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*Gastric Ulcers* - Mar 31st, 03

Gastric ulcer disease is common in foals and horses and the term Equine Gastric Ulcer Syndrome (EGUS) has been used to describe this disease because of its many causes and complicated nature. Prevalence estimates have been reported to range from 25 to 50 percent in foals and 60 to 90 percent in adult horses, depending on age, performance, and evaluated populations.

Gastric ulcers have been identified in the non-glandular stratified squamous mucosa, margo plicatus, glandular mucosa and pyloric regions of the equine stomach. Two age related clinical syndromes have been described, one in foals (< 9 months of age) and the other in yearlings and adult horses (> 9 months of age). Although ulcers are similar in foals and horses, the syndromes frequently have different inciting causes and may produce different clinical signs. A diagnosis of these clinical syndromes relies on recognition of clinical signs and endoscopic examination of the stomach.

### **The Horse Stomach**

The horse stomach is divided into two distinct regions, the esophageal or non-glandular region and the glandular region. The esophageal region or squamous mucosa covers approximately one-third of the equine stomach, is void of glands, and is covered by stratified squamous epithelium similar to the esophagus. The glandular region covers the remaining two-thirds of the stomach and contains glands that secrete hydrochloric acid, pepsin, bicarbonate and mucus. A sharp demarcation or margo plicatus (cuticular ridge) separates the squamous mucosa from the glandular mucosa. Gastric ulcers in foals (less than 50 days of age) and adult horses are commonly located in the non-glandular region of the stomach adjacent to the margo plicatus along the greater curvature and lesser curvature. However, foals and adult horses with a concurrent medical disorder or being given non-steroidal anti-inflammatory drugs (NSAIDs) (Bute or Banamine) may have gastric ulcers located in the glandular region of the stomach near the pylorus. Foals, and to a lesser extent in adult horses, may have duodenal ulcers, which may lead to gastric and esophageal ulcer, secondary to delayed gastric emptying.

The horse stomach continuously secretes variable amounts of hydrochloric acid throughout the day and night and secretion of acid occurs without the presence of feed material. Foals secrete gastric acid as early as 2-days-of-age and acidity of the gastric fluid is high. High acid in the stomach may predispose foals to EGUS. The adult horses, the stomach secretes approximately 1.5 liters of gastric juice hourly and acid output ranges from 4 to 60 mmoles hydrochloric acid per hour. The pH of gastric contents ranges from 1.5 to 7.0, depending on region measured. A near neutral pH can be found in the dorsal portion of the esophageal region (saccus cecus) near the lower esophageal sphincter, whereas, more acidic pHs can be found near the margo plicatus

(3.0-6.0) and in the glandular region near the pylorus (1.5-4.0). Gastric emptying of a liquid meal occurs within 30 minutes, whereas complete gastric emptying of a roughage hay meal occurs in 24 hours.

### **Causes of Clinical Syndromes of Gastric Ulceration in Foals and Horses**

Equine Gastric Ulcer Syndrome in foals and horses results from a disequilibrium between mucosal aggressive factors (hydrochloric acid, pepsin, bile acids, organic acids) and mucosal protective factors (mucus, bicarbonate). Since mucosal protective factors are more developed in the glandular mucosa of the equine stomach when compared to the squamous mucosa, different causative mechanisms may lead to ulceration in these regions. Ulcers in the squamous mucosa are primarily due to prolonged exposure to hydrochloric acid, pepsin, bile acids or organic acids. Ulcers occurring in this region are similar to Gastroesophageal Reflux Disease Syndrome (GERDS) in humans, since this region lacks well-developed protective factors, similar to the esophagus.

The severity of squamous ulcers is probably related to length of time of acid exposure. The squamous mucosa near the margo plicatus is constantly exposed to these acid and this region is where gastric ulcers are frequently found in foals and horses. Ulcers in the glandular mucosa are primarily due to disruption of blood flow and decreased mucus and bicarbonate secretion, which results in back diffusion of hydrogen ions and damage to the underlying submucosa. Inhibition of prostaglandins may play a major role in the pathogenesis of gastric ulcers in the glandular region of the equine stomach. Gastric ulceration in the squamous mucosa is directly related to the degree and severity of gastric acid exposure.

Several factors have been implicated in causing ulceration and these include, fasting, gastric acid clearance (gastric motility and emptying), aggressiveness of the gastric juice (acid, pepsin, bile acids, organic acids) and the process of desquamation. Fasting is an important factor in causing ulcers in the squamous mucosa in foals and adult horses. In foals, infrequent or interrupted feeding and recumbency has been shown lead to lower gastric fluid pH in foals. These findings suggest that milk may have a buffering effect on gastric acid and recumbency may increase exposure of the squamous mucosa to acid. Low gastric pH from interrupted or infrequent nursing may play a role in the cause of squamous ulceration in foals.

Feed deprivation has been shown to cause ulcers in the squamous mucosa of horses, which is due to repeated exposure of the squamous mucosa to high acidity. In yearling and adult horses, hay and saliva (rich in sodium bicarbonate), may help buffer gastric hydrochloric acid. The timing of feeding and the type of roughage source may contribute to gastric ulceration in yearling and adult horses. In a study, horses fed hay continuously had less acidity, when compared to horses that were fasted. In another study, horses fed alfalfa hay had significantly less acidity and lower gastric ulcer scores, than horses fed bromegrass hay. High protein (21 percent) and calcium concentration in alfalfa hay provides buffering of stomach acid up to five hours after feeding. Also, high roughage diets stimulate production of bicarbonate rich saliva, which may contribute buffering of gastric acid.

