Stomach Ulcers

What are they, and what are their implications on performance?

By Dr. Steve Fisch

EQUINE GASTRIC ULCER SYNDROME IS A MULTIFACETED HEALTH problem that is prevalent in 60 to 90 percent of adult horses. The percentage of horses affected depends on the location of the ulcer in the stomach, other health problems that the horse has at the same time, performance level, feeding routine, degree of confinement, personality of the horse with the ulcers and the personality of horses they are comingled with or near, and many other factors of management.

It is a common misconception that gastric ulcers are found only in stressed horses or horses in training. Just as with people, stress is relative to an individual horse. Other causes of EGUS are social regrouping, unfamiliar environments, weaning and lay-ups. Gastric ulcers – confirmed with video endoscopy – have been diagnosed in pastured and infrequently ridden pleasure horses.

How Ulcers Happen

WHEN THERE IS AN IMBALANCE BETWEEN STOMACH MUCOSAL aggressive factors such as stomach acids and other degradative enzymes and mucosal protective factors such as bicarbonate production and mucous secretion, the stage is set for the development of EGUS.

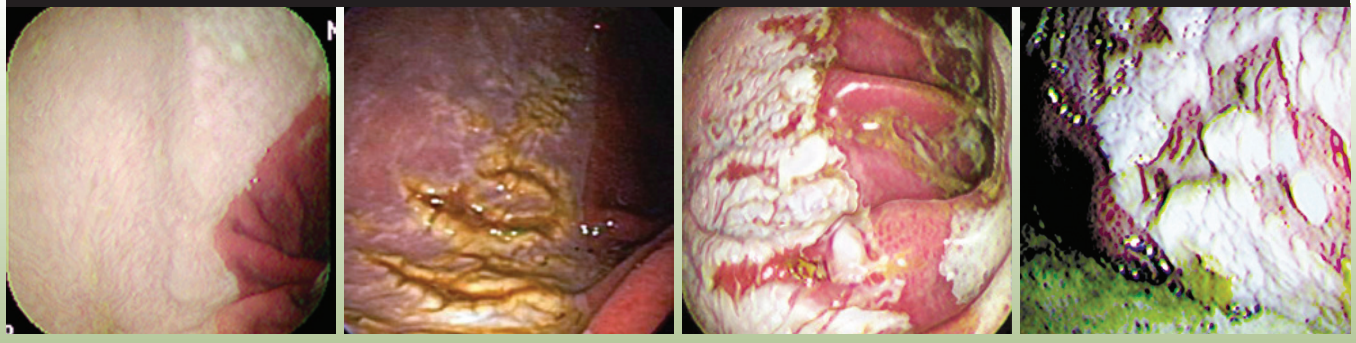
The stomach is divided into glandular and non-glandular sections. The glandular section has more protective factors than the non-glandular section, and therefore the cause of the ulcers in either section might be different.

Certain prostaglandins such as prostaglandin E2 promote secretion of the mucous layer and promote blood flow. Prostaglandins also promote the production of protective phospholipids, enhance mucosal repair and promote the health of the stomach in other ways. Therefore, if prostaglandin production is decreased, mucosal blood flow and the secretion of mucous and bicarbonate is decreased, and the secretion of gastric acid increases.

Stresses such as training, stall confinement, foaling (for both mare and baby) and hauling might lead to the release of steroids by the horse's body, and because steroids counteract prostaglandin, a vicious cycle begins. The decrease in the production of prostaglandin leads to the breakdown of mucosal protective factors, which can lead to the development of ulcers.

Exposure to acid is required for the glandular mucosa to ulcerate. However, if the mucosal protective factors are in place, the ulcerations might not occur. Nonsteroidal anti-inflammatory drugs inhibit the production of prostaglandin E2, which leads to increased acid production, decreased mucosal blood flow and degradation of the bicarbonate-

Photos of the equine stomach. From left, a healthy stomach lining, gastric ulcers, Grade 2 gastric ulcers, and a severely ulcerated stomach.



Dr. Joe Fisch scopes a horse for gastric ulcers.

KELLEY FISCH

mucus barrier. There is evidence that the medication furosemide, or Salix, acts to increase gastric blood flow by increasing production of prostaglandin E2 and thereby protecting the stomach from ulceration.

Ulceration in the non-glandular area of the stomach is caused by prolonged exposure to hydrochloric acid, certain enzymes and volatile fatty acids. The non-glandular area of the stomach has no mucous layer. This section of the stomach responds to acid irritation by hyper keratinization, or increasing the thickness of the keratin layer of the stomach. With video endoscopy, the increased thickness of the keratin layer can be seen as dense yellow plaques.

Low stomach pH is a major cause of gastric ulceration in adult horses. Low stomach pH is a result of high gastric acid levels in the stomach. The equine stomach continuously secretes gastric acid. Gastric acid levels are lowest when the horse is consuming feed. Feed consumption also stimulates the production of saliva, which is high in bicarbonate that neutralizes the low pH of the gastric acid. Stomach pH decreases six hours after feeding. Therefore horses should be on pasture or fed hay continuously or at least every six hours to keep the gastric pH in balance.

VFAs are present in the equine stomach in quantities great enough to cause injury. Many performance horses consume high amounts of VFA-rich grain diets. Horses fed high grain diets have been shown to have much higher incidence of EGUS than horses fed a primarily hay diet.

