EGUS: clinical signs, diagnosis, treatment and prevention

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ABSTRACT

Gastric mucosal disease is frequently recognised in equine practice, especially, but not exclusively, in actively performing race and competition horses.

Although true ulceration is commonly seen in squamous mucosal disease (Figure 1), its subjacent glandular mucosal neighbour is affected more frequently by inflammatory and erosive lesions (Figure 2). Research efforts have helped provide us with a better understanding of the aetiology of many of these cases, although some remain mysterious. Similarly, treatment and management changes with a sound evidence basis can be implemented with a high degree of success in many, but not all, horses suffering from the syndrome.

Initial suspicions of gastric mucosal disease are often based on signs such as poor performance, picky appetite, mild weight loss, sensitivity to girthing, poor coat, temperament changes or even colic, although none of these are very sensitive or specific for gastric disease.
Figure 1. Squamous mucosal ulcers at lesser curvature, which tend to heal predictably following omeprazole treatment.

Often, horses with highly suggestive clinical signs will be found to have normal mucosae, as the aforementioned clinical signs can always potentially be explained by other non-gastric factors. Furthermore, speculative investigations will occasionally find quite marked gastric lesions in horses with little clinical indication of such. Clearly, therefore, there is still much to learn about the diseases making up this syndrome and how best to tackle them.

Gastroscopy examination

Despite many efforts to develop simple predictive tests for gastric disease in horses based on blood, faeces or urine samples, no method allows diagnosis to be made, other than gastroscopy examination. Nevertheless, this is a simple procedure requiring only light sedation and pre-fasting, and is widely available in equine practice nowadays.

An obvious anatomical, and perhaps clinical, division is at the margo plicatus in the equine stomach, meaning an upper, relatively simple, squamous mucosa is distinct from a more specialised lower glandular mucosa. Although gastric lesions can arise at any stomach site, they are generally seen within fairly predictable zones.

On the caudal gastric surface (forward and to the left as you enter the stomach with a gastroscope), a rather flattened “shelf” usually appears just above the margo plicatus on the greater curvature of the stomach. Ulcers are frequently encountered here (Figure 3), perhaps as a result of slower “run-off” of splashed gastric acid. In more severe cases, these can extend cranially to the left and right of the stomach towards the lesser curvature.

The least frequently diseased area of the squamous mucosa is its most dorsal aspect, which is
least likely to be contacted by corrosive acids.

The second common site to encounter squamous mucosal ulcers is at the lesser curvature immediately below the cardia (Figure 1), which is visualised by continuing to pass the endoscope into the stomach so it deflects off the caudal gastric surface and comes to face cranially towards the cardia and pylorus.

![Figure 1](image1.png)

**Figure 2.** Inflammatory pyloric nodular lesions, which frequently appear quite resistant to treatment with acid suppressant therapy.

The margo plicatus at the lesser curvature appears to be situated slightly lower than in other areas of the stomach, meaning the squamous mucosa is more likely to have contact with gastric acid here. Further passage of the endoscope, while the tip falls ventrally, will lead to entry into the pyloric antrum in the glandular mucosal area. The vast majority of gastric glandular disease is encountered in this peripyloric area (Figure 2), although the precise explanation of this predisposition is yet to be determined.

**Pharmacological choices**

Relatively few choices are available for pharmacological treatment of gastric lesions in horses. Indeed, omeprazole is the only drug licensed for this purpose. It appears very effective at healing squamous mucosal ulcers based on a multitude of well-conducted studies. H2-receptor antagonists are not licensed for use in horses.

Ranitidine may have beneficial effects on ulcer healing at 6.6mg/kg per os when given three times daily, generally making it unattractive. Omeprazole paste was first licensed as Gastrogard (Merial) and is also now available in two similar products (Peptizole: Norbrook; UlcerGold: Zoetis). Protection of the drug against acid degradation during transit through the stomach prior to intestinal
absorption is a crucial element of equine omeprazole formulations, and debate continues regarding the relative ability of the three products to achieve this.

Once absorbed into the bloodstream, omeprazole is a reversible blocker of the H+/K+ ATPase, and has been shown to suppress gastric acid secretion for 24 hours in horses. Several studies have indicated 70% to 90% healing of squamous mucosal ulcers after a month of treatment, which is superior to H2 antagonists. Given the relatively high cost of omeprazole products, a study investigating both dose rate and strategy produced interesting findings.

When Thoroughbred racehorses with squamous mucosal gastric ulcers were given omeprazole paste one to four hours before morning exercise, it was found lower doses of 1mg/kg to 2mg/kg omeprazole were no less effective than a “full” dose of 4mg/kg in healing these ulcers. In this study, the benefits of omeprazole dosing may have been augmented by increased bioavailability when given on an empty stomach and also by neutralising gastric pH during the daily exercise period when acid is likely to be splashed on to the squamous mucosa.

