Advances in Diagnostics and Treatments in Horses and Foals with Gastric and Duodenal Ulcers

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KEYWORDS

• Horse • Equine • Gastric ulcer • Equine gastric ulcer syndrome • Diagnostics • Therapeutics

KEY POINTS

- Equine gastric ulcer syndrome (EGUS) primarily describes ulceration in the terminal esophagus, nonglandular squamous mucosa, glandular mucosa of the stomach, and proximal duodenum.
- There are no pathognomonic clinical signs that indicate this diagnosis; however, clinical signs, fecal occult blood, urinary sucrose concentration, and response to treatment suggest EGUS, but the only definitive diagnosis is made with endoscopic examination of the stomach.
- Lesions in the squamous mucosa improve with altered management, dietary changes, and pharmacologic agents, whereas ulcers in glandular region for the most part are less likely to improve or are slow to improve.
- Omeprazole, a potent proton pump inhibitor, is currently the drug of choice for treatment and prevention of ulcers; however, many other pharmaceutical agents including antacids, H_2-receptor antagonists, sucralfate, and prostaglandin analogues have been used alone or with omeprazole to treat and prevent EGUS.

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INTRODUCTION

Equine gastric ulcer syndrome (EGUS) is common in all breeds and ages of horses and foals. This article focuses on the current terminology for EGUS, etiologies and pathogenesis for lesions in the nonglandular and glandular stomach, diagnosis, and a comprehensive approach to the treatment and prevention of EGUS in adult horses and foals.

DEFINITION AND TERMINOLOGY

EGUS terminology was introduced in 1999[1] as an umbrella term to highlight the similarities to peptic ulcer disease in people[2] and described ulceration in the terminal esophagus, nonglandular stomach (squamous mucosa), glandular stomach, and proximal duodenum. A recent consensus statement reinforced this original umbrella terminology of EGUS (**Fig. 1**).[3]

Lesions in the nonglandular squamous mucosa (ESGD) are the result of increased exposure to aggressive factors, such as gastric acids (hydrochloric acid [HCl], volatile fatty acids, bile acids, and lactic acid) and pepsin **Table 1**.[4,5]

Equine glandular gastric disease (EGGD), lesions in the glandular mucosa, is emerging as an important disease.[3,6] HCl and a low stomach pH contribute to acid injury in the glandular mucosa. However, the mucosal defense mechanisms are likely compromised first, which allows backflow of HCl and organic acids into and between glandular cells, resulting in damage to the sodium pump **Table 1**.[7] Stress, infection with bacteria, nonsteroidal anti-inflammatory drugs (NSAIDs), and inhibition of secretion of protective prostaglandins are likely culprits.[6] Furthermore, observed lesions in the glandular stomach are frequently raised, with petechial hemorrhages and hyperemia, secondary to inflammation (**Fig. 2**). Such lesions might be an indication of inflammatory bowel disease and further evaluation of the gastrointestinal tract might be warranted, similar to Crohn disease in people affecting mucosa and deeper layers of the bowel wall and duodenum and leading to pyloric inflammation.[8]

PREVALENCE AND CLINICAL SIGNS

Adult Horses

The prevalence of ESGD varies from 40% to 90% in performance horses.[9] The prevalence of EGGD varies from 8% in thoroughbred racehorses in the United Kingdom to

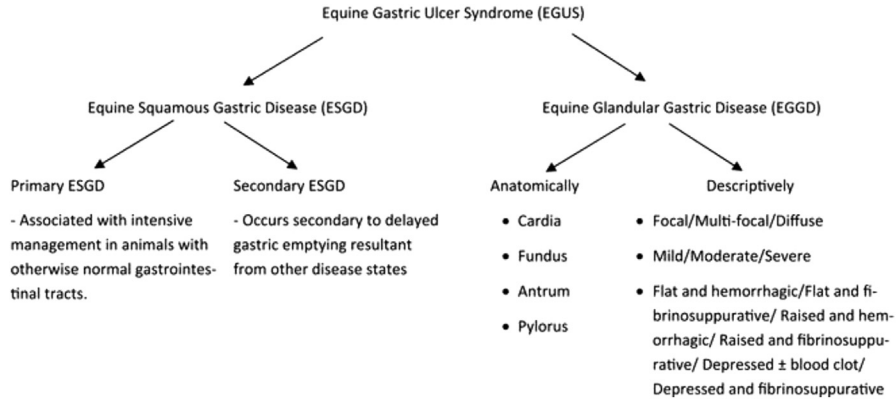

Fig. 1. Expanded terminology for equine gastric ulcer syndrome. (*From* Sykes BW, Hewetson M, Hepburn RJ, et al. European College of Equine Internal Medicine Consensus Statement—Equine Gastric Ulcer Syndrome in Adult Horses. J Vet Intern Med 2015;29:1289; with permission.)