Equine gastric ulcer syndrome and the challenges facing clinicians

Equine gastric ulcer syndrome is the most common disease of the equine stomach. This article gives an overview of the syndrome and reviews the available literature to answer questions clinicians face when treating and managing these cases in equine practice. Equine gastric ulcer syndrome has, in recent years, been further defined into two distinct syndromes: equine squamous gastric disease and equine glandular gastric disease. Primary equine squamous gastric disease is the most common form of the disease and results from prolonged exposure of the mucosa to gastric acid in an otherwise normal gastrointestinal tract. Secondary squamous gastric disease occurs as a result of delayed gastric emptying caused by inflammatory bowel disease, pyloric stenosis, severe glandular gastric disease or gastric impaction. https://doi.org/10.12968/ukve.2024.8.S1.17

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Equine gastric ulcer syndrome is highly prevalent: squamous gastric disease is found in up to 93% of endurance horses during competition season and up to 100% of racehorses in training (Tamzali et al, 2011). It is described in equine athletes and pleasure horses alike and is not limited to domesticated horses – it has been reported in up to 60% of feral horses (Lamglait et al, 2017). Gastric ulcer syndrome is reported in foals with a prevalence of 22–57%, (Elfenbein and Sanchez, 2012). Glandular gastric disease has been reported in 71% of domesticated horses vs 30% of feral horses in a UK abattoir study (Ward et al, 2015). Squamous gastric disease is more prevalent in Thoroughbreds and Standardbreds, while glandular gastric disease is more prevalent in Warmbloods (Sykes et al, 2015a; Banse and Andrews, 2019). It is not known if this is a true breed predilection or reflective of the management practices typical in these breeds.

Equine gastric ulcer syndrome: presentation and diagnostics

Clinical signs in adult horses are typically non-specific and include weight loss, poor performance and mild colic. Dog-sitting, stretching out and becoming cast are indicative of cranial abdominal pain, and colic associated with feeding, bruxism and 'girthiness' may also be seen. Changes in behaviour, poor coat quality and increased faecal water are also described (Sykes et al, 2015a; Vokes et al, 2023). Clinical signs in foals include recurrent colic, dorsal recumbency, intermittent nursing, diarrhoea, ill-thrift, bruxism and ptyalism (Murray, 1999; Elfenbein and Sanchez, 2012). Sudden death as a result of gastric or duodenal perforation with no previous clinical signs is also reported (Rebhun et al, 1982; Traub-Dagartz et al, 1985; Becht and Byers, 1986).

Gastroscopy remains the gold standard diagnostic technique for further investigation of these cases, allowing visualisation of the oesophagus, stomach and proximal duodenum, as well as the identification and classification of any lesions. Gastroscopy is performed using a >3 m video-endoscope under standing sedation, typically after a 16-hour fast and withholding water for 1–3 hours. The horse is ideally stabled on rubber matting rather than bedding to reduce the risk of the horse eating the bedding.

The entire stomach must be examined, including the pylorus and proximal duodenum (Murray, 2002). Gastroscopy is typically well tolerated but is not without limitation, there are some associated risks which are discussed later in this article. Inter- and intra-operator agreement in grading lesions is reported as moderate in squamous gastric disease but variable in glandular gastric disease (Wise et al, 2021), and the severity of glandular gastric disease lesions established by histopathology does not correlate well with gastroscopy findings (Crumpton et al, 2015). Despite this, it is the diagnostic technique of choice. Research into whether there is a link between inflammatory markers and gastric ulcers is ongoing,

Table 1. Grading system for equine squamous gastric disease lesions

Grade 0	Epithelium intact, no appearance of hyperkeratosis
Grade 1	Mucosa intact, areas of hyperkeratosis
Grade 2	Small, single or multifocal lesions
Grade 3	Large single or extensive superficial lesions
Grade 4	Extensive lesions with areas of deep lesions

(Sykes et al, 2015a)

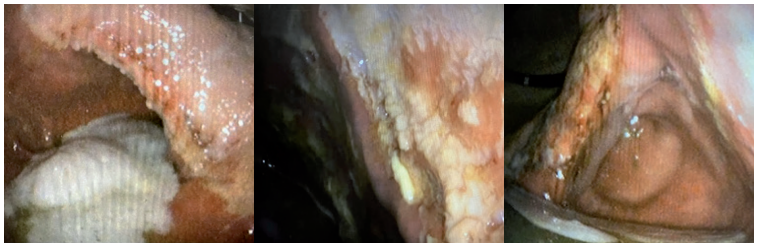

Figure 1. Examples of equine squamous glandular disease – grade 2, 3 and 4.