A further interesting finding was the relatively poor response seen in healing of glandular lesions in treated horses, where around a third improved, a third stayed the same and a third got worse. This illustrates the concerning lack of understanding we still have about glandular mucosal disease and its treatments. The author has seen several cases where horses were found to have squamous mucosal ulceration, but normal glandular mucosae.

These cases were treated for a month with omeprazole and the squamous mucosae healed, but glandular lesions had developed during treatment.

**Barrier agents**

Use of artificial barrier agents may promote healing alongside acid suppressant therapy. Sucralfate, a hydroxyl-aluminium salt of sucrose octasulfate, is the most common. Supply difficulties with sucralfate in the UK are documented, although foreign products may be sourced with appropriate VMD approval.

Sucralfate is transformed from a white liquid into a sticky gel at low pH and binds to ulcers, offering physical protection in addition to increasing bicarbonate secretion and prostaglandin production. Given the poor understanding we have about glandular mucosal disease, this offers a logical choice – as barrier protection appears sensible regardless of how glandular lesions are formed. Apolectol, a pectin-lecithin complex, has also been subject to some trials where a beneficial barrier function is also claimed and is contained in several feed supplements.
Helicobacter species have not been shown to cause gastric ulcers in horses and, although many ulcers are often seen to be invaded by multiple bacterial species, this does not necessarily implicate the bacteria in causation. The equine stomach normally contains a high bacterial load, many of which are acid tolerant, and it should be no surprise they are found, perhaps opportunistically, in areas of mucosal compromise.

No evidence is available of bacterial pathogens being causally associated with equine gastric lesions, and some bacteria may even have a beneficial effect on ulcer healing. Responsible use of antimicrobials dictates more evidence of benefit and indication in cases of gastric ulceration should be sought prior to routine usage, although it is understandable antimicrobial administration sometimes becomes irresistible in very persistent glandular lesions.

Prostaglandin E analogues, such as misoprostol, have been used to treat gastroduodenal ulcers in humans and have a logical basis for treatment of glandular (pyloric) ulcers in horses, as this is a prostaglandin-dependent area in the equine stomach. Doses of 5µg/kg orally q8-12 hours have been used in horses and shown to raise gastric pH. No evidence of clinical efficacy has yet been demonstrated, though.

Gastric ulcers are most likely a disease of domestication and management and, therefore, have an inherent risk of recurrence unless ulcerogenic risk factors are controlled. Therefore, in addition to the previously discussed pharmacotherapy, management and dietary changes should be implemented as far as the required use of the horse allows.

**Forage**
Free access to forage helps maintain pH stratification in the stomach so the squamous mucosa is protected from gastric acid. This effect is probably at its most important during fast exercise, when acid splashing is inevitable if the stomach is empty or poorly filled, hence the use of strategic dosing as described by Sykes et al (2015)¹.

Given the marked anatomical and physiological differences between the human and equine gastrointestinal tracts, the anthropomorphic assumption that fast work is best undertaken on an empty stomach is both unfounded and potentially injurious to the equine stomach. Forage feeding should be encouraged right up to the onset of fast exercise training, although the argument for weight reduction associated with forage withholding is harder to counter during a competition or race.

Forage quality is also important, with evidence suggesting a beneficial effect of feeding high quality forages, such as alfalfa, versus bromegrass hay or coastal Bermuda grass hay, and of hay or haylage versus straw. Forage should ideally be ever-present to encourage chewing and salivation. Feeding intervals greater than six hours represent a strong risk factor (greater than five times the risk) for ulceration of the gastric squamous mucosa. Fine-weave hay nets or obstructive hay feeders may be useful in extending feeding times without encouraging excessive intake.

It is generally assumed grazing reduces the risks of gastric ulceration in horses, presumably due to similar mechanisms as those described previously for forage feeding. However, grazing horses are encountered with severe squamous mucosal and/or pyloric ulceration, so the relationship is not totally clear and might depend on factors like pasture fructan and simple sugar content, which, under certain circumstances, might possibly promote acid injury.

Associations between cereal feeding and ulceration of the gastric squamous mucosa are well recognised, with one study reporting consuming more than 1.0g starch per kg bodyweight per meal (for example, 1kg to 2kg concentrates or cereal for a 500kg horse) more than doubled the risk of having squamous mucosal ulcers.

Theoretically, beneficial mechanisms of action of dietary oils in high-fat feeds might include provision of substrate for prostaglandin synthesis (which is likely to primarily aid gastric glandular mucosal defence) or possibly by binding potentially injurious lipophilic short chain fatty acids (SCFAs) and bile acids within the gastric fluid. Possible increases in gastric emptying rates may also be found in horses fed high-fat versus high-starch diets, thereby potentially limiting SCFA accumulation.

Although one study found reduced gastric acid secretion and higher levels of potentially gastroprotective prostaglandins in horses fed small amounts (45ml) of corn oil, a further study involving feeding higher levels of dietary fat found no benefit on ulcer severity.
References

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