 8/2. She was offered hay spread out around the stall starting on 7/31, and 1 lb Purina Equine Senior in a mash. She was meant to receive ranitidine, sucralfate, and Platinum Balance as a slurry offered orally, but that quickly became a problem. She vehemently refused oral medications and her senior mash but drank plain water and ate hay well. After trying to mix her oral medications into her grain with no success, her ranitidine, sucralfate, and Platinum Balance were discontinued the evening of 8/1. Cash was maintained on regular Purina Equine Senior grain and timothy hay for the duration of her stay, was allowed to briefly hand graze every 8 hours from 7/31 on and was discharged on 8/3/19. She has not returned to MSU CVM since.

Conclusion

Duodenitis-proximal jejunitis (DPJ) is an idiopathic inflammatory condition of the small intestines that results in colic signs, ileus, small intestinal distention, and gastric reflux. Diagnosis can be difficult and involves multiple factors including response to sedation and gastric decompression, character of reflux produced, serial rectal examination and ultrasonographic findings, and monitoring throughout the course of disease. DPJ may be treated medically or surgically, and the approach is determined by severity and duration of patient clinical signs. Treatment is largely supportive, and involves aggressive fluid therapy, analgesia,

serial gastric decompression via reflux, antibiotic therapy, and cautious re-feeding. Prognosis is variable, but many horses make a full recovery and continue to work as intended.

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