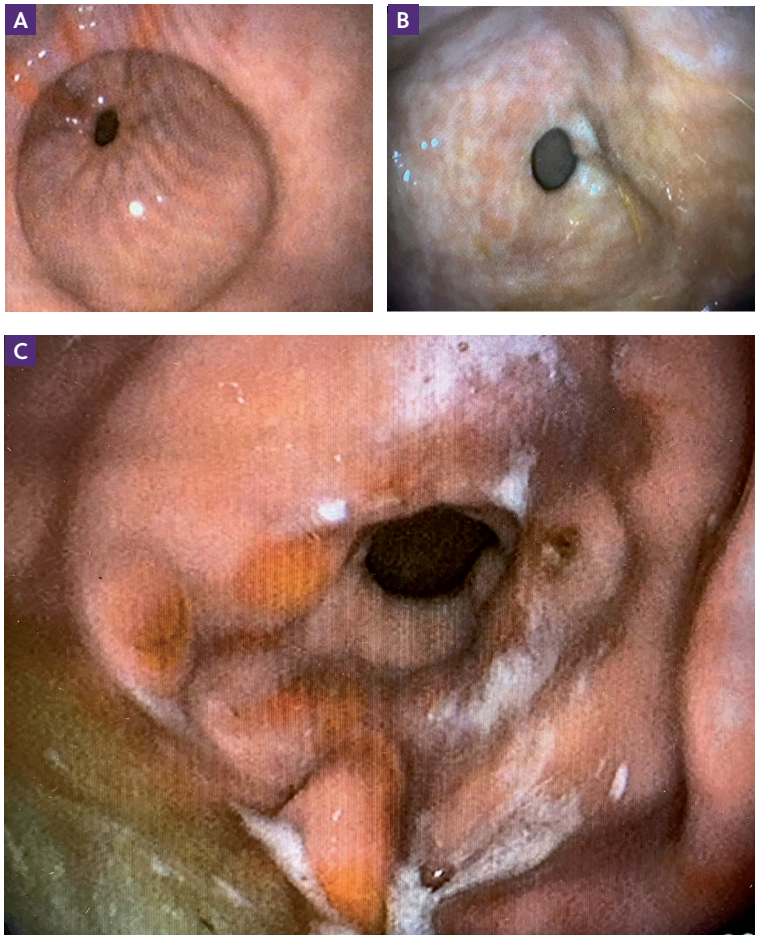

Figure 2. Examples of equine glandular gastric disease. a) Mild, multi-focal, flat, hyperaemic lesions of the pyloric antrum. b) Mild, diffuse, flat, hyperaemic lesions of the pyloric antrum. c) Moderate, multi focal, raised, fibrino- suppurative lesions of the pyloric antrum.

with some studies having conflicting results (Spanton et al, 2019a; Shawaf et al, 2020) – and the faecal occult blood test is not as useful as once thought. Research into salivary proteins shows promise (Muñoz-Prieto et al, 2022; 2023), and sucrose permeability testing may be a useful screening tool in weanlings to identify those to gastroscope, but not in adult horses (Hewetson et al, 2017; 2018).

Equine gastric ulcer syndrome has, in recent years, been further defined into two distinct syndromes: equine squamous gastric disease and equine glandular gastric disease. Primary equine squamous gastric disease is the most common form of the disease and results from prolonged exposure of the mucosa to gastric acid in an otherwise normal gastrointestinal tract. Secondary squamous gastric disease occurs as a result of delayed gastric emptying caused by inflammatory bowel disease, pyloric stenosis, severe glandular gastric disease or gastric impaction.

Equine squamous gastric disease

Equine squamous gastric disease is thought to be the more common form of gastric ulcer syndrome and is certainly the better understood. Ulceration is found in the squamous mucosa of the margo plicatus, greater and lesser curvatures and dorsal squamous fundus. Gastric acid, primarily hydrochloric acid, alongwith volatile fatty acids and bile acids, mix in high concentration in the proximal part of the stomach when gastric content stratification is altered. At pH ≤4 these acids are lipid soluble; they reduce mucosal barrier function and then diffuse into the stratum spinosum, causing ulceration. Lesion severity is time, dose and pH dependent (Argenzio, 1999; Nadeau et al, 2003; Andrews et al, 2006).

