DOES MY HORSE HAVE GASTRIC ULCERS??

Sometimes our horses are just not doing right and we just do not know why. We start thinking of all the possible reasons, and one of them just might click …What about gastric ulcers? You have heard of gastric ulcers and the multitude of clinical signs that accompanies the disease including colic, decreased appetite, irritability or attitude changes, diarrhea, weight loss, decreased performance, and “girthiness” to name a few, but you are not sure if your horse has been affected by this disease, what causes it, how it can be diagnosed, and how it can be prevented and/or treated. This brief summary will attempt to answer some of those questions.

Peptic ulcer disease, by definition is the development of erosions or ulcers in the gastrointestinal tract due to exposure to acid. The gastric mucosa is particularly affected by this acid which can cause inflammation, erosions and/or ulceration if acid is present in excess.

The two main areas in the stomach can be affected; the stratified squamous mucosa near the esophagus and the glandular mucosa towards the pylorus. The junction of these two areas is named the Margo Plicatus. Along the margo plicatus is the most common location for gastric ulcers in horses. The broad term, Equine Gastric Ulcer Syndrome (EGUS) encompasses a wide variety of clinical syndromes that affects different areas of the stomach. EGUS can affect horses of any breed, sex and age. Thoroughbreds in race training, Arabians, and Standardbred trotters seem to be more affected. The prevalence of ulcers within the stomach tends to be highest in the non-glandular squamous mucosa. Within the glandular mucosa, prevalence differs between the mucosa of the body of the stomach (<10%) and that of the antrum and pylorus (>50%).

One specific feature of the equine stomach is that it secretes hydrochloric acid (HCL)
continuously so that the gastric acidity of a horse or foal is very high between periods of eating or nursing. Ulcers in the squamous mucosa can be secondary to prolonged periods of not eating or nursing, intensive exercise, or delayed gastric emptying. Meanwhile the etiology of most ulcers in the glandular mucosa of the stomach is not known. The effects of different feeds on gastric acidity and development of ulcers have not been thoroughly studied, although one report indicated that alfalfa hay was associated with reduced ulcer severity in research horses. Excessive doses of, prolonged therapy with, or a sensitivity to NSAID’s (including phenylbutazone, flunixin meglumine) are known to induce ulceration in some horses.

Equine Gastric Ulcer Syndrome affects two main groups of horses: 1. foals and 2. horses in training or performing at an intense level of exercise. Most foals with gastric ulcers do not exhibit clinical signs. Clinical signs become apparent when the ulceration is widespread or severe. The classic clinical signs for gastric ulcers in foals include diarrhea, bruxism (teeth grinding), poor nursing, dorsal recumbency, and ptyalism (salivation). None of these signs are specific for gastric ulcers. In fact, ptyalism is a clinical sign of esophagitis, which in most foals, is secondary to gastric outflow obstruction and gastroesophageal reflux. Importantly, when a foal exhibits clinical signs, the ulcers are severe and should be diagnosed and treated immediately. Sudden gastric perforation without prior signs occurs sporadically and rarely in foals.

Adult horses with ulcers display nonspecific signs that can include abdominal discomfort (colic), poor appetite, mild weight loss, poor body condition, and attitude changes. Ulcers in the proximal duodenum or at the pylorus can cause fibrosis and stricture. This complication is seen in both foals and adult horses. Neither clinical signs nor laboratory tests are specific for gastric ulcers, and abnormalities found from a particular diagnostic test do not preclude the possibility that another disorder may be present. Gastric ulcers can also develop secondary to other disease
processes and problems in other organ systems. Therefore, the only accurate means of diagnosis of gastric ulcers is by endoscopy, particularly gastroscopy to assess the interior of the stomach and proximal duodenum.

Once the diagnosis had been confirmed, the primary treatment is the suppression of gastric acidity. This can be accomplished with antacids which neutralize acid present in the stomach or with medications that suppress gastric acid secretion such as histamine type 2 antagonists {(H2-blockers) including cimetidine, ranitidine, famotidine} and proton pump inhibitors like omeprazole. Omeprazole is the active ingredient in GastroGard and UlcerGard, which is effective for treatment and prevention of ulcers, respectively. Another method of therapy is the use of medications that act on gastric mucosal protection. Sucralfate promotes the healing of lesions in the equine glandular mucosa by binding to the ulcerated mucosa, stimulating mucus secretion, and enhanced mucosal blood flow and prostaglandin E synthesis. With prompt and proper treatment, the prognosis for healing is good to excellent.

Always keep in mind that a good balanced diet with plenty of fresh water and regular exercise is essential to maintain a healthy gastrointestinal (GI) tract and a healthy horse. If you have further questions about any GI issue or think that your horse might be suffering from gastric ulcers, please contact New England Equine Medical and Surgical Center for further information or evaluation.

Loures Rivera, DVM, MS
Jacqueline Bartol, DVM, DACVIM