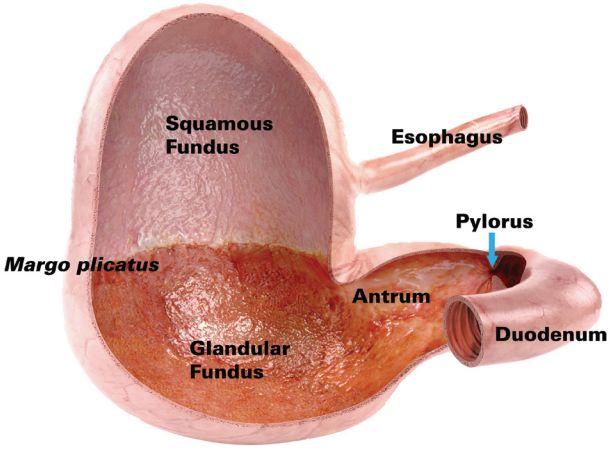
EQUINE GLANDULAR GASTRIC DISEASE AND EQUINE SQUAMOUS GASTRIC DISEASE:
How Are They Different?

Clinical signs such as poor performance, behavior change, girthiness, low-grade recurrent colic and/or inappetence might all lead a horse owner or veterinarian to suspect gastric ulcers. Equine gastric ulcer syndrome (EGUS) is the term commonly used to describe ulcers or erosive lesions in the stomach of the horse. New information, however, continues to highlight that not all ulcers are created equal, and EGUS is actually made up of two different diseases.

The equine stomach has two different linings, and over recent years we have learned to distinguish two distinct diseases, depending on where within the stomach the lesions occur. This has resulted in the current nomenclature of equine squamous gastric disease (ESGD) and equine glandular gastric disease (EGGD). The two conditions are distinct yet sometimes overlapping entities, as evidenced by differences in pathophysiology, prevalence, risk factors and response to treatment.[1]

The squamous mucosa lining the dorsal-proximal one-third of the stomach has little means of its own protection, and is reliant upon the presence of saliva and forage to buffer acid. Gastric acid is produced by the glandular epithelium lining the distal two-thirds of the stomach. Unlike in humans and dogs, acid production occurs continuously in horses, regardless of feeding patterns. Therefore, the glandular mucosa has several inherent mechanisms to protect itself from the acid it is producing. These include mucus production, bicarbonate secretion, growth factors and blood supply to allow rapid healing as damage occurs.



Note: Image represents an empty, air-filled stomach as observed during gastroscopy.

PATHOPHYSIOLOGY AND RISK FACTORS

A low stomach pH is the primary cause of ESGD, occurring when the unprotected squamous mucosa has increased duration of contact with acid (hydrochloric acid, volatile fatty acids and bile). Therefore, risk factors for ESGD are most commonly associated with feeding practices and increased levels of stress. Pasture turnout and feeding of alfalfa hay are considered practices that may help to reduce the risk of ESGD,[2] while increased time between forage meals (> 6 hours between meals), intermittent access to water, and intake of large quantities of concentrates (especially those high in starch) increases, the likelihood of ESGD.[3] Stall confinement also has been implicated as a risk factor for ESGD. In addition, intense exercise increases abdominal pressure, decreases stomach volume, and allows acid from the glandular region to splash up to the squamous region.[4]

The pathophysiology of EGGD is less well understood, but seems to result from disruption of the normal defense mechanisms, leaving the glandular tissue now susceptible to acid damage. The factors that contribute to the breakdown of the barrier are under continued investigation in the horse, but stress and inflammation likely play a role. Risk factors for EGGD are also still under investigation but may include Warmblood breeds,[5] frequency of exercise ≥ 6 days per week,[6] and sensitivity to stress (as evidenced by increased cortisol responses to exogenous ACTH).[7] Interestingly, in both polo ponies[8] and show jumpers,[9] EGGD has been inversely correlated to the experience level of the horse. This suggests that lack of consistency may contribute to stress and thereby EGGD. *Helicobacter pylori* is a common cause of gastritis in people and dogs, but research in horses has not found clear causal evidence of its role in EGGD or ESGD.[1] Ongoing microbiome work may elucidate the role of bacteria in EGGD. Likewise, the role of NSAIDs in EGGD remains unclear.[1]

