**Clinical Snapshot #2**

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**CASE PRESENTATION**

A 4-week-old Thoroughbred colt presented with a history of colic, depression, and partial anorexia. The foal was depressed and in poor body condition. Abdominal distention, profuse salivation, and teeth grinding were noted. Ultrasonography of the abdomen revealed a distended stomach, distended small intestine with poor motility, and thickened (7.7 mm) duodenal wall. A nasogastric tube was passed, and 3000 mL of reddish-brown reflux was obtained. Hematology revealed a stress response (neutrophilia and lymphopenia), and a serum biochemistry profile identified mild hyponatremia, hypochloridemia, and metabolic acidosis. An endoscopic examination revealed severe erosions and ulcerations in the stomach and pylorus. During the first 24 hours of hospitalization, the colt continued to grind his teeth and had a significant amount of gastric reflux. No further signs of colic were noted.

1. What is your diagnosis?
2. What are the treatment options?

(See page 480 for answers and explanations.)
Clinical Snapshot

ANSWERS & EXPLANATIONS

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1. Gastroduodenal ulcer disease. This disease is a clinically significant entity in suckling and weanling foals. Unlike the typical squamous mucosal ulceration in adult horses, the lesions in foals often occur in the glandular mucosa and/or the duodenum. Gastric ulcer disease in foals may be divided into four clinical syndromes, including subclinical or silent ulcers, clinical or active ulcers, perforating ulcers, and ulcers associated with duodenal outflow obstruction. Foals may appear clinically normal, present with clinical signs consistent with recurrent or chronic colic, or display more specific clinical signs, including ptyalism, bruxism, acute colic, gastric reflux, diarrhea, or fever. Some affected foals may rapidly develop life-threatening endotoxic shock indicative of gastric or duodenal perforation. The pathogenesis of the disease in foals is unknown but is assumed to be similar to ulceration in other species: an ulcer forms when the effect of acid – pepsin overcomes the protective factors in the gastric or duodenal mucosa. The only documented cause of gastroduodenal ulcers in foals is the use of NSAIDs that inhibit prostaglandin synthesis. The clinical signs, history, and findings obtained with one or more diagnostic imaging modalities (e.g., ultrasonography, endoscopy, radiography) are useful in diagnosing a gastric outflow obstruction. Abnormalities in peristalsis, duodenal wall thickness, and lumen diameter are evident on ultrasonographic examination. Survey radiography may reveal a dilated, fluid-filled stomach and/or esophagus and aspiration pneumonia. A barium contrast study may reveal reflux of gastric fluid, an enlarged gastric silhouette, and delayed gastric emptying of more than 2 hours. Endoscopy allows visualization of gastric ulcerations and stricture of the pylorus.

2. Medical therapy includes supportive care, decompression of the stomach, and administration of gastroprotectants. When gastric erosions and ulcerations become severe, prolonged medical treatment may not alleviate the ulceration, and surgical bypass around the duodenal stricture may be required. The procedure performed depends on the location of the obstruction. Surgical correction has been accomplished by gastrojejunostomy, duodenojejunostomy, jejunojejunostomy, and gastrojejunostomy. The literature reports a poor prognosis (36% to 46% survival rate) for foals that undergo gastrojejunostomy. In one study, gastroenterostomy was performed in 14 foals. At follow-up, successful outcome (defined as alive) of five foals was reported. Another study reported a 46% survival rate among foals that underwent surgical correction of gastroduodenal obstruction. Few reports compare medical versus surgical management. Further research in this area is warranted.

REFERENCES