Gastric Ulcers in Horses

(Equine Gastric Ulcer Syndrome)

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Equine gastric ulcer syndrome (EGUS) is an umbrella term used to describe erosions, ulcerations, and lesions in the terminal esophagus, nonglandular squamous and glandular regions of the stomach, and proximal duodenum. Performance horses and foals are at greatest risk to develop EGUS. Clinical signs are vague, and treatment requires pharmaceutical agents that increase gastric pH and foster an environment conducive to ulcer healing.

Gastric ulcers are common in horses and foals. The term equine gastric ulcer syndrome (EGUS) was introduced in 1999 as an umbrella term to describe a spectrum of inflammatory and disruptive mucosal pathophysiological changes that affect mucosal tissues in the terminal esophagus, nonglandular stomach (equine squamous gastric disease), glandular stomach (equine glandular gastric disease, and proximal duodenum.

The risk factors for horses to develop this syndrome include involvement in performance disciplines; high-concentrate (grain) diets, intermittent (bolus) feeding, environmental stress (pasture vs stall-confinement), stress due to travel or social interaction, and illness. The prevalence of EGUS in racehorses in active training and racing has been estimated to be at least 90%, whereas in nonracing performance disciplines the prevalence varies from 40% to 60%. The prevalence of EGUS in neonatal foals is estimated to be 25%–50%, and foals have a high risk of developing perforating peptic ulcers until they are several weeks old because their gastric mucosa is not developed to full thickness at birth and they have continuous gastric acid secretion. Although spontaneous healing of gastric ulcer lesions has been noted, if training, racing, and maintaining the risk factors that incite EGUS continue, lesions are unlikely to heal without medical intervention.

Endoscopic surveys indicate that ~80% of these lesions are found in the nonglandular squamous mucosa of the stomach (equine squamous gastric ulcer disease), especially on the lesser curvature just proximal to the margo plicatus. However, appreciable portions of the squamous mucosa along the greater curvature can be affected. On the other hand, ~20% of lesions can be found in the glandular region of the stomach (equine glandular gastric disease). Duodenal ulceration in

adult horses and foals is considered part of EGUS and, hence, a peptic acid-induced disorder. Duodenal ulceration, perforation, or stricture can occur, and it is not known whether these problems develop solely as a result of enteritis (duodenitis) or whether peptic factors such as acid might play a role. Once a stricture occurs, gastric and esophageal ulcers are often severe, having been exacerbated by delayed gastric emptying.

Etiology of Gastric Ulcers in Horses

Equine squamous gastric ulcer disease is associated with repeated direct insult and the erosive effects of extremely low pH fluid (acidic fluid) normally found in the glandular region of the stomach. Pressure increases inside the abdomen (associated with exercise), collapsing the stomach and forcing the acid gastric contents upward (acid splash), especially in the unfed horse during exercise. Highly acidic contents of the distal glandular portion of the stomach come in contact with the nonglandular squamous mucosa during intense exercise, causing acidification, inflammation, and, potentially, erosions and ulceration to varying extents.

Dietary factors play a role in the development of EGUS. Dietary factors play a role in the development of EGUS. Diets high in concentrates (grain) and low in roughage (hay) generate short-chain fatty acids (SCFAs; acetic, butyric, and propionic acids) as a result of fermentation of sugars. SCFAs are absorbed by nonglandular mucosal cells, leading to cellular acidification as well as inhibition of cellular sodium transport, leading to cellular edema and eventual ulceration. Alfalfa hay, on the other hand, is high in protein and calcium and acts as a dietary buffer, increasing pH in the stomach and preventing ulcer formation. When gastric pH is high, SCFAs are not lipid soluble and cannot be absorbed by squamous mucosal cells, so their effect is lessened.

The causes of ulcers in the glandular mucosa (equine glandular gastric disease) of the stomach are less well defined. Factors that have been proposed to contribute to spontaneous equine glandular gastric disease include breakdown of mucosal defenses (decreased blood flow, loss of the mucus-bicarbonate layer, and inhibition of prostaglandins), bacterial colonization, environmental and psychological stress, and inflammation.

