Obstruction of the cecocolic orifice by ileoceocolic intussusception following jejunocecostomy in a horse

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A 4-year-old Thoroughbred stallion was referred for mild to moderate signs of abdominal pain, loose feces, anorexia, and decreased water intake of 3 weeks’ duration. An anthelmintic had been administered 2 to 3 months before referral. Dental floating had been performed the week before admission. The horse’s diet consisted of 1.5 kg of a 9% protein pelleted ration twice daily, 1 flake of alfalfa hay once daily, and grass hay ad libitum. There was no history of prior surgery. Signs of colic were indicated by prolonged periods of recumbency and biting at both flanks. The horse also appeared to have stranguria; however, the referring veterinarian had catheterized the urinary bladder without encountering an obstruction. Rectal examination by the referring veterinarian identified tenderness in the upper right abdominal quadrant. Results of serial CBCs performed by the referring veterinarian indicated an increased Hct ranging from 36 to 64% (reference range, 32 to 52%). Flunixin meglumine (1.1 mg/kg [0.5 mg/lb], IM) had been administered intermittently, but not more than once every 24 hours as needed for discomfort for the duration of clinical signs. Fenbendazole (dose unknown, PO) and vitamin B complex (dose unknown, IM) had been administered once daily for the 5 days before admission.

On physical examination, the stallion had signs of depression. Heart and respiration rates were 36 beats/min and 24 breaths/min, respectively. Rectal temperature was 37.8°C (100.0°F). Mucous membranes were pink with a capillary refill time < 2 seconds. Borborygmi were identified in all abdominal quadrants, and no heat or increased digital pulses to the hooves were palpable. On rectal examination, a moderate amount of soft fecal material was evacuated, and a mass in the upper right abdominal quadrant, gas distension of the cecum, and a tight ventral cecal band were identified. There were no abnormal findings on palpation of the scrotum. Nasogastric intubation was performed; however, no gastric or duodenal reflux was obtained. Two liters of mineral oil and 5 L of water were administered via the nasogastric tube. No fluid was obtained during abdominocentesis performed during the initial examination. Transabdominal ultrasonography performed with a 3.5-MHz curvilinear transducer revealed ingesta-filled small intestine and a small amount of gas within the large colon and cecum.

Blood samples were obtained for a CBC, serum biochemical analyses, and plasma fibrinogen concentration. Results of the CBC indicated that the Hct was within reference range (46%; reference range, 35 to 50%). Plasma fibrinogen concentration was increased (500 mg/dL; reference range, 100 to 400 mg/dL). Biochemical abnormalities included slightly high creatinine concentration (1.9 mg/dL; reference range, 0.6 to 1.8 mg/dL), hypoalbuminemia (1.7 g/dL; reference range, 2.5 to 3.8 g/dL), and high creatine kinase activity (724 U/L; reference range, 88 to 565 U/L). Exploratory celiotomy was recommended; however, the owner declined because of loss of use of the stallion for the next breeding season.

Medical management included IV administration of an isotonic polyionic solution at maintenance rate (50 mL/kg/d [22.7 mL/lb/d]). Flunixin meglumine (1.1 mg/kg, IV q 12 h) and xylazine HCl (0.3 mg/kg [0.14 mg/lb], IV, once) were administered for signs of pain. The horse was recumbent through the night. The heart rate (32 to 42 beats/min) remained within reference limits, and the respiration rate (16 to 24 breaths/min) increased intermittently. A small amount of loose feces was passed during the night. The horse would occasionally posture to urinate, but did not void. Despite normal heart and respiratory rates, it was the clinical impression of the attending clinicians that the horse had signs of moderate pain.

On day 2 of hospitalization, the horse was sedated with xylazine (0.3 mg/kg, IV) and butorphanol tartrate (0.01 mg/kg [0.005 mg/lb], IV) for reevaluation. Results of rectal palpation remained unchanged. Gastroscopy revealed mild hyperkeratosis and erosions of the nonglandular portion of the stomach. The urinary bladder was catheterized, and no urethral obstructions were found. Urine was obtained; results of urinalysis were within reference limits. Abdominal fluid was obtained via abdominocentesis, and cytologic examination revealed a transudate with no abnormalities.
Transabdominal ultrasonography was performed with a 2.5-MHz curvilinear transducer that made deeper penetration of the abdominal cavity possible. Ultrasonography of the right cranial ventral portion of the abdomen revealed that the apex of the cecum or right ventral colon had a bull's-eye or target appearance. The concentric alternating hyperechoic and hypoechoic rings were consistent with telescoping of the intestine (Fig 1). There was measurable thickening of the wall of the cecum or right ventral colon with increased echogenicity indicating edema. Hypoechoic areas were consistent with free fluid within the abdomen. The ultrasonographic findings were consistent with cecal inversion or an ileocecal intussusception with increased abdominal fluid secondary to inflammation. Surgery was recommended for correction of the intussusception.

Preanesthetic medication consisted of IV administration of fentanyl (0.001 mg/kg [0.0005 mg/lb]) and xylazine (0.4 mg/kg [0.2 mg/lb]). General anesthesia was induced with IV administration of ketamine (2 mg/kg [1 mg/lb]) and diazepam (0.1 mg/kg [0.5 mg/lb]) and maintained with sevoflurane. Before surgery, it was noticed that the horse had a scar on the ventral midline indicating the possibility of a prior surgical procedure. During surgery, palpation of the ventral colon revealed a large, firm cylindrical mass within the lumen. The mass began at the proximal aspect of the right ventral colon, close to the cecocolic orifice, and continued aborally toward the sternal flexure. The ileocecal fold and ileum were not in the normal anatomic position. An enterotomy was performed in the midventral colon to determine the extent and content of the mass. The mass appeared to be intestinal tissue, possibly colon or ileum. On the basis of enterotomy findings, it was suspected that a jejunocecostomy had been performed previously; however, the area of the jejunocecostomy could not be exteriorized during surgery. Attempts to reduce or exteriorize the intussusceptum were not successful; the horse was euthanized, and necropsy was performed.

