Use of cisapride in the resolution of pelvic flexure impaction in a horse

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A 21-year-old, Appaloosa stallion was presented to the Western College of Veterinary Medicine with a 5-mo history of weight loss and difficult mastication. Additionally, for 3 d preceeding presentation, the horse had been anorectic.

Upon presentation, the horse was very emaciated, weighing 400 kg, and exhibited marked muscle wasting. Severely worn lower molar arcades and 2 missing lower molar teeth contributed to a "wave" appearance of the dental arcades. The horse demonstrated signs of colic, characterized by pawing, kicking at his abdomen, looking at his flank, restlessness, and rolling. On auscultation, there was a marked decrease in borborygmi in all 4 abdominal quadrants. On transrectal palpation, a 30-cm to 40-cm impaction of the pelvic flexure was noted.

Hematological findings indicated hemoconcentration manifested by increased packed cell volume (PCV = 0.44 L/L) and total protein (TP = 88 g/L). Fluid characterized as a normal transudate was recovered by abdominocentesis.

The physical examination and laboratory findings were compatible with a nonstrangulating obstruction due to feed impaction of the pelvic flexure. Medical management was instituted: all feed was withheld and 4 L of mineral oil (Mineral Oil USP; Vetoquinol Canada, Winnipeg, Manitoba) with 2 L of warm water were administered via a nasogastric tube. In addition, IV fluid therapy was begun, using lactated Ringer's solution at a flow rate of 2 L/h. Over the next 24 h, intestinal motility remained decreased on auscultation and no feces were passed. Nasogastric intubation was repeated and 50 mL of 25% dioctyl sodium sulphosuccinate (DOSS) in glycerine was administered in 6 L of warm water. Intravenous fluids were continued at 2 L/h. By day 3, no feces had been passed and intestinal motility was unchanged. Examination per rectum revealed that the impaction was palpably unchanged.

Over the next 4 d, mineral oil and water, and DOSS and water on alternate days, were administered via nasogastric intubation, and IV fluids were given, as previously described. The horse was allowed water ad libitum and fed small bran mashes 2 to 4 times/d. The impaction remained essentially unchanged, as measured by daily palpation per rectum and monitoring of fecal output. As economic constraints precluded surgical exploration and a prolonged course of medical therapy, additional means of medical management were considered.

Since intestinal motility was decreased, as noted by a decrease in borborygmi throughout hospitalization, motility modification was undertaken to break up and provide passage of the impacted feed. Horses with impaction colic frequently experience episodes of reduced intestinal motility; however, motility usually increases in response to oral cathartic therapy (1,2). This had not been observed on auscultation in this case.

Cisapride monohydrate (Prepulsid, Janssen Pharmaceutica, Mississauga, Ontario) was administered at 0.1 mg/kg of bodyweight (BW), PO, q8h for 72 h. Nine doses were administered in total. Five hours following the initial dose of cisapride monohydrate, intestinal motility was increased in all abdominal quadrants, as noted by an increase in borborygmi. At 8 h after the initial dose, signs of discomfort, such as lying down, rolling, and an increased heart rate became evident. Flunixin meglumine (Banamine, Schering-Plough, Calgary, Alberta) was administered at 1.1 mg/kg BW, IV, for analgesia. Thirty-six hours after the initial dose of cisapride monohydrate, 4 kg of very firm, dark, and dry feces were passed. Four to 5 kg of similar feces were passed at 40, 42, and 48 h. Palpation per rectum at 44 h following initiation of cisapride monohydrate treatment revealed that the pelvic flexure was empty and the small colon filled normally with feces. The horse was noticeably brighter, and more alert and responsive.

Prokinetic drugs, such as cisapride monohydrate, increase propulsive movement of ingesta within the intestine and have been used in horses for the prophylaxis and treatment of postoperative ileus (3,4). Cisapride monohydrate's prokinetic gastrointestinal properties have previously been noted in humans and dogs, where it causes stimulation and increased amplitude of contractions of the stomach, and small and large intestines, and restores suboptimal activity to normal activity without causing overall hypercontractility (5,6). In humans, it has been used for resolution of both functional and mechanical gastrointestinal obstructions, including chronic colonic pseudo-obstruction, postoperative ileus, chronic idiopathic constipation, and colonic inertia

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(4,7). In horses, it has been shown to increase motility of the left dorsal colon and cecum, improve coordination of the ileocecocolic junction, and increase transit through the stomach, and small and large intestines (4,5,8). The above properties are mediated by an enhanced release of acetylcholine from intramural postganglionic interneurons and by direct action on smooth muscle cells, especially in the colon (4,9). The interaction of cisapride monohydrate with its receptor causes an increased influx of calcium, which leads to enhanced contractile activity (4). Cisapride monohydrate does significantly increase intestinal motility soon after administration, as noted by increased borborygmi and increased rate of passage of feces (4,5). This response is thought to be greatest after the 3rd administration of the drug. In horses with ileus, borborygmi could be detected within 3 h after the administration of cisapride monohydrate, and defecation usually occurred within 24 h (3). In this report, the patient displayed signs of abdominal discomfort at 8 h following the initial administration of cisapride monohydrate. Dose-related side effects have been observed in ponies given infusions of cisapride monohydrate, including increases in heart rate and transitory signs of abdominal discomfort. Intravenously administered doses of 0.25 mg/kg BW were necessary to evoke these signs of colic in this study (5). In addition, humans with impaired colonic activity have experienced abdominal cramping with administration of cisapride monohydrate (8). The signs of abdominal discomfort demonstrated in this horse may have been directly associated with the dosage of cisapride monohydrate administered, or with the breakup and passage of the fecal mass itself.

Successful medical therapy to relieve fecal impaction in the horse is likely dependent on the presence of good intestinal motility to facilitate mechanical breakdown of the impaction. The results of this report suggest that the effects of cisapride monohydrate when administered PO are adequate to facilitate the breakdown and passage of intraluminal feed impactions when suboptimal colonic motility is present. Observations made in this case also indicate that not only are therapeutic levels achieved from oral administration, but lower dosages than previously reported may be sufficient to cause side effects, such as abdominal cramping, though these side effects appear to be transitory. Further clinical studies are necessary to document the pharmacokinetics and range of therapeutic modalities of this drug in the horse. CVJ

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