PREVALENCE

The prevalence of gastric ulceration varies with breed, use, level of training, as well as between ESGD and EGGD. The highest reported prevalence of ESGD occurs in thoroughbred racehorses, with over 80% of horses affected within two to three months of race training.[2] Standardbred racehorses have a similar overall ESGD prevalence, with up to 87% of horses in training affected, while up to 58% of show/sport horses are affected.[5] Endurance horses have an ESGD prevalence of 48% during the out-of-competition period, with rises to 93% during the competitive period, with lesions most prevalent in elite horses.[7] Horses that rarely compete and are predominantly used in their home environment have the lowest ESGD prevalence of 11%.[2]

Until the past decade, prevalence data for EGGD was hard to come by. Whether it is truly increased incidence or just increased awareness, numbers are on the rise. EGGD has been reported in up to 64% of sport horses and 54% of leisure horses.[10] EGGD was more prevalent than ESGD in a group of competitive polo ponies, with 69% having glandular lesions and 54% having squamous lesions.[8] With better availability of endoscopes capable of reaching the pylorus, reports of EGGD will likely continue to increase.

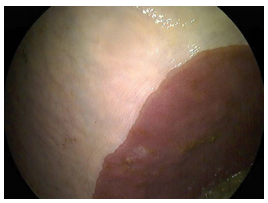
DIAGNOSIS

Gastroscopy is the only definitive diagnosis for EGUS.[2] When performing gastroscopy, it is essential to examine the entire stomach, including the pylorus and proximal duodenum, as lesions in these areas will alter treatment recommendations and expected outcome. There is not a strict relationship between the presence of ESGD and EGGD; as such, the presence or absence of one cannot be used as predictor for the presence or absence of the other.[1,2] Fecal occult blood testing has low diagnostic accuracy, and is not recommended for use in the diagnosis of EGUS.[2]

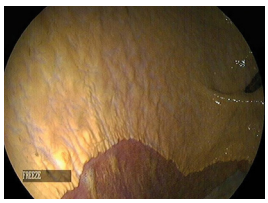
Gastric lesions should be recorded according to description and/or location of the lesions. In the case of ESGD, a well-accepted, 0-4 grading system has been established, based on repeatability and consistency. This Equine Gastric Ulcer Council 0-4 scoring system is outlined in Table 1.

Table 1. Grading system for equine squamous gastric disease (adapted from 1999 EGUS Council[11])

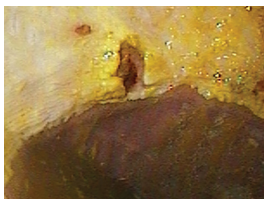
Grade	Squamous Mucosa Description
0	Epithelium is intact, no appearance of hyperkeratosis
I	Mucosa is intact, but there are areas of hyperkeratosis or gastritis
II	Small, single or multi-focal lesions
III	Large single or extensive superficial lesions
IV	Extensive lesions with areas of apparent deep ulceration



