Intussusception in the Horse

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Introduction:

Intussusception is an uncommon cause of colic in horses, characterized by a portion of bowel (the intussusceptum) telescoping into a more distal segment (the intussuscipiens). It most frequently affects young horses.[1,2,4] There are no breed or sex predilections for the different types of intussusception.[1,2] The etiology of intussusception is not completely understood, but it is believed to be caused by abnormal intestinal peristalsis, intestinal inflammation, dietary changes, colitis or typhlitis, and parasympathomimetic drug administration.[2] Intussusception can occur in several locations throughout the intestinal tract, with the small intestine being the most common location.[4] Ileocecal intussusception is the most common small intestinal intussusception. Other types reported include jejunojejunum, jejunoilium, and ileoileum.[4] Large intestinal intussusceptions can occur as cecocecum, cecocolon, colocolic or colorectum.[2,4]

Cecal intussusceptions result from the cecal apex inverting into the cecal body (cecocecal intussusception) or the cecal apex and body inverting through the cecocolic orifice into the right ventral colon (cecocolic intussusception) and most commonly develop in 2 to 3-year-old horses.[1,2] Cecal intussusceptions are believed to be caused by alterations in intestinal motility and intestinal inflammation.[2,4] Clinical signs are nonspecific and vary depending on the degree of luminal obstruction. Signs can range from acute abdominal pain to a chronic colic that results in weight loss, tachycardia, tachypnea, and eventually vascular compromise.[1,2] In general, the severity of clinical signs decreases with chronicity.[2,4]

Diagnosing small and large intestinal intussusception can be difficult preoperatively, but the patient's history, clinical signs, and physical examination may provide insight to the form of disease and indicate nonspecific colic.[1,4] With cecal intussusceptions, rectal palpation may reveal a painful cecum when retracted in the right dorsal quadrant or the base of the cecum may be

unpalpable due to its relocation into the cranial abdomen.[2,4] An edematous mass (the edematous cecum) in the right caudal aspect of the abdomen may also be noted on rectal palpation.[2] Abdominal ultrasonography may reveal a “target” or “bull’s eye” lesion and is pathognomonic for intussusception.[1,4] The outer intussuscepiens is usually sacculated and separated from the inner thickened (>3 mm), congested, and edematous intussusceptum.[4] Cecocecal intussusceptions are usually located in right ventral abdomen (the apex is invaginated and displaced) and cecocolic intussusceptions can be found in the upper right abdominal quadrant (cecum invaginates into the right ventral colon) with abdominal ultrasound.[1,2,4] Peristalsis in the affected region may be reduced or absent.[2] Definitive diagnosis is achieved with exploratory ventral midline celiotomy or necropsy. On abdominal exploration, the apex of the cecum is absent, and a firm mass is palpable in the base of the cecum or the right ventral colon.[2,4] Characteristic pathologic lesions include adhesions, bowel ischemia and necrosis, and a friable, edematous, hemorrhagic cecum.[4]

Intussusception can only be treated surgically. Manual reduction followed by resection and anastomosis of affected segments of intestine (if necessary) is the treatment of choice for all types of intussusception.[4] Prognosis is highly dependent on the amount of cecum intussuscepted, the amount of necrotic bowel, the amount of surgical contamination, and the surgeon’s technique.[3,4] The following report describes the history, presentation, diagnostics, treatments, and outcome of a 28-year-old Tennessee Walking Horse with a cecocolic intussusception.

History and Presentation:

Cody, a 28-year-old Tennessee Walking Horse gelding, presented to MSU-CVM Equine Services on February 22, 2018 for colic of seven days duration. On February 17, the first day he began showing signs of colic, Cody was administered flunixin meglumine intravenously by his

referring veterinarian, then every six hours intramuscularly by his owner for the next two days (February19). Cody's colic signs resolved, so his owner discontinued the flunixin meglumine until Wednesday (February 21), when he stopped eating grain. His referring veterinarian administered flunixin meglumine intravenously once again along with one 1,000 pound dose of oral flunixin meglumine paste. The paste was given every six hours until Thursday afternoon. By Thursday, Cody was inappetent and was seen pawing, flank watching, and rolling.

