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GASTRIC ULCERS IN HORSES: A WIDESPREAD BUT MANAGEABLE DISEASE

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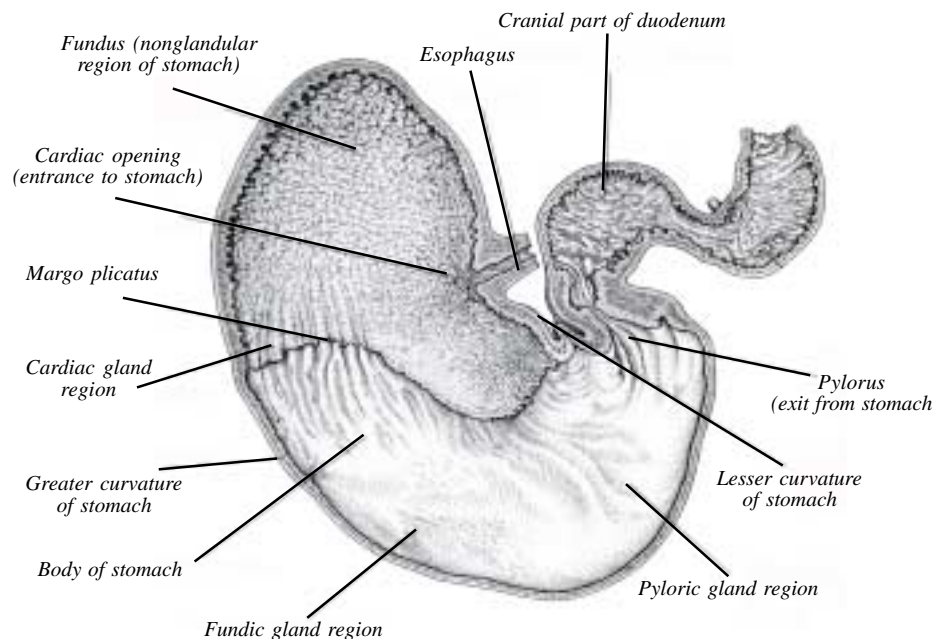
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Every equine practitioner appreciates the delicate nature of the equine gut. Problems related to the small intestine and large intestine are well understood and routinely treated. What may be surprising to many is how often the stomach is affected.

Specifically, the incidence of gastric ulcers is extremely high, particularly in performance horses. This article will review why horses suffer from gastric ulcers and provide guidelines for their treatment and prevention.

Many studies since the mid-1980s have documented that gastric ulcers are commonplace in racehorses. An early postmortem study in Hong Kong (Hammond et al., 1986) of 195 Thoroughbred racehorses showed that 80% of the horses in active training had ulcers. The incidence of ulcers in horses retired from racing for one month or longer was 52%. Murray et al. (1989) examined the stomachs of 187 horses ranging in age from one to 24 years. Eighty-seven horses had clinical problems including chronic, recurrent colic, poor body condition or chronic diarrhea. One hundred horses had no clinical signs of gastrointestinal problems.

Figure 1.



Ninety-two percent of the horses with clinical problems had gastric ulcers. Surprisingly, 52% of the horses displaying no clinical signs also had lesions. Racehorses in training had a higher incidence of ulcers (89%) than non-racers (59%). More recently, two studies evaluated the incidence of gastric ulcers in California racehorses. In one postmortem study (Johnson et al., 1994) of 169 horses in training, 88% of Thoroughbred horses in training had ulcers. A gastroendoscopic study of 202 Thoroughbred horses in training showed that 81% had ulcers (Vatistas et al., 1994).

Each of these studies produced remarkably similar results. 80-90% of racehorses in training have gastric ulcers. The vast majority of these lesions occur in the region of the stomach above the margo plicatus, with very few lesions in the glandular portion (Figure 1). The upper half of the horse's stomach consists of squamous epithelial cells that are very similar to the tissue found in the esophagus. Ulcers in this part of the stomach are more similar to esophagitis (heartburn) in humans than the ulcers that occur in the glandular region of the human stomach. It has also been determined that equine gastric ulcers are not caused by *Helicobacter pylori* bacteria, which are a common cause of ulcers in humans.

Gastric Acid is a Major Cause

Dr. M.J. Murray of the Marion duPont Scott Equine Medical Center in Leesburg, Virginia has proposed that the major cause of gastric ulcers in horses is prolonged exposure of the squamous mucosa to gastric acid.

Unlike the glandular portion of the stomach, this tissue does not have a mucous layer and does not secrete bicarbonate onto its luminal surface. The only protection that this portion of the stomach has from gastric acid and pepsin comes from saliva production. If adequate saliva is not produced to buffer the gastric acid and coat the squamous epithelium, gastric irritation occurs and lesions may develop.

The high incidence of ulcers seen in performance horses is a man-made problem resulting from the way that we feed and manage these horses, since ulcers are extremely rare in horses maintained solely on pasture. Horses evolved as wandering grazers with digestive tracts designed for continual consumption of forage. Meals of grain or extended periods of fasting lead to excess gastric acid output without adequate saliva production.