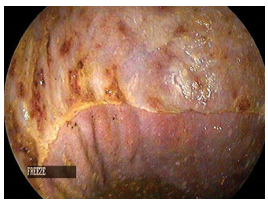
Grade 0



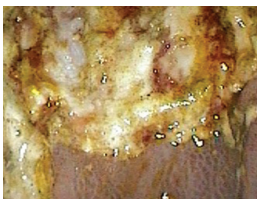
Grade I



Grade II



Grade III

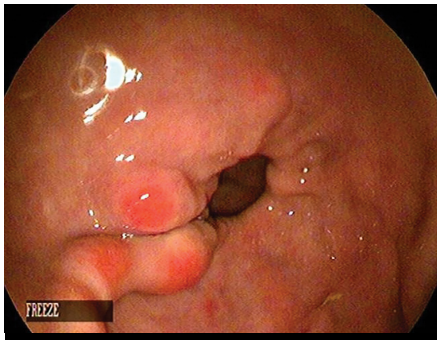


Grade IV

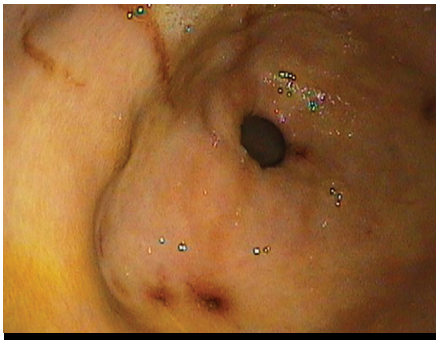
Currently, there is no agreed-upon scoring system for glandular lesions, as their relative severity and clinical importance are unknown. If EGGD lesions are identified, they should be described according to the anatomical location, distribution and appearance, as per the following recommendations:[1,2]

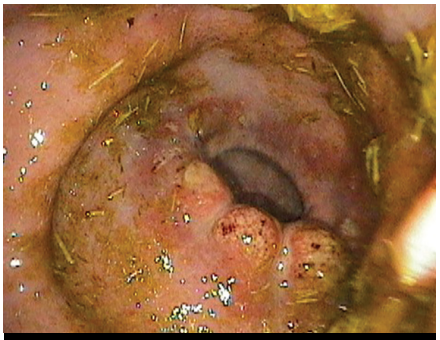
- **Anatomic location: cardia, fundus, antrum or pylorus**
- **Distribution: Focal, multi-focal or diffuse**
- **Mild, moderate or severe**
- **Nodular, raised, flat or depressed**
- **Erythematous, hemorrhagic or fibrinosuppurative**

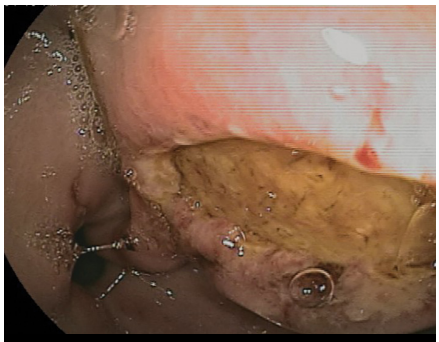
Examples of identifying equine glandular gastric disease lesions.

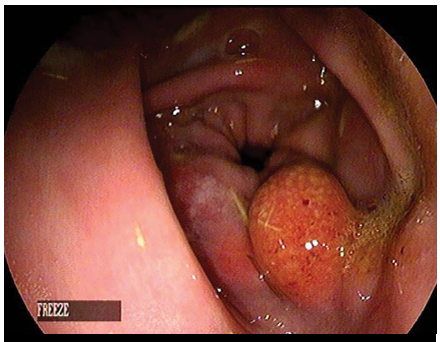


Multifocal Moderate Raised Erythematous Lesions Of The Pyloric Antrum

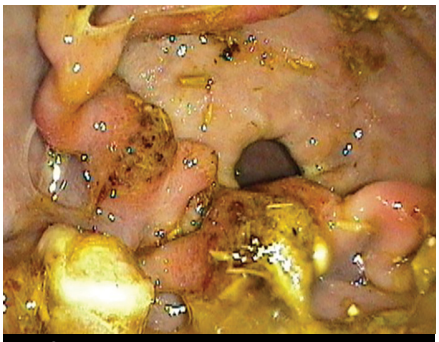

Multifocal Moderate Flat Hemorrhagic Lesions Of The Pylorus


Multifocal Moderate Raised Fibrinosuppurative Pyloric Lesions


Severe Focal Depressed Fibrinosuppurative Lesion Of The Pyloric Antrum



Polypoid Lesion Of The Pyloric Antrum


Severe Multifocal Raised Hemorrhagic And Fibrinosuppurative Lesion Of The Pyloric Antrum