Gastric motility and emptying may play a role in squamous mucosal ulcers in foals and horses. In humans with GERDS, acid clearance time and consequent exposure of the esophageal mucosa to potentially injurious agents is inversely proportional to the rate of gastric esophageal and

gastric motility. Delayed gastric emptying or decreased gastric motility could potentially increase exposure of the squamous mucosa to gastric juice and other aggressive factors leading to ulceration. In neonatal foals with concurrent disease or with a gastric outflow obstruction, decreased gastric motility and/or delayed gastric emptying may lead to prolonged acid exposure and ulceration, especially during periods of squamous cell desquamation.

In adult horses, the prevalence of gastric ulcers is high in the performance horse and may be due to prolonged exposure of acid to the squamous mucosa. The mechanical aspects of exercise and the abdominal pressure may be sufficient to provide prolonged exposure of the non-glandular mucosa to aggressive factors. Furthermore, especially in racehorses that perform at near maximal levels, exercise may have an inhibitory effect on gastric emptying.

Decreased gastric and esophageal motility and delayed gastric emptying have been implicated in the cause of GERDS in humans during exercise and may lead to gastric ulceration in the performance horses, especially the racehorse. Other organic acid may act synergistically with hydrochloric acid to play a role in the pathogenesis of gastric ulcer disease in horses.

Recently, volatile fatty acids (VFAs), fermentation byproducts of carbohydrates, were found to induce acid injury to the gastroesophageal (squamous) mucosa of horses. The VFAs easily penetrate the squamous mucosa of the stomach when acid concentrations are high. These VFAs enter the stomach tissue causing cell damage, inflammation and ulceration. In a previous report, VFAs were found to be present in the stomach of horses in significant enough quantities to lead to acid injury. Since performance horses are fed diets that are high in fermentable carbohydrates, VFAs, generated by resident bacteria, may cause acid injury and ulceration in the squamous mucosa.

Other gastric aggressive factors such as, Bile salts, from duodenal reflux and pepsin, have been implicated in causing gastric ulcer disease in other species and possibly the horse. Bile acids, in combination with pepsin act to increase the permeability of the esophageal mucosa to hydrogen ions. Furthermore, bile acids have been shown to act synergistically and in a dose-dependent manner with hydrogen ions to cause damage to the squamous mucosa of pigs. These studies suggest that pepsin and bile acids may contribute to the production of squamous ulceration in horses. In foals, gastric ulceration may be related to desquamation or "shedding" of the squamous epithelium of the stomach. Desquamation of the squamous mucosa, occurs in 80 percent of foals up to 35 days of age. In a study of rats, it was found that the loss of epithelial cells along the margo plicatus resulted in the increased susceptibility of this region to acid injury. Also, acid injury to this region resulted in a delay in reepithelialization.

Delayed reepithelialization could result in acid injury of the deeper layers from hydrochloric acid and lead to gastric ulceration. Glandular gastric ulcers occur most frequently in foals, but can occur in adult horses. The cause of glandular gastric ulcers is most likely due to decreased blood flow and decreased mucus and bicarbonate secretion. Decreased prostaglandin synthesis (primarily PGE<sub>2</sub>, I and A) has been implicated in the cause of glandular gastric ulcers in foals, since non-steroidal anti-inflammatory drugs (NSAIDs) administration caused gastric ulcers in foals. Blocking prostaglandin synthesis causes decreased mucosal blood flow, stimulates gastric acid secretion, and inhibits bicarbonate secretion by the glandular mucosa.

Prostaglandins may also help maintain the integrity of the squamous and glandular mucosa by

stimulating production of surface-active protective phospholipid, stimulating mucosal repair, and preventing cell swelling by stimulating sodium transport. Furthermore, stress of parturition in foals and stress of training and confinement in horses, may also lead to excess release of endogenous corticosteroid, which can inhibit prostaglandin synthesis. 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.

## Diagnosis

The diagnosis of EGUS is based on the presence of clinical signs and confirmation with endoscopic examination. Clinical signs in foals include intermittent colic (after suckling or eating), frequent dorsal recumbency, intermittent nursing (interrupted nursing due to discomfort), diarrhea or history of diarrhea, poor appetite, bruxism (grinding of teeth), and ptyalism (excess salivation). The later two signs are often signs of an outflow obstruction, such as pyloric obstruction.

Clinical signs in other horses include poor appetite or failure to consume a meal, dullness, attitude changes, poor appetite, decreased performance, reluctance to train, poor body condition, rough hair coat, weight loss, excessive recumbency, and low-grade colic. A presumptive diagnosis of EGUS can be made on these typical clinical signs and response to therapy. A definitive can only be made using a video or fiberoptic endoscope. The endoscope must be at least 7 feet long. A longer endoscope (11 feet) is necessary to observe the duodenum in adult horses. A shorter scope (5-6 feet) is sufficient to see the stomach of foals.

## Treatment

Inhibiting gastric acid secretion is the mainstay of gastric ulcer treatment in horses. A number of treatment modalities have been used for treatment and prevention of gastric ulcers in horses and foals. Currently, there is only one FDA approved treatment for gastric ulcers in horses, GastroGard (Omeprazole paste, Merial Limited, Atlanta, GA). However, many treatment modalities have been described in the literature. GastroGard (Omeprazole) is one of the most studied medication in horses. It is an "acid pump inhibitor" and inhibits gastric acid secretion regardless of the stimulus. GastroGard is a paste and is given to horses once daily for 28 days to treat EGUS. It is also labeled for prevention of recurrence of gastric ulcers at ½ dose. The medication contained in GastroGard is the same medication found in the "Purple Pill" Prilosec that is currently sold to humans for treatment of gastric ulcers.

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