Use of nonselective NSAIDs is known to inhibit prostaglandins, thus reducing blood flow to the stomach, causing decreased production of the mucus-bicarbonate matrix by the gastric glandular mucosa and resulting in ulceration. This is not a consistent finding, and in one study, prostaglandin concentrations in glandular mucosal tissue were not decreased when phenylbutazone was administered. Thus far, there is no evidence that bacteria play a role in causing glandular ulcers in horses.

Clinical Findings of Gastric Ulcers in Horses

Foals with gastric ulcers are often 2–6 months of age, and EGUS in foals has been divided into four clinical syndromes: subclinical (no apparent clinical signs), clinical (with clinical signs), perforating, and gastric outflow obstruction. Most foals have absent or vague clinical signs (subclinical form). However, when gastric ulcers become widespread and severe, classic clinical signs in foals include lethargy, colic, an unthrifty appearance, diarrhea, poor or interrupted nursing, dorsal recumbency, bruxism, ptyalism, frothing or drooling of milk from the mouth, and tongue rolling. Bruxism and ptyalism are signs of esophagitis and esophageal ulceration, which in most foals are secondary to gastric outflow obstruction and gastroesophageal reflux. In addition, esophageal obstruction and *Candida* spp infection should be considered if these clinical signs are observed in foals.

Importantly, when a foal has clinical signs, the ulcers are typically severe and should be evaluated and treated immediately. Complications related to gastric ulcers are most frequent and severe in foals; these include delayed gastric emptying, gastroesophageal reflux and esophagitis, megaesophagus, and acute perforation. Sudden gastric perforation without prior clinical signs occurs sporadically in foals.

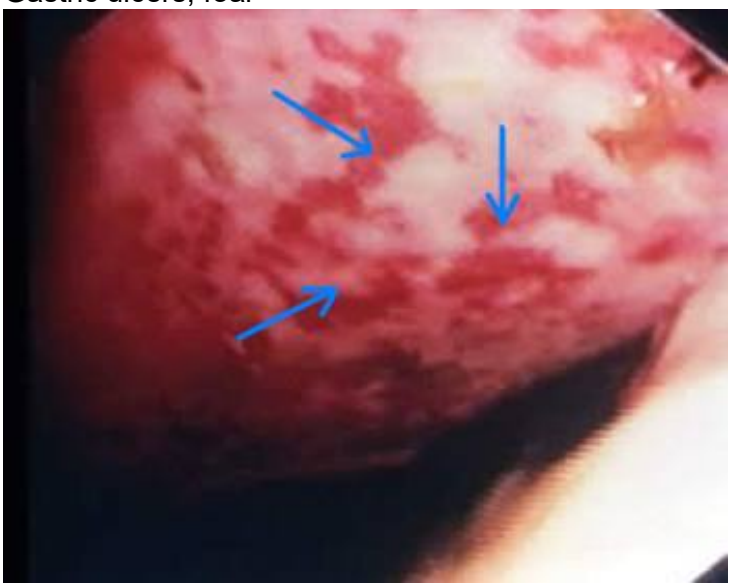
Adult horses with EGUS have nonspecific or vague clinical signs, including poor performance, abdominal discomfort (colic), poor appetite, mild weight loss, poor body condition, and attitude changes. Horses with signs of severe abdominal pain (colic) may have gastric ulcers; however, the ulcers are unlikely to be the primary cause of the abdominal pain. A strong correlation between the extent of ulceration and the severity of clinical signs has not been reported.

Complications related to gastric ulcers are most frequent and severe in foals, but they can also occur in adult horses too. They include delayed gastric emptying, gastroesophageal reflux and esophagitis, megaesophagus secondary to chronic gastroesophageal reflux, and, rarely, perforation. Clinical signs in adult horses with equine glandular gastric disease are less known; they are thought to be similar to horses with equine squamous gastric ulcer disease. Clinical signs of equine glandular gastric disease are typically vague and similar to those of equine squamous gastric ulcer disease. Severe ulcers in the pyloric region of the glandular stomach or proximal duodenum might lead to fibrosis and stricture formation. Duodenal and pyloric stricture can lead to delayed gastric emptying in adult horses, similar to what happens to foals. In rare cases, severe gastric ulceration causes fibrosis and contracture of the stomach.