Upon presentation to MSU-CVM on February 22, 2018, Cody was bright, alert, and responsive. He had a body condition score of 3/9 (5 being ideal) and weighed 840 pounds. He had a rectal temperature of 101.4 degrees Fahrenheit (98.0-101.5 degrees Fahrenheit), a heart rate of 60 beats per minute (28-42 beats per minute), and a respiratory rate of 60 breaths per minute (20-40 breaths per minute). His mucous membranes were pink with a capillary refill time of less than 2 seconds. No abnormalities were noted on auscultation of his heart and lungs. Normal borborygmic sounds were present in all 4 quadrants. His digital pulses were none to slight in intensity. Ocular discharge was present bilaterally. All other physical exam findings were within normal limits.

Pathophysiology:

Cecocolic intussusception is a relatively uncommon cause of colic in horses that involves the apex of the cecum telescoping into the body of the cecum, then the cecum invaginating through the cecocolic orifice into the lumen of the right ventral colon.[3,4] The exact cause is unknown, but it is most likely caused by the presence of inflammatory lesions that result from chronic NSAID use, *Anaplocephalata perfoliata* infestation, administration of organophosphates and parasympathomimetic drugs, etc.[2,3,4,5] The proximal end (the cecum) is the intussusceptum and the distal portion (the colon) is the intussuscipiens.[2,4] As the intussusceptum invaginates into

the intussuscipiens, the mesenteric blood is occluded.[2,4,5] If the intussusceptum is short, blood flow is not affected and the intussusception acts as a simple obstruction.[4] If the intussusceptum is long, mesenteric blood flow is occluding, resulting in edema of the intussusceptum. The edema causes retention of the intussusceptum, ultimately resulting in more severe vascular obstruction, adhesion formation, and bowel necrosis.[2,4] This obstruction eventually leads to acute colic and lack of response to pain management.[4]

Diagnostic Approach/Considerations:

On presentation, Cody was given Buscopan intravenously and rectal palpation was performed, revealing no abnormalities. A nasogastric tube was passed and 4 liters of water was administered with a net reflux return of -3 liters. A complete blood count and biochemistry panel were performed. This revealed a mild leukopenia, mildly elevated BUN and ALP, mild hypoproteinemia, mild hypoalbuminemia, mild hypocalcemia, and mild hyperphosphatemia. Abdominal lactate was within normal limits (1.6 mmol/L). Abdominal ultrasound indicated corrugation of the wall of the cecum and edema in the wall of the cecum. The CBC, biochemistry panel, and abdominal ultrasound results in combination of the history led to a presumptive diagnosis of typhlocolitis potentially caused by chronic NSAID toxicity. Gastrointestinal disease caused by NSAID toxicity is typically characterized by mucosal ulceration, inflammation, bleeding, and protein-losing enteropathy.[5]

Treatment and Management

Cody was administered Ranitidine at 7 mg/kg (9 tabs PO), Sucralfate at 25 mg/kg (10 tabs PO), Misoprostol at 3 mcg/kg (6 tabs PO) for gastroprotection, 2 scoops of Platinum Balance as a probiotic, and 2 pounds of Bio-sponge as a probiotic every 12 hours via nasogastric

tube throughout his hospital stay. On Friday, February 23, he would not drink, so he was given Lactated Ringers Solution intravenously at 2 liters per hour and 30 mls of salt orally every 6 hours to encourage drinking. Cody was offered several different types of hay and grain mashes, but he would not eat. That evening, he spiked a mild fever of 101.9 degrees Fahrenheit and became tachycardic, so he was given Polymyxin B to bind endotoxin and ice boots were applied to all four feet to prevent laminitis. On Saturday February 24, Cody showed improvement. He had a normal physical exam and ate small amounts of feed. His CBC revealed a normal neutrophil count.

On Sunday, February 25, 2018, Cody began showing mild signs of colic in the afternoon that progressed to recumbency and rolling. Despite administering medications for pain management (Lidocaine CRI), Cody continued to exhibit colic signs so a second abdominal ultrasound was performed. The ultrasound revealed multiple concentric rings separated by hypoechoic peritoneal fluid corresponding to the transverse view of the intestines ("target" or "bull's-eye" sign) in the upper right quadrant, which is a pathognomonic sign of intussusception.[1,2] Due to a grave prognosis Cody was humanely euthanized.

