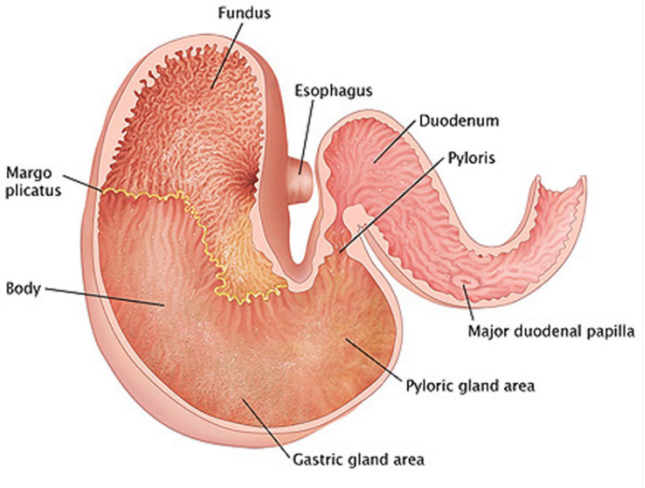
EQUINE GASTRIC ULCER SYNDROME

Gastric ulcer disease is common in all horses and the term Equine Gastric Ulcer Syndrome (EGUS) has been used because of its many causes and complicated nature. It is estimated that 25 to 50 percent of foals and 60 to 90 percent of adult horses are afflicted

Although ulcers in foals and horses can be similar, the syndromes frequently have different causes and may produce different clinical signs.

The Stomach

The horse stomach is divided into two distinct regions: the non-glandular (squamous) region and the glandular (non-squamous) regions. The squamous mucosa covers approximately 1/3 of the equine stomach and is void of glands. This portion of the stomach is designed for mixing of the contents and does not have as much protection from the acid. The glandular region covers the remaining 2/3 of the stomach and contains glands that secrete hydrochloric acid, pepsin, bicarbonate and mucus. A sharp demarcation, called the margo plicatus, separates the non-glandular from the glandular mucosa.



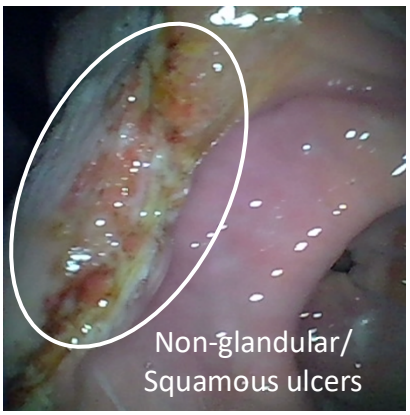
The stomach continuously secretes variable amounts of acids throughout different parts of the day, and secretion of acid occurs without the presence of feed material. The adult horse produces approximately 1.5 liters of gastric juice hourly. Gastric emptying of a liquid meal occurs within 30 minutes, whereas complete emptying of a roughage hay meal takes up to 24 hours.

Clinical Signs

The majority of horses with gastric ulcers do not show outward symptoms. Clinical signs in foals include intermittent colic (after suckling or eating), frequently rolling onto their back, intermittent or interrupted nursing, diarrhea or history of diarrhea, poor appetite, grinding their teeth, and excess salivation. Clinical signs in adults include poor appetite, dullness, attitude changes, decreased performance, reluctance to train, decreased body condition, rough hair coat, weight loss, excessive recumbency, and colic. It is important to note that the severity of clinical signs does not directly correlate to the severity of disease.

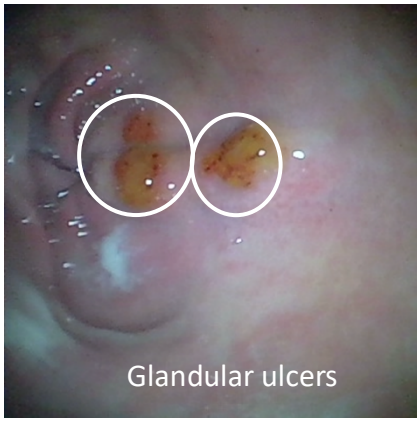
The Ulcers

Gastric ulcers in foals (less than 50 days of age) and adults are commonly located in the non-glandular region of the stomach adjacent to the margo plicatus. However, horses with concurrent medical issues or being given non-steroidal anti-inflammatory drugs (NSAIDs) such as bute or banamine may have gastric ulcers located in the glandular region of the stomach.



The severity of squamous ulcers is probably related to length of time of acid exposure. The mucosa near the margo plicatus is constantly exposed to acid and this region is where gastric ulcers are frequently found. Ulcers in the glandular mucosa are primarily due to disruption of blood flow, decreased mucus and bicarbonate secretion.

Glandular ulcers are often found in the lower part of the stomach, where the stomach meets the small intestines. This part is called the pylorus. These ulcers are less common in the general population, but are an important cause of gastric pain.


Glandular ulcers

Causes

Equine Gastric Ulcer Syndrome results from a disparity between mucosal destructive factors (acids) and mucosal protective factors (mucus and bicarbonate). Since mucosal protective factors are more developed in the glandular mucosa, different causative mechanisms may lead to ulceration in this region compared to the non-glandular mucosa. Ulcers in the non-glandular mucosa are primarily due to prolonged exposure to stomach acids.

