Gastric impaction and obstruction of the small intestine associated with persimmon phytobezoar in a horse

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- ➤ Phytobezoar formation consequent to the ingestion of ripe persimmon fruits (*Diospyros virginiana*) by horses during fall and winter may lead to weight loss, lethargy, and colic.
- ➤ Colic is associated with primary gastric impaction, gastric ulceration, and obstruction of the small intestine.
- ➤ Persimmon phytobezoars in the lumen of the stomach can be identified by gastric endoscopy.
- ➤ Clinical problems associated with persimmon phytobezoar formation are successfully treated by surgical removal of the phytobezoar.

A 2-year-old Quarter Horse gelding was referred to the **Veterinary Medical Teaching Hospital (VMTH)** for treatment of colic. The gelding had signs of mild colic, intermittent lethargy, and weight loss for 6 weeks prior to referral. During previous episodes of colic, the horse responded favorably to treatment that consisted of administration of mineral oil and flunixin meglumine. Referral had been prompted by an episode of colic that failed to resolve with conservative treatment.

At initial evaluation, the gelding was lethargic, but overt signs of abdominal pain were not evident. Gastric reflux caused fluid to drain from the nares. Heart rate was 84 beats/min, respiratory rate was 12 breaths/min, and rectal temperature was 103.6 F (39.8 C). Auscultation of the thorax revealed increased bronchovesicular sounds in the ventral aspect of both hemithoraces. Passage of a nasogastric tube facilitated the drainage of approximately 35 L of gastric fluid. Multiple loops of mildly distended small intestine were identified by palpation per rectum.

Results of CBC analysis revealed a slight left shift (316 band neutrophils/µl; reference range, 0 to 100 band neutrophils/µl) and mild toxic changes in neutrophils. All other parameters were within reference ranges. Abnormalities identified by serum biochemical analyses included hypochloridemia (85 mmol/L; reference range, 94 to 103 mmol/L), hyperglycemia (137 mg/dl; reference range, 72 to 114 mg/dl), hypocalcemia (8.1 mg/dl; reference range, 10.7 to 13.4 mg/dl), hyperbilirubinemia (3.9 mg/dl; reference range, 0.10 to 1.90 mg/dl), azotemia (creatinine, 2.1 mg/dl; reference range, 0.60 to 1.80 mg/dl), hypokalemia (2.7 mmol/L; reference range 2.70 to 4.90 mmol/L), and mildly increased activity of γ-glutamyltransferase (58 U/L; reference range, 12 to 45 U/L). Arterial blood gas analysis revealed metabolic alkalosis (pH, 7.523; P_{CO_2}, 38.4 mm Hg; bicarbonate, 31.8 mmol/L). Analysis of peritoneal fluid revealed changes consistent with inflammation (total nucleated cell count, 50,100 cells/µl; reference range, < 5,000 cells/µl; protein concentration, 3.1 g/dl; reference range, < 2.0 g/dl); most nucleated cells were neutrophils.

Ultrasonographic examination of the abdomen revealed multiple loops of mildly distended small intestine with moderate mural thickening consistent with a diagnosis of duodenitis-proximal jejunitis. An increased quantity of peritoneal fluid was also recognized, but additional evidence of peritonitis (such as adherent fibrin tags) was not seen. Thoracic radiography revealed an alveolar pattern in the cranioventral aspect of the lung fields, consistent with aspiration pneumonia. The tentative clinical diagnosis was aspiration pneumonia and obstruction of the small intestine; differential diagnoses included duodenitis-proximal jejunitis syndrome, ileocecal intussusception, early-stage incarceration of the intestine, obstructive mural lesions (such as granulomas or lymphosarcoma), and luminal obstructants or impaction.

Initial treatment included potassium penicillin G (22,000 U/kg [10,000 U/lb] of body weight, q 6 h, IV), gentamicin sulfate (4 mg/kg [1.8 mg/lb], q 24 h, IV), and flunixin meglumine (0.5 mg/kg [0.23 mg/lb], q 12 h, IV). The gelding was also treated with warmed isotonic saline (0.9% NaCl) solution (2 L/h) containing potassium chloride (20 mmol/L), administered IV. The gelding was not allowed access to food and water, and a nasogastric tube was inserted to permit gastric decompression every 2 hours.

