Repair of a full-thickness gastric rupture in a horse

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- Gastric dilatation in horses may be primary (eg, engorgement, *Gastrophilus* sp infection), secondary (eg, to obstruction or ileus), or idiopathic, and may result in rupture.
- Nasogastric intubation does not necessarily prevent gastric rupture.
- Gastric rupture in horses usually is associated with gross contamination of the peritoneal cavity and death.
- Successful surgical repair of gastric rupture in horses usually is limited to those horses in which the gastric mucosa is still intact.

A 14-year-old 550-kg Thoroughbred broodmare was admitted for evaluation of signs of mild to moderate abdominal pain of approximately 5 hours' duration. The mare had a history of having undergone resection of the large colon at another hospital 3 years earlier for treatment of large colon volvulus. The mare had foaled without complications 10 hours prior to admission and had expelled the placenta shortly after parturition. Though the farm manager had administered 2 doses of flunixin meglumine (1 mg/kg of body weight, IV) and the referring veterinarian had treated the horse for signs of pain with xylazine hydrochloride and butorphanol tartrate, the horse became increasingly refractory to medical management. One hour prior to admission, 12 L of gastric reflux was obtained via nasogastric tube.

The horse was alert and responsive but appeared mildly uncomfortable on admission to the clinic. Heart rate, respiratory rate, and rectal temperature were within normal limits, and hydration was judged to be adequate. Mucous membranes were normal in color, and capillary refill time was less than 2 seconds. Intestinal borborygmi were absent in all abdominal quadrants. Results of a CBC, serum biochemical analysis, and venous blood gas analysis were within reference limits. Although no reflux was obtained via nasogastric intubation, there was a large amount of rancid gas relieved from the stomach. Abdominocentesis yielded grossly normal fluid with a WBC count (1,100 cells/μl) and total protein concentration (1.4 g/dl) within reference limits. Examination per rectum did not reveal any palpable abnormalities. Because the mare continued to display signs of mild to moderate abdominal discomfort, it was elected to perform an exploratory celiotomy. Potassium penicillin (22,000 U/kg, IV, q 6 h), gentamicin sulfate (6.6 mg/kg, IV, q 24 h), and tetanus toxoid were administered prior to surgery.

A 35-cm ventral midline incision was made extending cranially from the umbilicus. The odor of intestinal tract gas was noticeable when the abdomen was opened, though no gross contamination of the peritoneal cavity was apparent. It was observed that a portion of the large colon previously had been resected at the sternal and diaphragmatic flexures. Exploration of the abdomen revealed a 20 × 8-cm subserosal hematoma overlying a longitudinal muscular tear along the parietal surface of the stomach, midway between the lesser and greater curvatures and approximately 12 cm from the saccus cecus. The serosa was bulging outward with gastric ingesta seeping subserosally through the tear in the mucosal and muscular layers of the stomach. Several branches of the left gastric artery appeared to be torn and thrombosed along the cranial aspect of the tear. Intestinal tract gas was leaking through a 4 × 4-mm perforation in the serosa, and the stomach was grossly distended with firm ingesta. As the large colon was exteriorized, the serosal perforation began to propagate, creating a full-thickness gastric rent. The margins of the gastric tear were grasped with towel clamps and traction applied. The abdominal incision was extended cranially to the xiphoid process and the remainder of the abdomen was isolated with moist towels in an effort to minimize gross contamination. Balfour retractors were used at the cranial aspect of the incision to facilitate surgical exposure. Traction sutures of No. 2 polyglactin 910[a] were placed in the seromuscular layer of the stomach, and the tear was extended to a 12-cm full-thickness defect by incision through the gastric serosa. Approximately 10 L of firm, moist feed material was evacuated from the stomach by use of a ceramic cup as a ladle, because the stomach could not be exteriorized. After removal of the gastric contents, the lesser omentum was transected from its attachment along the cranial aspect of the tear, and the seromuscular portion of the tear was closed in an inverting fashion by use of No. 1 polyglactin 910 in a continuous Cushing pattern oversewn with a continuous Lembert pattern. Care was taken to avoid incorporating the large blood vessels present along the surface of the greater curvature of the stomach in the closure. Further exploration of the abdomen revealed a small intestinal volvulus involving approximately 4 m of the proximal portion of the jejunum. Intestine involved in the volvulus was moderately dilated, and there was moderate distention proximal to it. The volvulus was easily reduced. Affected intestine was mildly edematous and slightly discolored, but motility was present and the segment was judged to be viable. The abdomen was lavaged with 10 L of sterile 0.9% NaCl solution prior to closure. The linea alba was apposed by use of No. 2 polyglactin 910 in 6 interrupted segments of a continuous suture pattern. The subcutaneous tissues and skin were closed by use of No. 2-0 polyglactin 910 in a simple continuous and continuous horizontal mattress pattern, respectively.