Horses secrete acid continually whether they are fed or not. The pH of gastric fluid in horses withheld from feed for several hours has consistently been measured to be 2.0 or less (Murray, 1992). Horses that received free choice timothy hay for 24 hours had mean gastric pH readings that were significantly higher than fasted horses (3.1 in fed vs 1.5 in fasted horses)(Murray and Schusser, 1989). Higher pH readings in hay-fed horses should be expected since forage consumption stimulates saliva production. Meyer et al. (1985) measured the amount of saliva produced when horses ate either hay, pasture or a grain feed. When fed hay and fresh grass, the horse produced 400-480 grams of saliva per 100 g of dry matter consumed. When a grain-based feed was offered, the horses produced only about half (206 g/100 g dry matter) as much saliva.

Grains and pelleted concentrates also increase the production of gastrin, a hormone that stimulates gastric acid production (Smyth et al., 1988). Hay fed alone affects gastrin production to a lesser degree. Therefore, horses that are fasted or that are fed high grain diets are more likely to produce more gastric acid with less saliva than horses offered free choice forage without grain.

It is easy to see why racehorses have such a high incidence of ulcers. Most horses in training are confined for most of the day and fed large grain meals. Often, racehorses are fasted for an extended period before exercise, allowing gastric acid to accumulate in the stomach. Intense exercise further increases the production of gastric acid so that the squamous mucosa of the stomach gets thoroughly bathed in acid during work.

Treatment and Prevention of Ulcers

Treating ulcers involves either inhibiting gastric acid secretion or neutralizing the acid produced. There are three classes of drugs that can be used to inhibit gastric acid secretion:

- 1) Histamine type-2 antagonists (H_2 antagonists). H_2 antagonists act by competing with histamine for histamine type-2 receptor sites on the parietal cell, and therefore blocking histamine-stimulated gastric acid secretion. The two most popular H_2 antagonists used in horses are cimetidine (Tagamet) and ranitidine (Zantac).
- 2) H^+/K^+ ATPase inhibitors. Direct inhibition of the proton pump can be achieved by substituted benzimidazoles. The only proton pump inhibitor licensed in the US and Europe is omeprazole.
- 3) Prostaglandin analogues.

An alternative to suppression of acid production is to neutralize stomach acid and protect the squamous mucosa from exposure to acid. The natural buffering mechanism in the horse is from saliva production and indeed the most effective way to treat ulcers is simply to turn the animal out on pasture. In situations where this is not possible, administration of antacids may be a useful adjunct to acid suppression therapy in horses.

An Equine Antacid

For an antacid to work well in horses it must possess several properties. First, administering it should be easy, preferably by adding to the horse's feed. A small dose should neutralize a large amount of acid and it should coat and protect the mucosa from gastric acid irritation. In addition, antacids should be able to adsorb pepsin and other substances that can damage the mucosa. Finally, it should not affect fecal consistency and should create no harmful side effects.

Over the last three years, Kentucky Equine Research has worked to develop an equine antacid that would satisfy the above criteria. The first attempt at an equine antacid used ingredients that are common in human preparations. Unfortunately, when this type of preparation was fed at high doses to horses, they became ataxic and displayed signs that were similar to tying-up. Apparently, the horses were sensitive to magnesium hydroxide, one of the common antacids used for humans. Therefore, magnesium hydroxide was removed from the formula.

Another class of antacids contains aluminum. A concern with these is that the aluminum will interfere with phosphorus absorption. Although we found no detrimental effect of aluminum intake on phosphorus absorption in digestibility experiments (Pagan, unpublished, 1996), supplemental phosphorus (balanced with calcium) was added to the formula. Schryver et al. (1986) also showed that only small amounts of aluminum are absorbed by the horse and aluminum retention was not affected by level of aluminum intake. Therefore, it appears that aluminum-containing antacids can be safely fed to horses.

After a few more modifications, a commercial product was developed and patented. This equine antacid (Neigh-Lox) is in a palatable pelleted form that can be fed alone or mixed in feed. It contains a very fast acting antacid with 240 mequ/dose of acid neutralizing capacity. A four ounce (120 g) dose will neutralize about 6 hours of basal acid production. The recommended dosage for adult performance horses is 4 ounces per meal with a maximum daily intake of 16 ounces. Neigh-Lox also contains a compound that serves as a coating agent to protect the gastric mucosa. This ingredient has an astringent and anti-inflammatory action and pepsin binding capacity.

Since its development, Neigh-Lox has been field tested in hundreds of horses. Many horses that displayed signs of gastric irritation such as poor appetite, chronic colic, and sour disposition have shown immediate improvement after receiving only a few doses of Neigh-Lox. Since there are no studies to show that Neigh-Lox heals ulcers, it is recommended only as adjunctive therapy to acid suppressive drugs in horses that have been positively diagnosed with gastric ulcers. Neigh-Lox's most important role, however, may be in preventing ulcers from occurring in the first place. Antacid therapy along with a dietary program that emphasizes continual forage consumption may greatly reduce the incidence of gastric ulcers in performance horses and foals.

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