Diagnosis of Gastric Ulcers in Horses

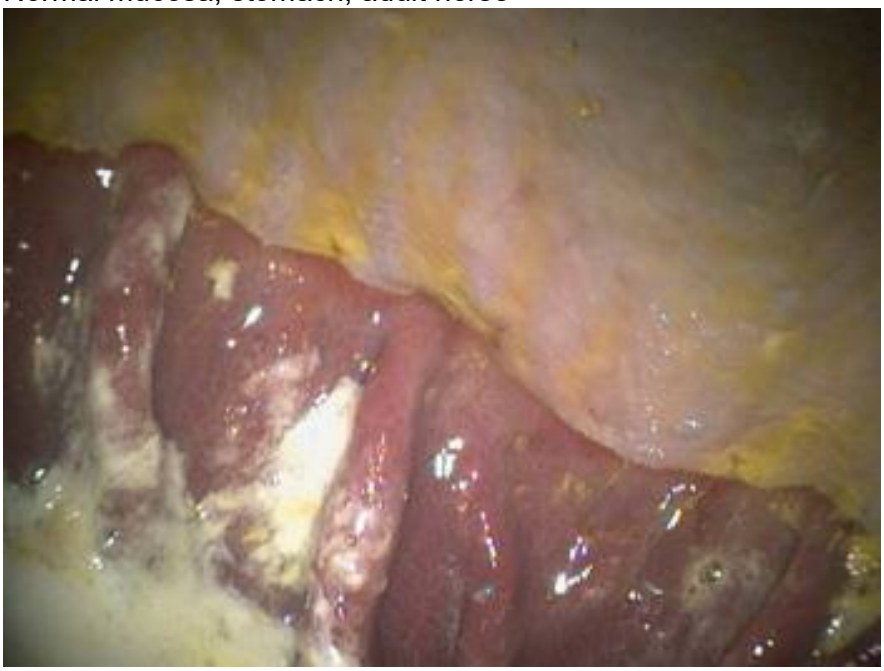
- Clinical signs (albeit vague and nonspecific)
- For definitive diagnosis, endoscopic visualization of lesions

Gastric ulcers, foal



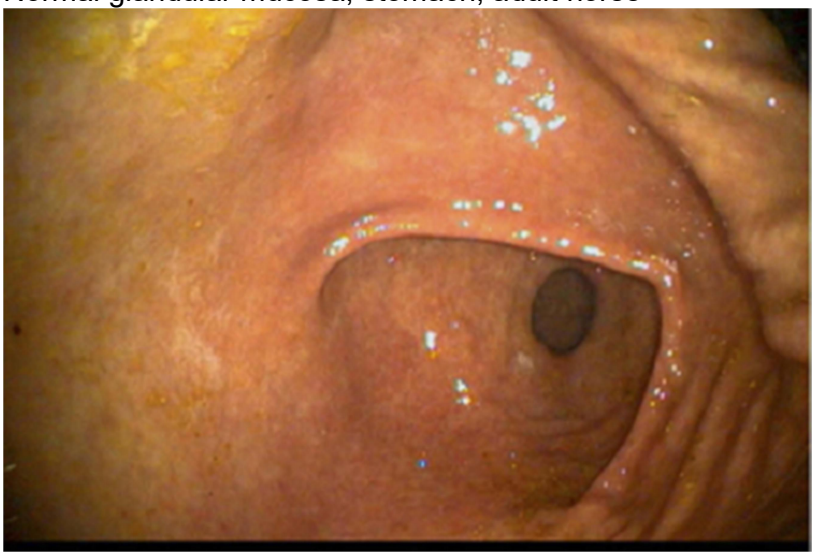
Courtesy of Dr. Thomas Lane.