Examination of the gastrointestinal tract revealed a 36-cm section of intestine that was attached at the ileocecal orifice and was intussuscepted into the proximal right ventral colon. After the intussusceptum was removed from the right ventral colon, the section of intestine was found to be comma shaped (Fig 2). The shorter curvature had a large mass approximately 15 cm in diameter. The segment of intestine was firm, and the outer mucosal surface of the intussusceptum was rough, dark red to black, with multiple foci of necrosis and deep ulcerations. The serosa was 1 cm thick, and the muscularis, submucosa, and mucosa had a white-yellow mottled color. The segment of intestine appeared to intussuscept on itself and end in a blind stump. The jejenum was attached to the cecum midway between the base and apex in a side-to-side anastomosis that contained several fine wire staples. Regional lymph nodes were diffusely tan and wet on cut surface.

Histologic examination of the ileal stalk revealed marked hypertrophy of external and internal smooth muscle layers. The mucosa was moderately autolytic; however, there was granulation tissue within the mucosa, and the muscularis mucosa was markedly hypertrophied. Vessels in the submucosa appeared viable, and there was mild submucosal fibrosis. The mucosal surface was necrotic, deeply ulcerated, and infiltrated with numerous neutrophils and fibrin, with extensive granulation tissue beneath and deep areas of fibroplasia extending the full thickness of the tissue. The submucosa of the cecum and colon was markedly edematous with dilated lymphatics and mild, diffuse infiltration of eosinophils and lymphocytes within the submucosa. There was marked submucosal thickening of the small intestine with extensive fibrosis between the muscular walls of the cecum and jejunum at the site of the anastomosis. Microscopic examination of the ileocolic lymph nodes revealed diffuse hyperplasia.
with high numbers of large lymphoid follicles and plasma cells and sinusoids filled with lymphocytes, plasma cells, and macrophages. Pathologic diagnoses were postsurgical development of an ileal stump with severe, chronic necrosis and fibrosis and marked smooth muscle hypertrophy; moderate cecal submucosal edema; and diffuse lymph node hyperplasia. Intussusception of the ileal stump into the cecocolic orifice caused hemorrhage and necrosis of the ileal stump and partial obstruction of the right ventral colon.

Colic is a nonspecific clinical sign in horses. Many causes of chronic colic involve the lower gastrointestinal tract. Intussusception should be considered in the differential diagnoses of young horses with signs of abdominal pain. An intussusception may cause acute abdominal pain or mild to moderate, intermittent, chronic signs of colic. Rectal palpation is less commonly performed in the clinical evaluation of colic in young horses because of the risks imposed by their small size. Transabdominal ultrasonography is an important diagnostic aid in young horses and may reveal the characteristic bull’s-eye pattern of an intussusception.

Intussusceptions are an uncommon cause of intestinal obstruction in horses. The etiology of the intussusception is often unclear; however, predisposing factors may include differences in segmental motility because of enteritis, heavy ascarid infection, *Anoplocephala perfoliata* infection, mesenteric arteritis, abrupt dietary changes, pedunculated mucosal masses, enenterotomies, end-to-end anastomoses, and jejunocecostomies. Intussusceptions are more likely to develop in horses ≤ 3 years of age, ≤ 1 to 14 and Thoroughbreds have a higher risk of developing intussusceptions. In 1 study, ileocecal intussusceptions accounted for approximately 50% of small intestinal intussusceptions. The length of the intussusceptum may be associated with the severity of clinical signs, the degree of obstruction, and the severity of abdominal pain. Regardless of the clinical signs, surgical intervention is required for correction of the intussusception.

Surgical correction of an intussusception may require resection of devitalized intestine. It may be necessary to redirect the flow of ingesta into the cecum by performing a jejunocecostomy after resection. Jejunocecostomy is indicated when the ileum is involved in a strangulating lesion. Jejunocecostomy and jejunocecal intussusception account for 76% of all small intestinal anastomoses. The remaining ileal stump is transected close to the cecum and is sutured closed. If the ileum is necrotic at the level of the transection and deeper toward the cecum, the ileal stump is invaginated into the cecum. In ileocecal intussusceptions, it may not be possible to reduce the intussusceptum, because the ileum is too edematous and hemorrhagic to permit safe reduction. In 1 study, of 7 chronic and 10 of 19 acute ileocecal intussusceptions were not reducible. Bypass of an ileocecal intussusception without reduc-tion of the intussusceptum has been described and caused obstruction of the cecocolic orifice by the intussusceptum. To avoid this complication, reduction of the intussusceptum via typhlotomy is recommended.

The indication for the original surgical procedure in the horse in this report could not be determined. The remaining ileal stump intussuscepted into the right ventral colon and became hypertrophied. Because of the chronic course of the disease, and because no nasogastric reflux was obtained after nasogastric intubation during the initial examination, it may be that the right ventral colon was partially obstructed, permitting fluid and gas to pass into the colon, and distension of the intestine did not develop. Hypoalbuminemia in this horse may have been because of necrosis of the stump and chronic protein loss.

References