Prostaglandins (a hormone) may help maintain the integrity of the mucosa by stimulating production of surface-active protective fats, stimulating mucosal repair, and preventing cell swelling. A decrease in prostaglandins leads to a breakdown in mucosal protective factors and may be the primary cause of glandular gastric ulcers in foals and horses.

Feed

Horses are designed to be grazers with regular intake of roughage. Since the horse's stomach continually secretes acid, gastric ulcers can result when the horse is not eating regularly due to there being less feed to neutralize the acid and an increased exposure of the non-glandular mucosa to the acids. When horses are fed two times per day, the stomach is subjected to a prolonged period without feed to neutralize the acid. In foals, infrequent or interrupted feeding and increased time laying down has been shown lead to lower gastric pH.

In yearling and adult horses, hay and saliva (rich in sodium bicarbonate), help buffer gastric acid. The timing of feeding and the type of roughage source may contribute to gastric ulceration. In studies, horses fed hay continuously had less acidity, when compared to those that were fasted. In another study, horses fed alfalfa hay had significantly less acidity and lower gastric ulcer scores, than horses fed bromegrass hay. The high protein and calcium concentration in alfalfa hay provides buffering of stomach acid for up to five hours after feeding. Also, high roughage diets stimulate production of bicarbonate rich saliva, which contributes to the buffering of gastric acid.

Many horses, especially performance horses, are fed diets that are high in fermentable carbohydrates. High-grain diets produce volatile fatty acids (VFAs) that can contribute to the development of ulcers by causing a more pronounced acid injury to the non-glandular mucosa of horses. These VFAs enter the stomach tissue causing cell damage, inflammation and ulceration.

Housing

Stall confinement alone can lead to the development of gastric ulcers. Horses maintained without direct contact to other horses and horses kept and trained in urban areas are more likely to develop squamous gastric ulcers.

Stress

Stress (both environmental and physical) can increase the likelihood of ulcers. Even typical training and recreational showing have been shown to induce ulcers within a five to seven day period. In addition, the mechanical aspects of exercise and the abdominal pressure may be sufficient to this provide prolonged exposure. Exercise, especially at high levels, has an inhibitory effect on gastric emptying. The stress of parturition in foals, of training, of confinement in horses and that of illness, also leads to increased release of

endogenous steroids, which has a negative impact on the GI tract. These impacts include decreased gastric motility, delayed gastric emptying and decreased gastric blood flow.

Medication

Chronic administration of any non-steroidal anti-inflammatory drugs such as phenylbutazone, flunixin meglumine or meloxicam, can decrease the production of the stomach's protective mucus layer, making it more susceptible to the formation of ulcers in the glandular portion of the stomach. Other medications such as antibiotics can destroy the natural microbial population of the GI tract and make it more likely that an individual would develop ulcers.

Diagnosis

Diagnosis is based on the presence of clinical signs and confirmation with endoscopic examination. A definitive diagnosis can only be made using a video endoscope. The endoscope must be at least 7 feet long, though an 11 foot endoscope is ideal.

To perform this, the stomach must be empty, so most horses are held off feed for 16 to 24 hours and not allowed to drink water for 4 hours. With light sedation, and possibly a twitch, the endoscope is passed through the nostril and down the esophagus into the stomach. The light and camera on the end of the endoscope allow the veterinarian to observe the stomach lining.

Treatment

Treatment of ulcers is aimed at removing the predisposing factors and decreasing acid production. When possible, horses should be allowed free-choice access to grass or hay. Environmental factors also need to be addressed. This may include changing the relationships with other horses or the horse's job description. Horses that must be stalled should be arranged so they can see and socialize with other horses as well as be allowed constant access to forage. Some horses appear to enjoy having a ball or other object in the stall to occupy their time. Decreasing types of grain that form the volatile fatty acids may help some horses. The energy from the grain can be replaced by using a feed higher in fat.

Inhibiting gastric acid secretion is the mainstay of medical treatment for non-glandular ulcers. Currently, there is only one FDA/CFIA approved treatment for gastric ulcers: GastroGard (*Omeprazole paste, Merial Limited, Atlanta, GA*). It is an "acid pump inhibitor" and inhibits gastric acid secretion regardless of the stimulus. GastroGard is a paste and must be administered at the appropriate time to see maximal effects. It is also labeled for prevention of recurrence of gastric ulcers at ½ dose. The medication contained in GastroGard. Treatment for glandular ulcers can become quite complicated and different medications can be used at different frequencies and different dosages. If these are found in your horse then your veterinarian will work to come up with the best possible plan.

There are many supplemental products on the market to help with prevention of gastric ulcers. None of them are sufficient to treat ulcers, but can be a valuable additive to your daily feed regimine. Some of the top feed supplements recommended for prevention of GI ulceration are SecureGuard, SecureGuard Gold and MadBarn Visceral+. Other supplements such AFX or sucralfate can also be beneficial, but their use should be done under the guidance of a veterinarian.