Normal mucosa, stomach, adult horse



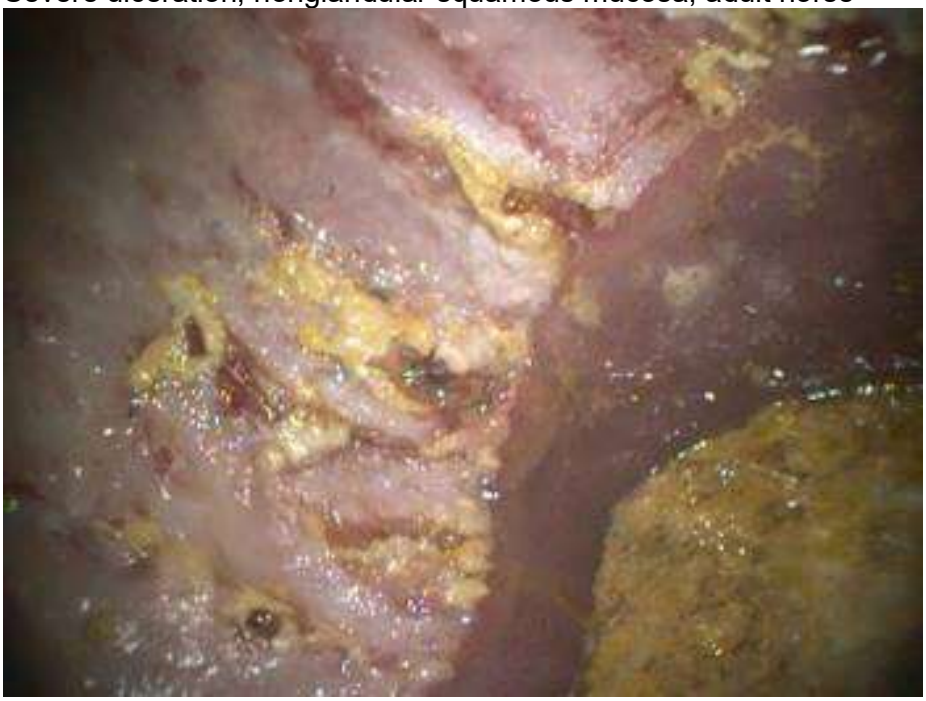
Courtesy of Dr. Frank M. Andrews.

Normal glandular mucosa, stomach, adult horse



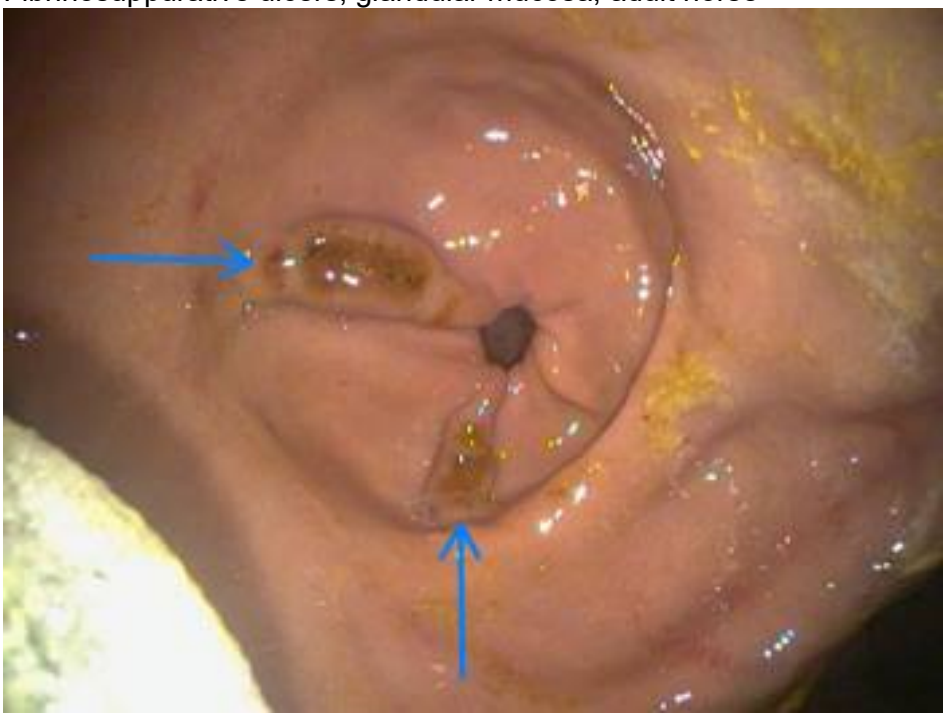
Courtesy of Dr. Frank M. Andrews.