Polyps or polypoid lesions in the antrum and pylorus are of particular note. These lesions may be due to hyperplasia of gastric or mucus glands, rather than true gastric ulcers, as evidenced by histopathologic examination of a polyp removed via transendoscopic electrocautery.[12] Adenomatous polyps can be precancerous in people, but the cause and outcome of these lesions in horses has yet to be determined. Glandular mucosal biopsies should be considered in cases of EGGD that are resistant to treatment, as they may reveal other pathologies such as neoplasia or parasitic infection. *Draschia* or *Habronema spp.* spend part of their life cycle in the stomach, and may play a role in inflammation and EGGD.[13]

EGUS remains a significant concern in horses and their caretakers. Further research is necessary to improve our understanding of the different pathophysiology of ESGD and EGGD, and how that likely contributes to different treatment responses and management recommendations.

REFERENCES

[1] Rendle D, Bowen M, Brazil T, et al. EGGD Consensus Statement: Recommendations for the management of equine glandular gastric disease. *UK-Vet Equine* 2018;2 (Suppl. 1):2-11.

[2] Nadeau J, Andrews F, Mathew A, et al. Evaluation of diet as a cause of gastric ulcers in horses. *Am J Vet Res* 2000;61(7):784–790.

[3] Luthersson N, Nielsen KH, Harris P, Parkin TDH. Risk factors associated with equine gastric ulceration syndrome (EGUS) in 201 horses in Denmark. *Equine Vet J* 2009;41(7):625–630.

[4] Lorenzo-Figueras M, Merritt AM. Effects of exercise on gastric volume and pH in the proximal portion of the stomach of horses. *Am J Vet Res* 2002;63(11):1481-1487.

[5] Monki J, Hewetson M, Virtala A-MK. Risk Factors for Equine Gastric Glandular Disease: A Case-Control Study in a Finnish Referral Hospital Population. *J Vet Int Med* 2016;30(4):1270-1275.

[6] Habershon-Butcher JL, Hallowell GD, Bowen IM, Sykes BW. Prevalence and risk factors for gastric glandular disease in thoroughbred racehorses in the UK and Australia [abstract]. *J Vet Int Med* 2012; 26:731.

[7] Scheidegger MD, Gerber V, Bruckmaier RM, et al. Increased adrenocortical response to adrenocorticotropic hormone (ACTH) in sport horses with equine glandular gastric disease (EGGD). *Vet J* 2017;228:7-12.

[8] Macleod H, Windeyer C, Crosby C, et al. Prevalence and risk factors for gastric ulceration in polo horses (Abstract). *J Vet Intern Med* 2015; 27:1244.

[9] Pedersen S, Windeyer C, Read E, et al. Prevalence of and risk factors for gastric ulceration in show-jumping warmbloods [abstract]. *J Vet Intern Med* 2015;29:1239.

[10] Hepburn RJ. Endoscopic examination of the squamous and glandular gastric mucosa in sport and leisure horses: 684 horses (2005-2011) [abstract], in *Proceedings* 111th International Equine Colic Research Symposium. 2014;S.

[11] Andrews F, Bernard W, Byars D, et al. Recommendations for the diagnosis and treatment of equine gastric ulcer syndrome (EGUS). *Equine Vet Educ* 1999;11(5):262–272.

[12] Marley LK, Repenning P, Frank CB, et al. Transendoscopic electrosurgery for partial removal of a gastric adenomatous polyp in a horse. *J Vet Intern Med* 2016;30(4):1351-1355.

[13] Andrews FM, Banse HE. An update on gastric ulcers: Equine Gastric Ulcer Syndrome (EGUS). *The Practitioner* 2021;3:10-20.