Feed deprivation, such as when hauling or between meals in a stall, are a cause of non-glandular mucosal irritation because of the persistent exposure of the non-glandular mucosa to high acid conditions. Horses fed hay continuously have higher median 24-hour gastric pH (almost twice as high) than horses that have not been fed hay within six hours.

Horses fed a legume hay, such as alfalfa or perennial peanut hay that is high in protein and calcium, have significantly higher gastric pH than horses fed straight grass hay. However, it only takes about three pounds of the legume hay per day to obtain the higher pH effect. The high calcium or protein in the legume hay has a protective effect on the stomach mucosa.

High-roughage diets stimulate the production of bicarbonate-rich saliva, which buffers gastric acid.

Diagnostics

HORSES IN TRAINING HAVE GREATER SEVERITY AND HIGHER prevalence of gastric ulcers. Exercise can cause delayed gastric emptying and increased gastric acid secretion. This might be caused by the increased intra-abdominal pressure and resulting gastric compression during exercise. This increased pressure pushes acidic contents into the section of the stomach nearest the esophagus, which is where most ulcers occur.

In different studies of horses in training – whether it be racing, western performance, dressage or hunter/jumpers – horses that had a complaint of poor performance had a 60 to 90 percent incidence of EGUS confirmed with gastroscopy.

EGUS is not always easy to recognize and diagnose. Most horses with ulcers have subclinical signs such as irritability, poor hair coat, lack of "heart" and desire, "not racing fast enough" and ADR ("ain't doing right"). The more overt signs are colic, gastric rupture, weight loss, poor body condition, poor appetite, poor performance, etc.

However, the severity of the clinical signs does not always coincide with the severity of the ulcers. Since silent ulcers are a

ANDREA CAUDILL

Omeprazole is available in the form of a USDA-approved drug in the medications GastroGard and UlcerGard by Merial.

very common presentation of EGUS, it is very important to have horses with any of the clinical symptoms properly diagnosed.

EGUS is diagnosed through evaluation of the horse's history, clinical signs, results of endoscopy of the stomach and response to treatment. Besides the symptoms mentioned previously, other clinical signs include excessive salivation, teeth grinding, gastric reflux, depression, poor appetite and abdominal pain.

Endoscopy with a three-meter endoscope is required to confirm the presence of gastric ulcers in the adult horse. With the endoscopic examination, the location and severity of the lesions can be observed. Follow-up endoscopic examinations can be used to evaluate response to treatment.

The lesions are usually graded 1-3, with a 0 score having no lesions; 1 having intact mucosa but mild reddening and mild thickening of keratin layer of cells; 2 indicating large single or multifocal lesions or extensive superficial lesions; and 3 indicating extensive lesions with areas of apparent deep ulceration.

Treatment

THERE ARE SEVERAL TREATMENTS FOR EGUS. THESE TREATMENTS include omeprazole, sucralfate, antacids, H-2 receptor antagonists and a few others that are meant to stop the physiological pathway of ulcer development.

Antacids that are commonly available increase the gastric pH for about two hours post-administration and might decrease signs of EGUS for a very short time, but there appears to be no long-term effect. These antacids would have to be given six times per day to affect the pH in a consistent manner, but even then there has been no evidence that these medications are effective in healing gastric ulcers.

H-2 receptor antagonists suppress gastric acid secretion by binding to histamine (H-2) receptors. H-2 receptor antagonists include medications such as ranitidine and cimetidine. Ranitidine with every-eight-hour dosing has been shown to treat and heal gastric ulcers. Cimetidine, even with every-six-hour dosing, has not been shown to heal equine gastric ulcers.

Omeprazole completely suppresses acid secretion by binding with the cell membrane H+-K+ proton pump. The effects of omeprazole can last as long as 27 hours. A dose of omeprazole can suppress gastric acid production within 30 minutes and has been noted at a dose of 4mg/kg to provide significant healing of gastric ulcers in horses receiving NSAIDs when given once daily for 28 days. In one study, horses in training receiving omeprazole at a dose of 1mg/kg for 28 days, 82 percent remained free of gastric ulcers when compared to the control group where only 10 percent remained ulcer free. Omeprazole is available in the form of a USDA-approved drug in the medications GastroGard and UlcerGard by Merial.

EGUS is primarily a quiet disease. Many aspects of the disease can be prevented by the way we manage horses, such as keeping them as stress free as possible and gradually introducing them to stressful situations so that instead of being stressful, those situations become nonstressful or at least less stressful. A few pounds per day of legume hay will go a long way to act as a buffer in the stomach.

EGUS is just another aspect of total health management for athletes, and the difference between acknowledging the problem and ignoring it can be the difference between a healthy and happy horse and a sick, poor-doing horse, or the difference between winning and losing. Remember that taking care of the horse is job No. 1. Everything else is secondary.

Dr. Steve Fisch owns AVS Equine Hospital, a full-service hospital and reproductive center in Tallahassee, Florida, where he and son Dr. Joe Fisch operate a referral hospital for performance-related lameness. He is also a racehorse owner and a member of the AQHA Racing Council. For his efforts in bringing American Quarter Horse racing back to Florida, Dr. Fisch was the recipient of the 2010 Gordon Crone Award. To comment, write to jrnlracing@aqha.org.