During the first 24-hour period, approximately 4 to 7 L of gastric fluid was obtained every 2 hours. On the second day of hospitalization, heart rate was 40 beats/min, respiratory rate was 16 breaths/min, and rectal temperature was 100.7 F (38.2 C). The gelding was alert, quiet, and responsive to its environment; further signs of abdominal pain had not developed. Feces were loose and oily. Results of repeated CBC and serum biochemical analyses did not reveal abnormalities, and alkalemia had resolved (pH, 7.418). Repeated peritoneal fluid analysis indicated worsening peritonitis (total nucleated cell count, 120,000/µl; protein concentration, 3.2 g/dl). Most nucleated cells were neutrophils, and bacterial organisms were not seen. Peritoneal fluid was submitted for bacteriologic culture, which yielded no growth of organisms. Repeated palpation per rectum did not reveal abnormalities; distended loops of small intestine were no longer evident.

Following removal of the nasogastric tube, endo-

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scopic examination revealed moderately severe and extensive ulceration of the gastric lining of the glandular and squamous regions of the stomach. The squamous epithelial lining appeared to be hyperkeratotic. Furthermore, a large conglomeration of apparently impacted material was identified, which appeared to occupy > 50% of the gastric lumen. It was concluded that this was most likely a large mass of impacted feed.

At this time, treatment of the gelding included continued administration of antimicrobials and reduced dosages of flunixin meglumine (0.25 mg/kg [0.11 mg/lb], q 8 h, IV), ranitidine (6.6 mg/kg [3.0 mg/lb], q 8 h, PO), and lactated Ringer's solution (1 L/h, IV). Plasma concentrations of gentamicin sulfate were monitored and determined to be in the appropriate therapeutic range (predose plasma concentration of gentamicin sulfate, 0.2 µg/ml; target concentration, < 1.0 µg/ml[1]). Specific treatment for gastric impaction included oral administration of mineral oil, dioctyl sodium sulfosuccinate, and warm water lavage of the stomach (q 12 h).

Further signs of abdominal pain were not observed, and the gelding defecated scant, loose feces laden with mineral oil. The gelding ate small quantities of hand-picked grass, offered every 2 to 4 hours, with good appetite. Gastric reflux was not evident after the second day of hospitalization. Fecal consistency was semiformed on the third day. Results of peritoneal fluid analysis on day 5 revealed normal peritoneal fluid (total nucleated cell count, 3,200/µl; protein concentration, < 2.0 g/dl). The gelding appeared to be progressing well and was eating hay. Signs of colic were not observed, and fecal consistency was returning to normal. Treatment with flunixin meglumine was discontinued.

On day 9 of hospitalization, repeated gastric endoscopic examination revealed that ulceration of the squamous and glandular mucosal lining was slightly less severe and extensive than previously observed. However, appearance of the gastric impaction was unchanged. Treatment to resolve the gastric impaction was continued orally; IV administration of fluids was discontinued.

Signs of moderately severe, progressively worsening lethargy and inappetence were observed during the next 5 days. Although overt signs of abdominal pain were not observed, the gelding spent protracted periods in lateral and sternal recumbency. Vital signs remained within reference ranges during this time.

Signs of acute abdominal pain and weight loss were evident on day 14 of hospitalization. Heart rate was 84 beats/min, respiratory rate was 32 breaths/min, and rectal temperature was 104.5 F (40.3 C). Twenty-one liters of gastric reflux fluid were obtained following passage of a nasogastric tube. Palpation per rectum revealed multiple loops of mildly distended small intestine. Results of peritoneal fluid analysis were normal. Results of a CBC revealed mature neutrophilia (14,310 cells/µl; reference range, 2,260 to 8,580 cells/µl) and hyperfibrinogenemia (0.6 g/dl; reference range, 0.10 to 0.40 g/dl). Results of serum biochemical analyses were within reference range, except for hyperbilirubinemia (8.1 mg/dl; reference range, 0.10 to 1.90 mg/dl), which was attributed to the combined effects of negative energy balance and, possibly, partial bile duct obstruction.