Potassium penicillin, gentamicin sulfate, and metronidazole (7 mg/kg, PO, q 6 h) were administered for 6 days after surgery. Flunixin meglumine (1 mg/kg, IV, q 12 h) was discontinued on day 3. The horse was maintained on balanced polyionic fluids (supplemented with

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500 ml of 50% dextrose, q 24 h) for 48 hours after surgery and was slowly introduced to small increments of water (2 L/h) and fresh green grass per os. A commercial nutritional supplement[b] (64 oz, q 4 h) was administered via an indwelling nasogastric tube from days 2 to 4. After surgery, all physical parameters remained within normal limits, and the horse had a good appetite with frequent passage of normal feces.

On day 2, the horse was fitted with a muzzle having a 5 × 3-cm hole in the bottom that allowed continuous oral intake of small amounts of fresh green grass and hay. Access to feed was allowed in this controlled fashion until day 22, at which time hay was fed ad libitum. On day 10, the horse began to pass black, loose feces containing digested blood. A CBC indicated a PCV of 21% and a serum total protein concentration of 2.0 g/dl. The heart rate was slightly high (44 to 48 beats/min), but all other physical parameters remained within normal limits, and the horse had no signs of hypoproteinemia (dependent edema). Ultrasonography of the stomach through the left body wall was inconclusive.

Endoscopy was not performed because of the unavailability of an endoscope long enough to evaluate the gastric lumen. Treatment with ranitidine[c] (6 mg/kg, PO, q 12 h) and sucralfate[d] (20 mg/kg, PO, q 6 h) was initiated and continued for 4 and 7 days, respectively. Feed was withheld for 48 hours. A cross-match with a suitable donor was performed in the event a blood transfusion was required. Twenty-four hours after the initial passage of melena, the serum total protein concentration had increased to 3.8 g/dl and was 5.2 g/dl by 48 hours. By day 12, heart rate had returned to within normal limits, the melena had resolved, and fecal occult blood test results were negative. The mare was discharged on day 17, at which time the PCV was 24%, and serum total protein concentration was 6.0 g/dl. Two years after surgery, the horse had successfully returned to service as a broodmare and had experienced no recurring signs of abdominal discomfort.

Gastric rupture often is a fatal form of colic in horses, because it results in acute septic peritonitis, toxemia, and circulatory shock.[1] The reported prevalence of gastric rupture, relative to other types of equine colic, ranges from 5[2] to 8%.[3] There does not appear to be an age, breed, sex, or seasonal predilection for this disease,[4] although findings in 1 retrospective study[5] indicated that geldings may be overrepresented. Clinical signs are usually compatible with severe abdominal crisis, and death is imminent once there is gross contamination of the peritoneal cavity. The retrieval of notable amounts of nasogastric fluid or gas on intubation can be a misleading and inconsistent indicator of gastric decompression, because the presence of an indwelling nasogastric tube does not preclude gastric rupture.[5]

Gastric dilatation has been classified as primary (eg, grain engorgement, excessive water intake after exercise, or severe *Gastrophilus* sp infestation),[1] secondary to a small or large intestinal obstruction or ileus,[1,5] or idiopathic.[2] In 1 retrospective study of 54 cases of gastric rupture,[5] approximately 65% of the gastric dilatations were attributed to secondary causes, whereas only 17% were classified as idiopathic. In another study involving 50 horses,[4] 60% of the ruptures were believed to be idiopathic. The reason for this discrepancy is unknown. In the horse of this report, it is possible that gastric rupture occurred secondary to the small intestinal volvulus, which caused mechanical luminal obstruction and concurrent ileus proximal to the twist. There did not appear to be vascular compromise to the segment of intestine involved, however, even though the history indicated that there was abdominal discomfort of 5 hours' duration. It cannot be ruled out that the volvulus may have been an artifact of distention of the proximal portion of the small intestine, resulting from a primary ileus of unknown origin and that gastric dilatation either was idiopathic or secondary to ileus. The horse was reported to have had free access to hay during the 5-hour period of abdominal discomfort prior to admission. This may have contributed to the gastric load and promoted further distention of the stomach with ingesta and gas.

