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## Equine Gastric Ulcer Syndrome (EGUS)

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### Introduction

Equine gastric ulcer syndrome (EGUS) is characterized by ulceration of the distal esophagus, stomach (proximal squamous or nonglandular mucosal part, distal glandular part) and proximal duodenum. EGUS represents a significant clinical and economic problem in horses due to its high prevalence, nonspecific clinical picture and negative effect on the horse's performance. All ages and both sexes of horses are susceptible to EGUS. Even the exact ethiology is not clear yet, factors including exercise, stress, feed management, administration of nonsteroidal drugs, microbial infection and several others have been discussed. Identifying the real cause of gastric and duodenal ulceration in horses can lead to specific therapy and thus disease eradication. Recent antiulcer treatment options are expensive and their effect is usually only short-term. Therefore the recurrence of EGUS is common and it increases the clinical importance of this disease.

### Mechanism of the disease

An increase in aggressive factors including acid content, decreased pH and decrease in protective mucosal factors have been confirmed as causes for gastric ulceration in horses. Horses are continuous gastric acid secretors. The hydrochloric acid and gastric pH lower than 4.0 are the most important pathogenetic causes of gastric ulceration in horses. The proximal third of the equine stomach is lined with nonglandular stratified squamous epithelium. Nonglandular stomach mucosa is predisposed to acid injury because it does not secrete any protective substance such as mucus or bicarbonate. Gastric pH is higher in the nonglandular part of the equine stomach in comparison with the glandular part. HCl induces injury in this mucosal type by damaging the outer cell barrier, followed later by diffusion into the squamous cells of the *stratum spinosum* resulting in inhibition of cellular sodium transport, cell swelling and ulceration. The distal two thirds of the equine stomach are lined with glandular mucosa. This mucosal part is responsible for hydrochloric acid (HCl) and pepsinogen secretion as well as for protective mucus and bicarbonate layer production. The dense capillary network in the glandular mucosa is an important factor in mucosal secretion and its healing properties, but gastric ulcerations affect both mucosal parts in horses.

## Prevalence

Equine gastric ulcer syndrome seems to be extremely prevalent in the equine population. EGUS was first described as a serious highly prevalent disease of foals in 1964. Later, frequent occurrence of the syndrome in adult horses was also found. According to various authors, prevalence of the disease varies from 55% to 100%. Erosion and ulceration of the gastric mucosa is a common finding in racehorses. Racing Thoroughbreds were found to have a prevalence as high as 97%. Equine gastric ulceration syndrome is highly prevalent in Standardbred racehorses too (63% in the Czech Republic). Similarly high EGUS prevalence was found within endurance horses. Horses used for pleasure, riding lessons or show have a lower EGUS prevalence (37%) and severity of gastric ulcers. Recently a surprisingly high prevalence of gastric ulcers was found within a broodmares population kept at pasture. These results suggest that multiple risk factors influence the occurrence and development of gastric ulceration in horses.

## Risk factors

The etiology of equine gastric syndrome is multifactorial and many risk factors have been studied including exercise, stress, stomach microbial infection, age, sex, breed, non-steroidal drug administration, horse diet and feeding management. Various results were obtained.

Intense exercise has been proven as a risk factor for EGUS. The mechanism is that compression of the stomach by the abdominal viscera and diaphragm leads to the release of acid contents into the proximal region of the stomach which is lined with squamous mucosa. This is thought to result in low pH secretion onto the nonglandular mucosa which could result in mucosal injury. The predisposition for ulceration of the nonglandular mucosa along the *margo plicatus* and on the lesser curvature in comparison with the *saccus caecus*, could be explained by this theory. It was proven that the prevalence and severity of gastric ulceration increases with the duration of race training, endurance training and simulated race training.

According to the positive correlation between training or racing intensity and gastric ulcer occurrence, it is supposed that stress associated with these activities plays an important role in equine gastric ulcer pathogenesis. Severe stress may impair gastric mucosal flow leading to mucosal hypoxia, autodigestion and subsequent ulceration. It is considered a contributing factor for ulcers found in the glandular portion of the stomach. Stress accompanies exercise in horses as well as clinical disorders, environmental factors and other variables. In Australia, the horses trained in urban areas were 3,9 x more likely to have gastric ulcers in comparison with horses trained in non-urban areas. Time in work, crib-biting, difficulty maintaining bodyweight and playing a radio in the barn were all identified as other risk factors.