General anesthesia was induced with xylazine hydrochloride (1.1 mg/kg [0.5 mg/lb], IV) and ketamine hydrochloride (2.2 mg/kg [1.0 mg/lb], IV) and maintained with halothane in oxygen. An exploratory celiotomy revealed a moderately distended small intestine. Partial obstruction of the small intestine at multiple locations was associated with 5 discrete, hard, and irregular intraluminal concretions, which were identified in the stomach, the duodenum (2), the jejunum, and right portion of the ventral colon.

It was not possible to manually attenuate any of the concretions. The appearance of the serosal surface of the intestine overlying the concretion in the jejunum suggested that localized pressure necrosis was developing. The 2 duodenal concretions had been manually pushed to the location of the jejunal concretion, which was immobile. Removal of the 3 concretions was accomplished by resection of an approximately 60-cm length of jejunum and an end-to-end anastomosis. The concretion in the right portion of the ventral colon was pushed into the pelvic flexure and removed via pelvic flexure enterotomy. After careful packing-off of the surrounding abdominal viscera with moistened laparotomy towels, removal of the gastric concretion was facilitated by a 15-cm gastrotomy at the greater curvature of the stomach. The peritoneal cavity was irrigated with 10 L of warmed saline (0.9% NaCl) solution containing heparin (20,000 U), potassium penicillin G (10,000,000 U), and gentamicin sulfate (1 g) prior to routine closure. The gelding recovered from general anesthesia without complications. Following removal, the concretions were identified as persimmon phytobezoars.

Treatment with potassium penicillin G, gentamicin sulfate, metronidazole, and ranitidine was continued after surgery. Additional treatment included administration of lactated Ringer's solution (1 L/h, IV), flunixin meglumine (1.1 mg/kg, q 12 h, IV), and heparin (40 U/kg, q 8 h, SC). Following the celiotomy, the gelding's attitude and appetite progressively returned to normal. Treatment with flunixin meglumine, heparin, and lactated Ringer's solution was discontinued on postoperative day 3, and antimicrobials were discontinued on postoperative day 6.

The gelding was discharged into the care of the owner 40 days after initial hospitalization for an 8-week convalescent period, during which time enforced exercise would not be allowed. At 6 months after discharge, the gelding had reestablished clinically normal body composition and did not have further signs of colic. The gelding was reexamined at the VMTH 9 months after discharge, at which time endoscopic examination was repeated; no abnormalities were evident.

Engorgement with ripe persimmons by many avian and mammalian species happens commonly in the fall.[2] Consumption of persimmons is the most common cause (worldwide) of phytobezoars in human beings.[3] Development of colic following ingestion of persimmons is widely believed to develop more commonly in horses than would be anticipated, on the basis of the paucity of reports in the literature.[2,4,5] Persimmon fruit contains a water-soluble tannin that polymerizes to

form an adhesive coagulum on exposure to gastric acid. This coagulum reacts with cellulose, hemicellulose, and protein to form a progressively hardening solid mass, into which persimmon seeds are incorporated.[2] Persimmon seeds, which become cemented into the developing phytobezoar, are oblong, flat, pale-brown, hard-surfaced objects measuring approximately 1.0 to 1.5 × 0.5 cm.[2] A phytobezoar in the stomach leads to colic and gastric ulceration caused by luminal obstruction and mechanical abrasion. Ulceration of the glandular lining of the stomach of horses is also facilitated by the loss of cytoprotective mucin, which is degraded by persimmon-derived tannic acid.[2] Fragmentation of the primary gastric phytobezoar has led to obstruction of the small intestine in humans.[6,7]

Clinical signs of gastrointestinal tract obstruction associated with persimmon phytobezoar formation in horses are vague, nonspecific, and may be characterized by intermittent signs of mild to moderate abdominal pain that persist despite conservative treatment with laxatives and analgesics.[2,4,5] Problems associated with ingestion of persimmons should be suspected in specific geographic locations when signs of colic develop in horses that have access to persimmon fruit in the fall, when ripe fruit falls to the ground. In our horse, gastrointestinal tract obstruction affected the stomach and the small intestine and led to gastronasal reflux, palpably distended small intestine, gastric ulceration, and peritonitis.