Equine squamous gastric disease is graded 0–4 (*Table 1*; *Figure 1*). Grade 1 is often not deemed clinically significant and treatment for this grade may not be covered by some insurance companies on this basis. Anecdotally, there is a small subset of horses with hyperkeratosis or hyperaemia only that appear to respond to treatment. Risk factors for the development of squamous gastric disease have been identified and must be considered when recommending management changes. Carbohydrate-rich rations and large carbohydrate feeds increase the volatile fatty acid content and lower the gastric pH. Feeding >2 g/kg starch per day is associated with a twofold increased risk of squamous gastric disease (Kranenburg et al, 2023). Reduced access to forage means there is a reduction in saliva production, which is a natural gastric acid buffer, and a reduced fibre mat within the stomach that protects against 'splash back' lesions on the squamous mucosa (Galinelli et al, 2021).

High intensity exercise predisposes to squamous gastric disease, as the volume of the stomach is reduced because of negative intra-abdominal pressure, thus increasing the time in which the squamous mucosa is in contact with gastric acid (Nicol et al, 2002; Sykes et al, 2019; Patino et al, 2020). A similar mechanism is proposed during crib-biting. Crib-biting and other stereotypies are associated with increased risk of squamous gastric disease.

Treatment is based on acid suppression with the proton pump inhibitor omeprazole buffered oral paste (4 mg/kg every 24 hours) or enteric-coated granule formulation (1 mg/kg for 21 days), with healing rates of 67–100% reported (Vokes et al, 2023). Histamine receptor antagonists are not as effective, but can be added to the

treatment regimen if omeprazole alone is unsuccessful (oral ranitidine 6.6 mg/kg every 8 hours). Omeprazole oral bioavailability is improved if given on an empty stomach; it is a prodrug and requires activation by feeding-induced gastric acid for it to bind to proton pumps. Therefore, it is recommended that the drug is administered first thing in the morning, ideally after an overnight fast of 8 hours, 30 minutes before breakfast. Long-acting injectable omeprazole has superior healing rates to oral omeprazole – 86% vs 67% in one study (Gough et al, 2020). Esomeprazole also has superior healing rates to oral omeprazole, (85% vs 59%), particularly in a horse with concurrent glandular disease (Sundra et al, 2024). However, only buffered oral omeprazole is licensed in horses in the UK and so other medications are used through the prescribing cascade, and consideration should also be given to patient compliance with injection.

Equine glandular gastric disease

Equine glandular gastric disease is found in the glandular mucosa of the cardia, ventral glandular fundus, antrum and pylorus, most commonly the pyloric antrum. Its pathophysiology and cause are not as well understood as for squamous gastric disease. Lesions are inflammatory rather than ulcerative on histopathology (Crumpton et al, 2015). Lesion severity is not well correlated with appearance and the recommendation is that lesions are described by location, distribution, severity and appearance, rather than graded (*Table 2*; *Figure 2*). Glandular polyps are also considered a form of glandu- lar gastric disease. Some veterinarians recommend biopsy to further classify the glandular gastric disease with a transendoscopic 'double bite', or a snare technique for polyp lesions (Crumpton et al, 2015; Banse and Andrews, 2019).

Stress may play a role in the pathogenesis of glandular gastric disease. This is supported by increased cortisol levels in clinical cases, and the identification of the following risk factors for glandular gastric disease: multiple handlers or trainers (Kranenburg et al, 2023), reduced interaction with other horses, solid fronted stables and even a particular radio station. High intensity or regular exercise has been associated with glandular gastric disease. Blood flow to the stomach is altered during exercise, which is thought to interfere with mucosal barrier function. Therefore, in horses with glandular gastric disease, routines should be maintained, handlers kept to a minimum and horses exercised less than five times a week (Sykes et al, 2015a; Banse and Andrews, 2019; Vokes et al, 2023).

Despite not knowing the true pathophysiology of equine gastric glandular disease, acid suppression and mucosal protectants are beneficial for ulcer healing. Treatment was typically based on a 4–8-week course of omeprazole with the addition of sucralfate 12 mg/kg orally every 12 hours. Improvement has been reported in 80% and healing in 63% of 204 sport horses (Hepburn, 2014) with this regimen, although healing was only 20% (note that there is a conflict in definition of healing between the studies) and an improvement of 65% in another study (Varley et al, 2019). Sucralfate is a polyaluminium hydroxide complex salt which provides a physical barrier against acid, increases mucosal bloodflow, promotes re-epithelialisation, stimulates mucus secretion and inhibits pepsin release (Banse and Andrews, 2019). However, greater success rates and shorter treatment courses have been reported with injectable omeprazole (4 mg/kg intramuscularly every 7 days for 4 treatments; Gough et al, 2022), and more recently with a 5 day dosing interval; 97% healing with 5-day dosing interval vs 82% with 7-day dosing interval (Sundra et al, 2024). Injection site reactions are reported but typically resolve with minimal intervention; however, oedema, heat and pain are described and can impact sport horse performance (Lehman et al, 2021). The recommendation by the manufacturer is to not inject into the pectoral muscles and that less reaction is seen if the gluteals are used.