Severe ulceration, nonglandular squamous mucosa, adult horse



Courtesy of Dr. Frank M. Andrews.

Fibrinosuppurative ulcers, glandular mucosa, adult horse



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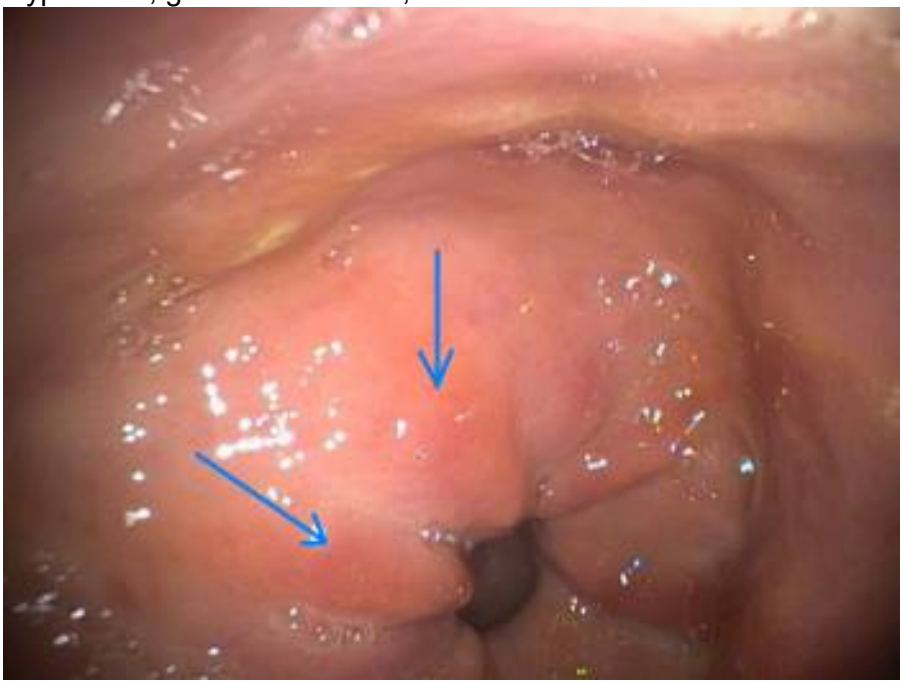
Hyperemia, glandular mucosa, adult horse



Courtesy of Dr. Frank M. Andrews.

Neither clinical signs nor clinicopathologic laboratory tests are specific for diagnosis of 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 stress because of problems in many organ systems, or as a result of environmental stress such as hospitalization or stall confinement. Gastroscopic visualization of the ulcers in an empty stomach is the only definitive method of diagnosis. The equine stomach consists of two regions: the squamous region is covered by nonglandular tissue and can have mild hyperkeratosis (yellow appearance) because of exposure to stomach acids, whereas the glandular stomach is generally pink and can look mottled.

