EQUINE GASTRIC ULCER SYNDROME

Equine Gastric Ulcer Syndrome (EGUS) describes a unique set of conditions that are manifested as erosions and ulcerations in the distal esophagus, nonglandular and glandular stomach, and proximal duodenum of horses. It is common in racehorses, and in the study by Orsini et al, 82% (571/798) of standardbred and thoroughbred racehorses had gastric ulcers. Horses with moderate to severe ulcers are more likely to suffer subtle clinical signs such as poor body condition, rough hair-coat, interrupted and/or slow (picky) eating, and low-grade colic, especially after eating a grain meal. These signs are subtle and nonspecific, which may not be recognized by the owners and trainers. The presence of gastric ulcers has also been correlated with poor racing performance in horses.1

The cause of EGUS is likely due to secreted and/or organic acids in gastric juice and the resultant low stomach pH. Lorenzo-Figueras and Merritt2 recently showed that intragastric pressure sharply increased at a trot and gallop due to increased intra-abdominal pressure related to the exercise. They hypothesized that this increase pushed gastric contents into the proximal region of the stomach, exposing the nonglandular squamous mucosa to acidic inorganic and organic acids. This may explain the high prevalence of gastric ulcers in the racehorses in the study by Orsini et al. The findings by Lorenzo-Figueras and Merritt are consistent with observations that the lesions regress or disappear when a horse is taken out of training or treated with an acid-suppressing drug. The corrosive potential of the gastric content might also be influenced by the diet and feeding schedule to which racehorses are subjected.2

Using the prevalence figures found in Orsini et al and other articles and the fact that 50% of horses with gastric ulcers show clinical signs, it can be estimated that between 200,000 and 400,000 racehorses may suffer from clinical signs associated with gastric ulcers. The mainstay of EGUS treatment involves inhibition of gastric acid and raising intragastric pH above 4.0 for a 24-hour period. The various medications used in the racehorses described in the article by Orsini et al included buffers (antacids), sucralfate, H2-receptor antagonists, compounded omeprazole, and proprietary omeprazole paste (GastroGard, Merial) at 2 and 4 mg/kg body weight daily.3

This article is an excerpt from Equine Med Review, Volume 14, Number 1, March 2004 by John Spunaugle RPh, Valley Vet Supply/Direct Pet Superstore.

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