Infection with *Helicobacter pylori* is of primary importance in the etiology of gastric ulceration in man. The role of *Helicobacter* sp. in the horse is still unknown, but recently widely discussed. *Helicobacter* genus-specific (*Helicobacter*-like) DNA was found, but its relationship to mucosal lesion has not been proven. In addition a new *Helicobacter* species, *Helicobacter equorum*, was isolated from fecal samples of clinically healthy horses. These bacteria colonize the distal gastrointestinal tract and possibly do not play any role in EGUS. It has been confirmed that a diverse population of other bacteria live within the normal equine stomach. It was found that the diversity of bacteria adherent to the stomach lining decreases during ulceration, which implies the potential development of a dominant population of pathogenic organisms.

Age and sex predisposition are frequently studied in connection with equine gastric ulceration, but a strong association is rarely found.

Administration of nonsteroidal anti-inflammatory drugs (NSAIDs) was reported as a cause of gastric ulceration in many species. However the occurrence of ulcers is high even in horses with no history of drug use. Nonsteroidal anti-inflammatory drugs (phenylbutazone, flunixin meglumine), contrary to common belief, appear to have little impact on the development of gastric ulceration in horses. This is usually related to high dose and/or frequent administration of NSAIDs. Ketoprofen in comparison with phenylbutazone and flunixin meglumine has been found to have the less ulcerogenic effect to equine gastric mucosa. The less ulcerogenic effect of suxibuzone in comparison with phenylbutazone has recently been described. On the other hand, the more recent study did not show the difference between ulcerogenic effect of suxibuzone or phenylbutazone in horses given their therapeutic doses for two weeks. The NSAIDs combination therapy could result in a higher ulcerogenic risk to the horse than single treatment.

A high grain low roughage diet has been proven to be a risk factor in gastric ulceration in the horses. High concentrate diets are high in digestible carbohydrates, which are fermented by bacteria, resulting in the production of VFAs. The role of VFAs in EGUS ethiopathogenesis is proven. Horses fed a concentrated diet (and low roughage) have higher prevalence and higher severity of EGUS in comparison with horses at pasture. Low roughage leads to decreased chewing and decreased saliva production. Salivary bicarbonate is a gastric buffer and protects the gastric mucosa against ulcer development. A high starch diet is fermented to VFAs and lactic acid. Therefore a high starch diet should be avoided in horses prone to gastric ulceration. Alfalfa hay was proven to protect equine gastric mucosa against ulceration by increasing stomach pH. Calcium and protein seem to be responsible for buffer effect of alfalfa hay. Gastric pH and ulcer score were lower in horses fed a diet including alfalfa hay in comparison with the same horses fed dietary brome or coastal Bermuda hay. Therefore, alfalfa hay may have a protective and antiulcer effect in horses.

Grazing horses seem to have decreased EGUS prevalence because there is continuous saliva production which buffers the stomach environment. When feed is withheld from horses (before racing, in stabled horses during the night) gastric pH drops rapidly and the

nonglandular mucosa is exposed to an acid environment which predispose it to gastric ulceration. Intermittent and irregular feeding has been proven to cause and increase EGUS severity in the nonglandular stomach part.

### Clinical picture

Clinical signs are non-specific in equine gastric ulceration. Acute and recurrent colic, diarrhoea (in foals), poor hair coat, poor appetite, weight loss, attitude changes, depression and poor performance are described in horses which suffered from gastric ulceration. Partial anorexia (poor general appetite) is one of the nonspecific clinical sign which accompany the syndrome of gastric ulceration in horses. Poor performance is frequently mentioned as a common sign of gastric ulceration, but only one study showed a direct association. Evaluation of a larger number of horses is needed to prove that poor performance is indeed a sign of equine gastric ulceration.

### Diagnosis

*Ante mortem* diagnosis of equine gastric ulcer syndrome is based on history, clinical signs, gastroscopic examination and treatment response. Two point seventy-five meter to three point thirty meter gastroscopes are used to visualize the pyloric region and proximal duodenum. Many scoring systems have been developed for gastric ulcer classification in the horse. Biopsy samples are usually taken, but are not necessary for reliable diagnosis of EGUS. Other indirect diagnostic methods described include faecal occult blood testing (FOBT), sucrose permeability tests and serum alpha1-antitrypsin detection. There are no haematologic or biochemical markers which can be used to diagnose EGUS. If gastroscopy is not available it is recommend giving an antacid to which a local anesthetic (lidocain) is added through a nasogastric tube into the stomach of suspected horses. If gastric ulcers are the cause of abdominal pain, the pain should disappear within 15 minutes. If abdominal pain continues, then gastric ulcers are less likely and other possible causes should be considered.

### Complications

Gastroduodenal ulceration can lead to pyloric or duodenal stenosis which is the most common cause of delayed gastric emptying syndrome (gastric outflow obstruction syndrome) in horses. Duodenal and gastric ulceration can be complicated by perforation leading to fatal septic peritonitis (especially in foals). The prevalence of duodenal perforation in adult horses is not well documented, but it appears to be low. Chronic cholangiohepatitis and pancreatitis following gastroduodenal ulceration and duodenal stenosis in foals was also described.

