Gastric Ulcers in Horses: Clinical Signs, Diagnosis and Treatment

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The equine stomach

Horses are natural grazers that are designed to continuously eat around the clock. In relation to their size and overall feed consumption, horses have relatively small stomachs that consist of two distinct regions. The proximal (orad) portion of the stomach is the non-glandular, squamous cell-lined region while the distal (aborad) portion of the stomach is the fundic glandular portion. A stepped edge called the *margo plicatus* divides the non-glandular and glandular regions.

Ulcers form in horses' stomachs when there's an imbalance between the factors that incite erosion and the factors that protect the stomach. The most common inciting factor is hydrochloric acid while bile acids and pepsin may also play a contributing role in disease development.

Risk factors for the development of gastric erosion and ulceration include intermittent feeding, increased exercise intensity, and dietary factors such as feeding high-concentrate, low roughage diets to horses. For young horses, it's possible that illness — and the stress associated with being sick — cause ulcer development since the prevalence of gastric ulcers in critically ill foals is higher.

Clinical signs

While a horse's history and a description of clinical signs are important in diagnosing EGUS, veterinarians rely on an endoscopic examination to make a definitive diagnosis. Most practitioners use a grading system that was developed by the Equine Gastric Ulcer Council to classify the gastric lesions: the system ranges from Grade 0 (normal) to Grade 4 (severe ulceration).

• Adult horses: clinical signs of EGUS can include low-grade colic, poor body condition and decreased performance. But signs can vary: some adult horses with endoscopic evidence of gastric ulcers may show no signs or very subtle symptoms while other horses may show more typical clinical signs.

In adult horses, veterinarians most often find lesions in the gastric squamous mucosa — especially along the margo plicatus. Lesions in the *pyloric region* (opening from the stomach into the small intestine) are also important.

• Neonatal foals with gastric ulceration may suffer from colic and diarrhea, grind their teeth or salivate continuously, have little or no appetite, and tend to lie on their backs. Since very few foals with endoscopic evidence of EGUS show symptoms, chances are the ulceration is severe if you observe any of these clinical signs.

The gastric squamous mucosa is where veterinarians find most gastric lesions in young foals. Physiologic stress associated with illness has also been linked with gastric ulcers in neonatal foals: those lesions are found in the glandular epithelium.

• **Older foals:** clinical signs like diarrhea, poor appetite, poor growth and poor body conditions are associated with severe squamous epithelial lesions. Foals with duodenal ulceration often present similar clinical signs as the ones associated with gastric ulceration such as colic, teeth grinding, continuous salivation and diarrhea. They may also suffer from delayed emptying of their stomachs and gastroesophageal reflux.

Diagnosis

Gastroscopic examination

All horses are usually fasted for a period of 10-12 hours. Water is recommend to be withdrawn one hour prior to the examination. A 3 meter portable flexible videogastroscope , 10.4mm x 300cm working length, is used to conduct the gastroscopies. Horses are sedated with an intravenous administration of detomidine hydrochloride and butorphanol tartrate. A short nasogastric tube is placed into the proximal esophagus and the videoendoscope is then fed through the nasogastric tube. The endoscope is advanced into the stomach, which is insufflated and distended with air until the glandular and non-glandular portions are observed. Gastroscopic examination includes assessment of the fundus, nonglandular and glandular portions, margo plicatus and cardia. Once the endoscopic examination of the stomach is completed, the insufflated air remaining within the stomach is removed, in order to prevent potential signs of colic. The esophagus is endoscopically examined as the endoscope is withdrawn.

Lesion classification

Presence and severity grading of nonglandular lesions can be based on a lesion scoring system proposed by the Equine Gastric Ulcer Council in 1999, which ranges from grade 0 to grade 4. Grade 0 represents an intact epithelium with no appearance of hyperemia or hyperkeratosis; grade 1, an intact mucosa with areas of reddening or hyperkeratosis in the squamous portion of stomach; grade 2, small single or multifocal lesions; grade 3, large single or multifocal lesions or extensive superficial lesions and grade 4, extensive lesions with areas of deep ulceration.

Treatment

Since excess acid exposure is the main reason behind squamous mucosal erosion and ulceration, most veterinarians turn to anti-ulcer therapies with the aim of suppressing or neutralizing gastric acid.

• H_2 antagonists can successfully raise the gastric pH and resolve gastric ulcers in foals and adult horses. But the degree and duration of acid suppression by H_2 antagonists

varies from horse to horse. Practitioners routinely administer anti-ulcer drugs to critically ill neonatal foals as a prophylactic measure, but its effectiveness remains controversial. Treatment responses vary and there's also a concern that the use of prophylactic anti-ulcer therapy may suppress the function of gastric acidity in preventing bacterial translocation in neonatal foals.

• **Proton pump inhibitors** (such as omeprazole) have been effective in healing NSAID-induced gastric ulcers as well as naturally-occurring cases of EGUS. Omeprazole was effective in reducing or eliminating the severity of gastric ulcers in Thoroughbred racehorses undergoing intensive training. However, compounded preparations are not effective.

• Sucralfate is effective in treating peptic ulcers in humans, but its efficacy in treating ulcers in the equine gastric squamous mucosa is unknown. Sucralfate may be effective for treating stress-induced ulcers in neonatal foals, but so far, there's no clinical evidence to support that theory.

• Antacids can reduce gastric acidity in horses, but their effects are short-lived (last for approximately two hours) and require large doses several times a day.

• **Prokinetics** can be part of a therapy plan when veterinarians suspect delayed gastric emptying without any physical obstruction in a patient. This treatment is also useful in treating foals with duodenal disease and gastroesophageal reflux.