Persistent signs of pain were the primary indication for an exploratory celiotomy in this horse. Further delay would have probably resulted in an unsuccessful outcome. The gastric rent had progressed until only the serosa contained the gastric contents. The small perforation in the serosa had allowed evacuation of gastric gas into the peritoneal cavity. The semidry nature of the food material in the stomach and the intact serosa overlying the muscular tear temporarily prevented spillage of the gastric contents into the abdominal cavity. It appeared that the counterpressure of the large colon on the wall of the stomach had served to slow the progression of the rupture once the gas had escaped, because removal of the colon from the abdomen resulted in an immediate increase in the rate of gas expulsion and a visible propagation of the tear. When the large colon was exteriorized, the sudden lack of counterpressure against the exterior of the distended stomach allowed the weight of the gastric contents to stretch the injured gastric wall and resulted in further propagation of the tear. Immediate traction on the gastric wall halted the progression of the tear until the rest of the abdomen was sufficiently isolated and the gastrotomy extended to allow for manual evacuation of the ingesta.

Successful surgical repair of gastric rupture in horses has been limited to case reports of seromuscular tears in 2 horses.[6,7] In these horses, disruption of the gastric mucosal layer had not occurred; therefore, gross contamination of the peritoneal cavity was avoided.

Typically, gastric tears occur along the greater curvature of the stomach, with the seromuscular layer weakening and tearing before the mucosal layer.[5,8] The horse in this report had an unusual tear in that both the muscularis and the mucosa had ruptured, and the serosa remained intact except for the focal perforation that had allowed gas to escape. It is possible that puncture of the stomach may have inadvertently occurred during abdominocentesis and contributed to the propagation of the tear. This was considered unlikely because of the parietal location of the tear and because the peritoneal fluid had been easily obtained on the initial centesis without evidence of feed or blood contamination.

Adequate surgical exposure to the stomach is difficult, and contamination of the abdomen with gastric contents is a major risk. Extending the abdominal incision as far cranial as possible and removing the large

colon from the abdomen is helpful.[9] Surgical exposure in this case was aided by the fact that the tear had occurred midway between the greater and lesser curvatures along the diaphragmatic surface of the stomach, and that the distended viscus fell toward the center of the abdomen when the colon was exteriorized.

A single-layer continuous inverting suture pattern has been recommended for repair of gastric seromuscular tears.[9] Because of disruption of the arterial blood supply along the cranial margins of the tear, a double-layer continuous inverting pattern was used to invaginate the thrombosed margin of the gastric wall to confine ischemic tissues to the lumen of the gastrointestinal tract. It has been shown in dogs that invaginating devitalized areas of the gastric wall does not detrimentally affect gastric outflow, and subsequent sloughing of the devitalized invaginated segment causes no notable clinical disease other than melena.[10] The horse in this report sustained an acute gastrointestinal tract hemorrhage 10 days after surgery, as evidenced by the sudden dramatic decrease in PCV and plasma protein concentration and passage of melena. This was attributed to ulceration of the gastric mucosa, possibly attributable to sloughing of a portion of the devitalized invaginated gastric wall and subsequent hemorrhage. An H_2-receptor antagonist (ranitidine) and a mucosal adherent (sucralfate) were administered in an attempt to decrease hydrochloric acid secretion and provide cytoprotection to the potentially denuded gastric lining. Expense precluded use of these agents for an extended period of time and, clinically, the acute hemorrhage appeared to resolve after 48 hours with the return to passage of normal feces and the concurrent increase in serum total protein concentration.

A conservative postoperative feeding program was believed to be important after repair of the gastric rupture to minimize tension on the suture line and allow adequate healing. Frequent feedings of soft roughage (fresh grass and leafy hay) in small amounts, with a slow introduction to grain, were believed to be preferable. Nutritional support in the form of a commercial oral supplement was administered in the immediate postoperative period, because the mare was lactating and there was concern that its milk production would sharply decline if held off feed for an extended time. This provided approximately 75% of the mare's maintenance nutritional requirements. Additional calories were provided in the form of concentrated dextrose supplemented to the fluids administered IV in the initial 48 hours after surgery. Parenteral nutrition may have been the preferred method for nutritional supplementation, but financial constraints precluded its use. A controlled oral feeding program was initiated on day 2 after surgery and maintained until resumption of full feed by day 30.

[a]Vicryl, Ethicon Inc, Somerville, NJ.
[b]Nutrical, Ross Laboratories, Columbus, Ohio.
[c]Zantac, Glaxo Pharmaceuticals, Research Triangle Park, NC.
[d]Carafate, Marion Merrell Dow, Kansas City, Mo.

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