Prevalence, predisposing factors, diagnosis and treatment of EGUS (Equine Gastric Ulcer Syndrome).

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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 and Diagnosis

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.

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.

• H2 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 H2 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.