Misoprostol (5 µg/kg every 12 hours) has been reported to have greater success than the omeprazole/sucralfate combination – 72% healing and 98% improvement (Varley et al, 2019). This is a prostaglandin analogue that suppresses acid production, inhibits neutrophilic inflammation and increases mucus secretion and gastric mucosal blood flow. It is the drug of choice for non-steroidal anti-inflammatory drug-induced right dorsal colitis. However, it should not be administered with omeprazole for concurrent squamous gastric disease as it can compromise proton pump inhibitor efficacy (Rendle et al, 2018). Despite this, clinicians in the USA can use an omeprazole 228 mg/ml and misoprostol 0.14 mg/ml combined oral paste (Nexgen), and many practitioners will use misoprostol in combination with omeprazole. Further research is clearly warranted. In the author's experience of cases treated with misoprostol, glandular gastric disease has improved but squamous gastric disease deteriorated. This may be as a result of the different pathophysiologies of squamous vs glandular gastric disease, and a progression of squamous gastric disease despite glandular gastric disease responding to treatment. Misoprostol administration has an increased risk for diarrhoea and colic (although this is typically self-limiting), and there are human health and safety ramifications for handling misoprostol as it causes uterine contraction and fetal loss (Rendle et al, 2018).

Table 2. Classification of equine glandular gastric disease lesions

Anatomical	Cardia
	Fundus
	Antrum
	Pylorus
Severity	Mild
	Moderate
	Severe
Pronouncement	Raised
	Flat
	Depressed
	Nodular
Appearance	Hyperaemic (red)
	Haemorrhagic (bleeding)
	Fibrinosuppurative (exuding fibrin strands and pus)
Number	Focal
	Multi-focal
	Diffuse

(Sykes et al, 2015a)

Equine gastric ulcer syndrome in foals

Hypoxia, physiological stress, non-steroidal anti-inflammatory drugs and illness, particularly gastrointestinal, are all proposed causes of gastric ulcer synrome in foals (Lewis, 2003; Elfenbein and Sanchez, 2012). Infrequent or interrupted feeding and recumbency increases the exposure of the squamous mucosa to gastric acid and reduces the buffering capacity of milk. Desquamation of the squamous mucosa occurs in up to 80% of foals up to 35 days of age – this makes the area more susceptible to injury, as does any delay in re-epithelialisation. Despite this, equine gastric ulcer syndrome is less common in neonates (<1 month of age), and more commonly found in older foals (Elfenbein and Sanchez, 2012).

Gastroduodenal ulcer disease is a significant cause of morbidity and mortality in late suckling and weanling foals (25–51%; Murray, 1999). Lesions are primarily located in the pylorus and proximal duodenum and may cause outflow obstruction requiring surgical correction (Coleman et al, 2009). Clinical signs include ill thrift, inappetence, ptyalism, bruxism, colic and reflux (Murray, 1999; Elfenbein and Sanchez, 2012). Details of treatment are beyond the scope of this article but the reader is referred to the cited references.

Challenges and frequently asked questions when managing equine gastric ulcer syndrome

Which formulation of omeprazole should be used and at what dose?

There are now multiple forms of omeprazole available to clinicians, including long-acting injectable, oral pastes, granules, enteric coated and buffered formulations. A comparative study showed no or small differences in bioavailability (Busechian et al, 2023) and two further studies showed similar clinical outcomes with varying doses; 1/2/4 mg/kg (Sykes et al, 2015b; 2016). However, these doses were administered to a small sample of horses before exercise after a brief fast. The 'fed-state' and subsequent reduced bioavailability must be considered (Sykes et al, 2017), particularly when using lower than the recommended dose (4 mg/kg).

Omeprazole is acid labile, so buffered pastes and enteric coating that offer protection from gastric acid degradation must be used. It is recommended that the manufacturer dosing guidelines are followed for each product. Emphasis should be placed on time of day of dosing with oral formulations, ideally after a fast, to maximise bioavailability.