Weight loss in the horse of this report was evident at the time of referral and became more prominent during hospitalization. Weight loss was attributed to reduced appetite, chronic pain, and the periods of fasting that were instituted during treatment of colic. Reduced appetite was likely a consequence of pain, provoked by gastric filling and reduction of the gastric luminal volume by the phytobezoar.

Exploratory celiotomy was justified on the basis of the protracted course of colic, which did not resolve with fasting and treatment with mineral oil and dioctyl sodium sulfosuccinate. Furthermore, the appearance of a primary gastric impaction, which had been identified on endoscopic examination of the stomach, had not changed after 8 days of treatment. A simple primary gastric impaction associated with inspissated feed material should have resolved following treatment with mineral oil and dioctyl sodium sulfosuccinate. Retrospectively, recognition of persimmon seeds at the surface of the gastric impaction on endoscopic examination could have facilitated the diagnosis earlier in the clinical course, but the surface of the conglomerate was obscured with saliva and ingested grass. Possibly, vigorous irrigation of the surface of the impacted mass with water via the endoscope may have improved observation of superficial persimmon seeds and facilitated an earlier diagnosis.

Clinical signs observed in our horse were similar to those reported in other horses[2,4,5] with colic and weight loss associated with persimmon ingestion. Unfortunately, information pertaining to persimmon exposure was not available to us at initial evaluation. However, unlike other reports, obstruction of the small intestine was a predominant finding in this horse.

Of further interest, peritoneal fluid analyses performed during the first 24 hours of hospitalization were consistent with a severe inflammatory response (peritonitis). During treatment, however, peritoneal fluid abnormalities resolved prior to day 5 and were still within reference range at day 14 when the horse was anesthetized. A satisfactory explanation for the peritonitis, which resolved despite further obstructive episodes, is lacking. Possibly, an obstructive fragment of the primary gastric phytobezoar initially caused a reversible degree of intestinal devitalization, which led to peritoneal inflammation. Our conservative treatment may have facilitated sufficient peristaltic efforts, allowing the fragment to be passed along the intestinal tract and enabling local intestinal damage to heal.

Successful treatment of a gastric persimmon phytobezoar has been reported.[4] In 1 horse, gastric impaction was identified during exploratory celiotomy; the horse was treated by intraoperative massage with water pumped into the stomach via a nasogastric tube.[4] In our horse, the phytobezoar was considered to be too large and dense to be safely broken down by massage through the wall of the stomach. Furthermore, small phytobezoars had already caused obstruction of the small intestine.

Diagnosis of gastric impaction and obstruction of the small intestine associated with persimmon phytobezoar development should be considered in horses with signs of intermittent colic, obstruction of the small intestine, and weight loss that do not respond to treatment with analgesics and laxatives. Persimmon phytobezoar impaction is most likely to develop in the fall and winter in geographic locations where persimmon trees (*Diospyros virginiana*) are found. Diagnosis of gastric impaction associated with persimmon phytobezoar formation may be facilitated by endoscopic examination of the stomach, especially if persimmon seeds can be identified at the surface of the mass. If untreated and unrecognized, phytobezoar development may lead to death by gastric rupture and peritonitis.[2] Treatment with analgesics and laxatives appears to be generally unsuccessful, and exploratory celiotomy should be considered. Although treatment of a small gastric persimmon phytobezoar may be accomplished by intraoperative massage and manipulation,[4] successful treatment of larger phytobezoars can be accomplished by extraction via gastrotomy.

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