Equine Gastric Ulcer Syndrome in Adult Horses

Equine Gastric Ulcer Syndrome (EGUS) is prevalent in our equine population and can be a cause of suboptimal performance and behavioral issues. It has been estimated that approximately one to two thirds of adult horses have gastric ulcers depending on level of work and management practices.

Unlike humans, a horse’s stomach continuously secretes gastric acid because horses were designed to graze throughout the day. The equine stomach is divided into two parts – a smaller squamous portion (about 1/3) and a larger glandular portion (about 2/3) that are separated by a raised border of tissue called “the margo plicatus.” EGUS occurs when there is an imbalance between gastric acid secretion and the stomach’s normal protective mechanisms.

There are two syndromes of EGUS that have different predisposing factors – gastric ulceration in the squamous portion, and gastric ulceration in the glandular portion. The glandular portion of the stomach secretes acid and other compounds. It has intrinsic protective mechanisms to deal with acid and is accustomed to acid exposure. Ulceration in this portion is due to both an increased acid exposure as well as a decrease in its protective mechanisms. Gastric ulcers are more commonly seen in the squamous portion, which is not accustomed to, nor equipped for, acid exposure. The majority of gastric ulcers in the squamous portion of the stomach are along the margo plicatus, which is closest to the glandular acid-producing portion.

Factors that predispose horses to glandular ulceration include stress and administration of nonsteroidal anti-inflammatory drugs (such as phenylbutazone and flunixin meglumine). Stress decreases the stomach’s natural protective mechanisms. NSAIDs also decrease the stomach’s natural protective mechanisms, more often with high doses or long duration of treatment.

Factors that predispose horses to squamous ulceration include infrequent feedings, high concentrate diets, and intensive training. Stress and NSAID administration can augment these processes. Feed deprivation or infrequent feeding has been found to cause gastric ulcers because
when the stomach is small and contracted the squamous portion of the stomach is exposed to acid. The byproducts from fermentation of high concentrate diets can also cause acid injury of the squamous portion. Consuming hay and the salivation that accompanies eating (because saliva contains natural buffers) help to buffer stomach acid. Intense exercise potentially increases intra-abdominal pressure and delays gastric emptying time, which increases the exposure of the squamous portion to acid.

Most gastric ulceration probably goes unrecognized. Interestingly, the severity of a horse’s clinical signs does not correlate with the degree of gastric ulceration. A horse with EGUS can present with mild recurrent colic or a more severe acute colic episode. Horses with EGUS may grind their teeth, have excessive salivation, poor appetite, weight loss, rough hair coat, colic immediately after eating, poor performance, poor body condition, and changes in attitude (the horse may seem “grumpy”).

Suspicion of EGUS may be based on history and clinical signs, but evaluation of the stomach with an endoscope (a procedure called “gastroscopy”) is the only way to confirm the presence and severity of EGUS. This procedure is performed at a referral veterinary facility. The horse must be fasted for a minimum of 12 hours prior to gastroscopy so that the stomach is empty for evaluation. The horse is typically sedated and the endoscope is passed through the nostril down the esophagus into the stomach. It can be determined if the gastric ulceration is chronic or acute, and gastric ulceration is typically graded as mild, moderate, or severe.

Research is focused on a new sucrose absorption blood test, which would help to screen horses with possible EGUS and determine the need for gastroscopy. Gastroscopy would still be the diagnostic of choice to confirm the presence and severity of ulceration. A fecal occult blood test has also recently been marketed to help diagnose GI bleeding (such as gastric or colonic ulcers).

Several types of drugs are available and commonly used to treat EGUS. Antacids (such as aluminum or magnesium hydroxide and calcium carbonate) can help relieve immediate clinical signs, but do not effectively treat the ulceration. Antacids have a short duration of effect and need to be administered orally every 2 to 3 hours. Ulcer-coating agents are designed to adhere to ulcerated portions of the stomach as well as help promote some of the stomach’s own protective mechanisms. Strong evidence is lacking for their efficacy, however many veterinarians feel that they are helpful. A group of drugs called “histamine type 2 receptor antagonists” effectively suppress acid secretion by the stomach. There are oral and intravenous forms, which need to be given 3 to 4 times daily. Gastrogard™ (omeprazole) is a drug that directly blocks acid secretion. It is formulated as a flavored oral paste given once daily and has been proven to most effectively cure gastric ulceration.

Gastrogard™ given at a quarter dose once daily is effective at preventing the formation of gastric ulcers. Corn oil added to the feed has also been shown to help
increase gastric pH (i.e. decrease the amount of acid), although some horses can become “hot” with corn oil supplementation. Prevention of EGUS involves recognition of situations that may predispose a horse to gastric ulceration. Horses on high doses of NSAIDs or long-term treatment with NSAIDs are theoretically at higher risk for EGUS however EGUS is not frequently seen secondary to NSAID administration in most clinical practices. Horses in training or who are fed high concentrate meals without access to roughage are at an increased risk. Performance horses are not the only horses at risk for EGUS, as severe ulceration has been diagnosed in recreational and retired horses in seemingly calm environments. It is important to keep EGUS in mind if a horse is being exposed to potential stresses, particularly those horses that have had EGUS in the past. Recognition of clinical signs and diagnosis and treatment of EGUS could improve attitude, performance, and quality of life.

Nora Grenager, VMD

* Grew up on a small farm in central Pennsylvania
* 2000—Graduated magna cum laude with a B.A. in biology from Amherst College in Massachusetts
* 2005—Graduated magna cum laude from the University of Pennsylvania School of Veterinary Medicine
* 2005—Joined Steinbeck Country Equine Clinic and is currently finishing certification from the American College of Veterinary Internal Medicine
* Volunteer with Rural Area Veterinary Services
* House of Delegates Representative for the Monterey Bay to the California Veterinary Medical Board
* Committee Member of the International Conference on Laminitis and Diseases of the Foot
* Organizing Committee Member of Laminitis West Conference
* Member of the Monterey County Large Animal Evacuation Group

* **Publications:**

