Client: Underwood, Kirk **Patient:** Gastric Ulcer **Species:** EQUINE **Breed:** **Sex:**

Age:

SUBJECTIVE SECTION

diet: We need a diet with a good calcium phosphorus balance. 8-10% Ca and 10-12% Ph is recommended. A good supplement would be feeding Purina 12/12 or adding 20 ppm dicalcium phosphate to the feed. i strongly recommend feeding xtn made by nutrina. Alfalfa hay is also recommended to aid in emptying of the stomach, and also is a good source of calcium. Some debate is still going on with feeding alfalfa, but excess calcium will be excreted in the stool and urine. The calcium will also neutralize the acidity in the stomach. Also, this horse can handle 2 cups vegetable oil daily. This is a good source of omega 3, 6, and 9 fatty acids which also help neutralize the gastric acidity. The oil will also help empty the stomach to help reduce acid production. In addition, the oil will help with weight maintenance and the hair coat. This horse should be in a stress resistant environment, and we should be very strict as to what we give this horse medically. Steroids, anti inflammatories, and some types of treats could exacerbate gastric ulcers. Please contact me in the next few weeks to schedule a recheck and monitor food intake, water consumption, behavioral changes, and watch for diarrhea. Routine endoscopic examinations of the upper gastro intestinal track is strongly recommended to tract progress.

OBJECTIVE SECTION

ASSESSMENT SECTION

PLAN SECTION

NOTES

THERAPEUTIC NOTES:
Mild gastric ulcers are seen in ~50% of foals. In most cases, these ulcers heal without treatment or clinical signs. The prevalence of clinical signs from ulcers in foals is not known. In adult horses, ~30% have mild gastric erosions, and the prevalence and severity of ulcers increase as the intensity of work increases—90% of race horses have gastric lesions, and in 50% the lesions are moderate to severe. Prevalence also varies by location of ulcers within the stomach and tends to be highest in the nonglandular squamous mucosa. Within the glandular mucosa, prevalence differs between the mucosa of the corpus (<10%) and that of the antrum and pylorus (>50%).
Duodenal ulceration in foals has been considered part of the ulcer syndrome and hence a peptic (acid-induced) disorder. Duodenitis appears, instead, to be an enteritis syndrome. Duodenal ulceration, perforation, and stricture can occur, and it is not known whether these problems develop solely as a result of enteritis (duodenitis) or whether peptic factors have a role.
Etiology:
Causes of gastric ulcers vary and can differ for nonglandular squamous mucosa and glandular mucosa. Horses' stomachs secrete hydrochloric acid continuously, and gastric acidity of a horse or foal is very high between periods of eating or nursing. Ulcers in the squamous mucosa result from increased exposure to hydrochloric acid, which can be secondary to prolonged periods of not eating or nursing, intensive exercise, or delayed gastric emptying. The effects of different feeds on gastric acidity and ulcerogenesis have not been thoroughly studied, although one report indicated that alfalfa hay was associated with reduced ulcer severity in research horses. The causes of most ulcers in the glandular mucosa of the stomach are not known. Excessive doses of NSAID are known to induce ulceration, but most horses with ulcers in this part of the stomach have had no recent exposure to NSAID. Recent research suggests that horses may be infected with a species of Helicobacter , but a role for this organism in equine gastric ulcers has not been shown.

Clinical Findings:
Most foals with gastric ulcers do not exhibit clinical signs. Clinical signs become apparent when the ulceration is widespread or severe. The classic clinical signs for gastric ulcers in foals include diarrhea, bruxism, poor nursing, dorsal recumbency, and ptyalism. None of these signs is specific for gastric ulcers. In fact, ptyalism is a sign of

esophagitis, which in most foals is secondary to gastric outflow obstruction and gastroesophageal reflux. Other causes, including esophageal obstruction and Candida infection, should be considered. Importantly, when a foal exhibits clinical signs, the ulcers are severe and should be diagnosed and treated immediately. Sudden gastric perforation without prior signs occurs sporadically in foals. Adult horses with ulcers display nonspecific signs that can include abdominal discomfort (colic), poor appetite, mild weight loss, poor body condition, and attitude changes. Complications related to gastric ulcers are most frequent and severe in foals and include perforation, delayed gastric emptying, gastroesophageal reflux and esophagitis, and megaesophagus secondary to chronic gastroesophageal reflux. Ulcers in the proximal duodenum or at the pylorus can cause fibrosis and stricture. The latter complication is seen in both foals and adult horses. In rare cases, severe gastric ulceration causes fibrosis and contracture of the stomach.

Diagnosis:
Neither clinical signs nor laboratory tests are specific for gastric ulcers, and an abnormality in a laboratory test does not preclude the possibility that another disorder may be present. Gastric ulcers can develop secondary to problems in many organ systems. Endoscopy is the only reliable method of diagnosis.

Gastric ulcers, foal

Moderately severe ulcer, equine stomach

Severe ulceration, equine stomach

Treatment:
Suppression of gastric acidity is the primary treatment objective. This can be accomplished with over-the-counter antacids, with the histamine type-2 receptor antagonists (cimetidine and ranitidine), and with the proton pump inhibitor omeprazole. Of these, omeprazole is the most effective, and antacids are the least effective. Ranitidine (6.6 mg/kg, PO) is effective in healing and preventing gastric ulcers, but recent evidence suggests that cimetidine is not effective. Omeprazole is the only medication approved by the FDA for treatment of gastric ulcers in horses. It is effective for treatment and prevention of ulcers in race horses and other types of horses. Sucralfate binds to the gastric glandular mucosa and may promote healing there, although no efficacy data is available to support its use in horses.