Necropsy examination confirmed the apex and body of the cecum were intussuscepted into the right ventral colon. The distal half of cecum was constricted at the cecocolic orifice. Mucosal tissues were dull purple within the base of the cecum, and mottled dark red-to-purple in the portions occupying the right colon. The mucosa of the cecal tip had a thin fibrinous membrane attached to it. The submucosa was 3 cm thick and contained copious edema with mild petechiation. Multiple patches of villus lymphangiectasia were present and coincided with areas of marked mesenteric lymphatic distention; vessels were 2-4 mm wide and contained soft,

buttery content. Three pedunculated infarcted lipomas, 3-6 cm wide, were present in the jejunal mesentery.[6]

Histopathology findings revealed the cecal wall was severely expanded by edema and protein-rich fluid in the submucosa, muscularis, and serosa. Mucosal epithelium was largely sloughed and the lamina propria was moderately edematous; few small areas of necrosis and suppurative inflammation was present randomly. The submucosal lymphatics were distended by pale pink fluid. All fascial planes were accentuated and expanded by fluid and mild hemorrhage. The deep submucosa developed mild fibrosis, which increased through the muscularis and formed a thick layer on the serosa. A mat of fibrin and neutrophils covered the serosa, and many small caliber blood vessels were present in the subjacent granulation tissue matrix.[6]

Case Outcome:

Over the first night of Cody's stay in hospital, he was seen pawing once, rolling once, and flank watching throughout the night. He would not eat or drink, despite being offered several flavored waters and soaked alfalfa cubes. Cody did not show any signs of colic throughout the second day (Friday, February 23), but he spiked a mild fever of 101.9 degrees Fahrenheit and became tachycardic that night. Polymyxin B (4,000 u/kg) in one liter of LRS was given intravenously as a bolus to bind endotoxin and ice boots were applied to all four feet and changed every two hours to prevent laminitis.

By 4:00 am (Saturday, February 24), Cody's temperature was within normal limits (99.7 degrees Fahrenheit) and he began showing interest in grain when hand fed and began drinking water. By the afternoon, Cody seemed to be moderately improved, so the Polymyxin B and ice

boots were discontinued, and his fluid rate was reduced from 2 liters per hour to 1 liter per hour. He continued to seemingly improve throughout the night.

At 3:00 am (Sunday, February 25) Cody seemed mildly uncomfortable before passing a soft bile movement, but otherwise had no problems overnight. He was still eating hay, eating little grain, and drinking and no abnormalities were noted during his 8:00 am physical examination. Due to his continued improvement, the LRS and Biosponge were discontinued. By the evening, Cody began showing mild signs of colic that quickly progressed to recumbency and rolling, despite administering medications for pain management. A nasogastric tube was passed and several liters of dark green to brown reflux was attained, suggesting a blockage in the gastrointestinal tract. An abdominal ultrasound was performed, which revealed a “bullseye” or “target” lesion in the upper right abdominal quadrant, indicating intussusception. Due to age, unmanageable level of pain, and poor prognosis, Cody was humanely euthanized, and a necropsy was performed with owner’s permission.

In conclusion, intussusception is an uncommon cause of colic (especially in geriatric horses) but should always be included as a differential for any horse exhibiting signs of colic. Transabdominal ultrasonography and rectal palpation can be used to diagnose intussusception, but exploratory laparotomy and necropsy are definitive diagnostic procedures. Treatment for all types of intussusception is surgical intervention and the type of procedure is dependent on the severity and location of the intussusception. In this case, prognosis was poor due to the patient’s age, the location of the intussusception within the intestinal tract (cecocolic), and the duration and severity of clinical signs.

Treatment of choice for all types of intussusception is surgical intervention but is not always successful due to presence of peritonitis, rupture of the cecum, or a nonreducible lesion.[5]

Because cecocolic intussusceptions cause more edema and adhesion formations, manual reduction is not typically a treatment option.[2,4] Other techniques such as colotomy, total or partial typhlectomy, or bypass by ileocolostomy may be indicated.[1] Prognosis postoperatively is dependent on the amount cecum intussuscepted, the extent of necrotic bowel, the amount of surgical contamination, and the surgeon's amount of experience.[4] Postoperatively, patients should be medically managed on an individual basis.[2]

References:

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