### Treatment and management

Current therapy for equine gastric ulcer syndrome tries to provide pain relief, ulcer healing and prevention of secondary complications. Medical treatment is based on blocking gastric acid secretion and encouraging the subsequent increase in gastric pH. This higher pH creates a suitable environment for ulcer healing. Surgical treatment is an option for delayed gastric emptying following EGUS. Prevention of recurrence is focused on dietary management.

Treating pain originating from stomach ulceration in the horse is difficult. NSAIDs (especially repeated administration) should be avoided due to their role in EGUS pathogenesis. If necessary, ketoprofen or firocoxib may be used. The best choice seems to be use of alpha-2 adrenergic blocking agents (xylazine or detomidine, detomidine being longer acting) in combination with a synthetic opioid (butorphanol). CRI of lidocain is also recommended.

Ulcer healing is promoted by antacid therapy. Proton pump inhibitors (omeprazole) offer a better option than H<sub>2</sub> antagonists (ranitidine, cimetidine) in stomach ulceration treatment. They can be used once daily and their antiulcer effectiveness is long acting. Omeprazole (4 mg/kg of body weight orally every 24 hours) inhibits gastric secretion in horses and it was proven to be effective in EGUS treatment and prevention. It is difficult to recommend the duration of pharmacologic treatment. Gastric ulcerations (similarly to skin wounds) in horses are individual. Endoscopic examination is recommended after two weeks of omeprazole treatment to evaluate ulcer healing.

Medical treatment of horses with pyloric stenosis usually leads to a satisfactory clinical improvement. Permanent administration of omeprazole is recommended and these horses can survive for at least two years. Interruption of this therapy leads to a rapidly worsening status. Prokinetics can also form a part of the therapeutical regime for horses with gastric outflow obstruction (betanechol, cisaprid).

Coating and binding agents such as sucralfate and bismuth subsalicylate can promote ulcer healing in the equine stomach. Sucralfate is the hydroxyl aluminium salt of sucrose octasulfate and in the stomach environment is converted to a sticky mass covering the mucosal lesions. Sucralfate is widely used in neonate foals where antacid therapy can lead to increased risk of nosocomial infections.

The use of synthetic prostaglandin 1 (PGE 1) analog and somatostatin analog were studied in connection with EGUS. Prostaglandins would be most indicated in horses with NSAIDs administration or under stressful conditions. The cost limits their use in horses and it is contraindicated in pregnant and nursing mares because of its effect on the female reproductive tract.

Even though the role of the recently found *Helicobacter*-like sp. in EGUS has not been confirmed yet there are some studies focusing on the influence of antibiotics and probiotic treatment on gastric ulcer healing in horses. Probiotic preparations containing

*Lactobacillus* sp. given to horses improved healing of spontaneously occurring nonglandular gastric ulceration. Moreover the severity of nonglandular ulceration decreased with antibiotic treatment (trimethoprim sulphadimidine administration).

Surgical treatment (gastrojejunostomy, gastroduodenostomy) is an option for foals suffering from pyloric or duodenal ulceration or strictures accompanied by delayed gastric emptying syndrome.

## Prevention

The recurrence of gastric ulceration in horses is common, therefore preventive strategies for environmental, nutritional and dietary management have been developed and their effect is discussed. As is mentioned above, exercise, dietary management, stall conditions and diet are all considered as risk factors for EGUS. The protective role of alfalfa hay and the risk from a high starch diet are discussed above within risk factors of EGUS section. The following section considers the role of dietary supplements in treatment and prevention of EGUS.

Numerous dietary supplements have been developed for equine gastric ulcer supportive treatment but the majority of them lack scientific confirmation of their efficiency. For example, seabuckthorn berry extract does not have the effect of on equine gastric ulceration healing, but there is a possibility of a preventive use.

Feeding dietary oils (corn oil) is a popular management practice for horses suffering from gastric ulceration. Some results confirmed their effect while the others did not. Dietary oils are at least the very good option for energy supplementation in horses instead of starch which is contraindicated in horses suffering from EGUS.

Despite the widespread use of antiulcer treatment, the prevalence of EGUS remains high. This could be probably due to the cost of antiulcer medicaments which leads to shorter courses of treatment than is recommended, the administration of subtherapeutical doses or substitution of other ineffective medications or feed supplements.

## Further readings:

*Nadeau, J.A., Andrews, F.M.* Equine gastric ulcer syndrome: The continuing conundrum. *Equine Vet J*, 2009, 41, 611-615.

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