Therapeutics for Equine Gastric Ulcer Syndrome

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KEYWORDS

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

KEY POINTS

- Equine gastric ulcer syndrome (EGUS) describes ulceration in the terminal esophagus, nonglandular and glandular stomach, and the proximal duodenum.
- Clinical signs in many cases do not clearly indicate the diagnosis of EGUS, thus a diagnosis should be confirmed by gastroscopy.
- Omeprazole, a potent proton pump inhibitor, is currently the drug of choice for treatment and prevention of recurrence; 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.
- With the reduction of medications and the avocation of Clean Sport in racing and competitions, interest has grown in effective natural supplements and better nutrition to improve stomach health.

INTRODUCTION

Although gastric ulcers have been recognized for centuries, it was in 1999 that the term equine gastric ulcer syndrome (EGUS) was introduced to better characterize and describe lesions in the terminal esophagus, nonglandular and glandular stomach, and proximal duodenum.[1] Several recent reports were published to further explain the syndrome and highlight the differences in pathogenesis of lesions in the nonglandular versus glandular stomach. A better understanding of the differences in regional pathogenesis could lead to comprehensive therapeutic and preventive strategies.[2–5] EGUS is seen in all horse and breeds and is prevalent worldwide, leading to decreased productivity and economic loss to the horse industry. EGUS is seen in foals and adult horses and the relative risk for ulceration might increase with age in geldings, whereas stallions seem to be at greater risk than mares and geldings.[6] Recently, a consensus

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statement was published to better explain the meaning of EGUS and highlight the differences in the pathogenesis of lesions in the horse stomach.[3] A better understanding of EGUS terminology might provide practitioners with clarity to better understand how to plan rational therapy.

Current therapeutic strategies for EGUS focus on blocking gastric acid secretion and increasing stomach pH. To date, there is only 1 registered pharmacologic agent for treatment and 1 pharmacologic agent for prevention of EGUS (GastroGard paste and Ulcergard paste; Merial Limited, Duluth, GA). However, a more comprehensive approach to therapy for EGUS includes determining and correcting the underlying cause, environmental management, dietary manipulation, and pharmacologic intervention. This article focuses on current terminology used to describe the pathogenesis of EGUS and presents a comprehensive therapeutic approach to maintain stomach health. In addition, current information on effective use of natural supplements is reviewed.

DEFINITION AND TERMINOLOGY

EGUS terminology was introduced in 1999 to describe ulceration in the terminal part of esophagus, nonglandular (squamous mucosa) and glandular gastric mucosa, and proximal duodenum.[1] After introduction, there was some confusion in the meaning,[7] because early pharmaceutical trials concentrated on treatment of nonglandular (squamous) ulcers, because glandular ulcers were not observed or recognized in Thoroughbred racehorses at a high prevalence in the United States.[8] The term EGUS was originally coined to be equivalent to the term peptic ulcer disease, which is the umbrella term used to describe erosive and ulcerative disease of the stomach and duodenum in humans.[9] The consensus statement introduced new terminology to describe EGUS and how lesions differ in the nonglandular squamous mucosa (equine squamous gastric disease [ESGD]) and in glandular mucosa (equine glandular gastric disease [EGGD]). ESGD can be primary and secondary. Secondary ESGD occurs in horses with delayed gastric emptying, secondary to pyloric stenosis, and results in erosions and ulcerations in the terminal esophagus. This condition is primarily seen in foals with pyloric stenosis.

Primary ESGD is associated with intensive and multiple management factors in horses with otherwise normal gastrointestinal tracts and are likely the result of increased exposure to hydrochloric acid (HCl) and organic acids. The squamous mucosa has an osmiophilic phospholipid surfactant–like layer that contributes to the mucosal barrier, but it lacks a significant mucus and bicarbonate layer, has poor blood supply, and has a variable ability to spontaneously heal at a high rate once injured.[10,11] Previous studies indicate that primary ESGD is likely caused by mucosal exposure to HCl alone or in combination with volatile fatty acids (VFAs; acetic, butyric, and propionic acids) and lactic acid, produced by resident stomach bacteria.[12–16] When gastric juice pH is less than or equal to 4, VFAs and, to a lesser extent, lactic acid are lipid soluble and enter the squamous mucosal cells, resulting in acid injury, cell swelling, and eventual ulceration.

EGGD refers to lesions in the glandular mucosa. The glandular stomach is diverse and consists of the cardia, adjacent to the margo plicatus, which is responsible for secreting mucus and bicarbonate to protect the glandular tissue from acid injury. The fundus or ventral glandular mucosa is the largest portion of the equine stomach and is made up of parietal cells, which secrete HCl; zymogen or chief cells, which secrete pepsinogen; and enterochromaffinlike (ECL) cells, which secrete histamine to stimulate HCL secretion by parietal cells and function to maintain gastric blood