A 3-meter-long equine gastroscope with a high quality light source is required to visualize ulcers in both the nonglandular and glandular regions of the stomach. When ulcers appear in the squamous mucosa, they are typically erosive and can be hemorrhagic; ulcers in the glandular mucosa can be mildly hyperemic or fibrinosuppurative. During the gastroscopic exam, ulcers can be graded (i.e. 0-4, from normal to severe) using an established scoring system. This allows for concise communication of lesion severity and facilitates comparison on subsequent studies, in order to determine if treatment has been effective; however, use of scoring systems may be inconsistent between clinicians. If examined histologically, lesions in the glandular mucosa typically contain inflammatory cells, including lymphocytes and plasma cells or neutrophils.

Empirical treatment is indicated when endoscopy is unavailable. A presumptive diagnosis can be reasonably made when clinical signs are reduced or resolve after several days (3–5 days) of treatment with a medication known to be effective at raising gastric pH and facilitating healing of gastric mucosa.

Treatment of Gastric Ulcers in Horses

- Supportive care and dietary management
- Omeprazole (treatment of choice, few reported adverse effects)

Supportive care consists of dietary management, including feeding low-starch grains and alfalfa hay to buffer stomach contents.

Suppression of gastric acidity and maintenance of a pH ≥4 are the primary objectives in the treatment of equine squamous gastric ulcer disease. Studies have examined the use of surface-coating agents, antacids, histamine type-2 receptor antagonists (ranitidine and cimetidine), and the proton pump inhibitor omeprazole in a carrier designed to aid passage through the acid stomach into the small intestine for absorption.

Sucralfate binds to the gastric glandular mucosa and may promote healing, although studies using sucralfate alone have not shown it to be efficacious in the treatment of equine squamous gastric ulcer disease in horses or foals. When sucralfate is used with an omeprazole paste, it can reduce the severity of equine glandular gastric disease. Antacids have yet to be proven effective in either healing or preventing gastric ulcers. They must be administered in relatively high volumes every 2 hours to neutralize stomach acid. Ranitidine (6.6 mg/kg, PO, every 8 hours for at least 28 days [1]) has been shown to be effective in healing equine squamous gastric ulcer disease lesions when horses were removed from training. Studies have not shown cimetidine to be effective in treatment of either equine squamous gastric ulcer disease or equine glandular gastric disease.

Omeprazole is a proton pump inhibitor that blocks hydrogen ion (acid) secretion by binding to and changing the configuration of hydrogen-potassium adenosine triphosphatase (H^+,K^+-ATPase) in the lumenal surface of the gastric parietal cell. It is the only medication approved by the US FDA for treatment (4 mg/kg, PO, every 24 hours) for 28 days and prevention of recurrence (1–2 mg/kg, PO, every 24 hours) of EGUS (both equine squamous gastric ulcer disease and equine glandular gastric disease) in horses, and it has been shown to allow gastric ulcers to heal in horses while they continue their normal training. Omeprazole is approved for a 28-day treatment course for EGUS, primarily equine squamous gastric ulcer disease; however, treatment of equine glandular gastric disease may require 45–60 days of treatment. Repeat gastroscopy may be used to guide duration of treatment.

Misoprostol (5 mcg/kg, PO, every 12 hours for at least 28 days), a synthetic prostaglandin analog, either alone or with sucralfate (20 mg/kg, PO, every 12 hours for at least 28 days) and omeprazole, has been shown to be effective in treating equine glandular gastric disease.

Another pharmacological agent that shows promise is the S-enantiomer of omeprazole, esomeprazole. Esomeprazole (1–2 mg/kg, PO, every 24 hours for at least 28 days) has been shown successfully to treat equine squamous gastric ulcer disease; however, its treatment efficacy for equine glandular gastric disease is not known.

References

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Key Points

- Gastric ulcers (EGUS) in the nonglandular squamous mucosa and glandular mucosa are common in horses involved in competition.
- Risk factors include intense exercise and dietary factors, such as high concentrate diets and periods of food restriction.
- Clinical signs are often vague and nonspecific; definitive diagnosis requires endoscopic examination to identify ulceration.
- Treatment consists of pharmaceutical agents that increase gastric pH (eg, omeprazole), synthetic prostaglandins, and coating agents (eg, sucralfate).

For More Information

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