Rebound gastric acidity and an increased risk of gastric ulcer syndrome has been described (Helgadóttir et al, 2021). This is as a result of the elevated gastrin levels seen with omeprazole therapy; it is brief and generally only in the 48-hour period after discontinuation of therapy. For this reason, if treatment is for less than 8 weeks, omeprazole dose tapering before discontinuation is not recommended as the rebound gastric acidity is only for a short period. It should be ensured that the horse has adequate roughage during this 48-hour period and is not placed in situations deemed to increase the risk of gastric ulcer syndrome during this time (such as transport or high intensity work). Horses on longer treatment regimens may benefit from tapering of the omeprazole dose before discontinuing (Vokes et al, 2023) but further research is needed to verify this.

What nutritional recommendations should be made?

It is not quite as simple as recommending 'ad-lib' forage or grazing, although they are beneficial. At least 2% bodyweight/day of good quality roughage should be consumed, consideration should be given to the high non-structural carbohydrate content of grass, and water should be constantly accessible (Sykes et al, 2015a; Vokes et al, 2023). Non-structural carbohydrate content should be reduced and concentrate and grains fed as little as possible, with starch not exceeding >2 g/kg bodyweight/day or >1 g/kg bodyweight per meal (Sykes et al, 2015a; Böhm et al, 2018). Highly palatable hay or 1–2 litres of chaff should be fed before exercise to buffer the gastric acid and create a raft on the surface of gastric content (Galinelli et al, 2021; Vokes et al, 2023).

The above recommendations are for squamous gastric disease; dietary management does not have such a direct effect on glandular gastric disease. However, limiting grain intake and maximising access to pasture are thought to be important (Rendle et al, 2018; Banse and Andrews, 2019).

Should nutraceuticals be used routinely?

There is a plethora of nutraceuticals on the market and an increasing body of evidence looking at the efficacy of various ingredients and supporting their use. Nutraceuticals should be an adjunct to pharmaceuticals in treating gastric ulcer syndrome and can then be used long-term to reduce the risk of recurrence (Vokes et al, 2023). Pectin-lecithin complex has been shown to improve ulcer healing in squamous and glandular gastric disease (Venner et al, 1999; Ferrucci et al, 2003). Magnesium as a buffering agent in combination with *Saccharomyces cerevisiae* and pectin-lecithin has been shown to be protective against gastric ulcer syndrome following omeprazole therapy (Sykes et al, 2014). Corn oil has been shown to improve glandular lesions (Martinez at al, 2016), and is an alternative calorie source to allow reduction of carbohydrate in the ration, a known risk for squamous gastric disease. Specific long-chain fatty acids in particular ratios have anti-inflammatory effects and are beneficial for ulcer healing (Pagan et al, 2022). Further natural products such as liquorice and aloe vera have also reduced lesion severity and number in cases of gastric ulcer syndrome, although these were small sample sizes without controls (Stucchi et al, 2017; Bush et al, 2018; Ahmadnejad et al, 2022). Clinicians are encouraged to make evidence-based decisions when selecting nutraceuticals.

How often should these cases be monitored?

Repeat gastroscopy every 4 weeks is recommended to monitor response to treatment and the necessity for ongoing treatment (Sykes et al, 2015a; Rendle et al, 2018). It is recommended that not only gastroscopy findings but also any resolution of presenting clinical signs is used to assess response to therapy. Once gastric ulcer syndrome has resolved, repeat gastroscopy is indicated with recurrence of presenting clinical signs or 1–3 months following discontinuation of treatment to ensure adequate management.

What is the risk associated with gastroscopy?

The risk of colic in the 48-hour period after gastroscopy is low (17/573; 2.9%), falling to 5/561 (0.9%) if cases of gastric impaction were excluded (Spanton et al, 2019b). Colic typically responded to medical management; however, cases of small intestinal volvulus post gastroscopy have been reported, and for this reason it is recommended that the stomach is deflated after gastroscopy (Bonilla et al, 2014). With cases of gastric impaction , care should be taken not to over inflate the stomach and to provide appropriate analgesia.

The risk of the endoscope being chewed and damaged is a concern for many clinicians because of the expense of the equipment and possible trauma to the patient. This typically happens in two scenarios; first, the patient chews on passage of the endoscope and retroflexes it into the oral cavity. Some clinicians will place a dental gag to prevent this, although it can be difficult to get the patient to swallow the endoscope with a gag on. Placing a gag also has health and safety implications for handlers if the horse throws its head around during the procedure. In larger patients the endoscope can be passed through a pre-placed nasogastric tube, although finding tubing large enough to accommodate the endoscope and the patient can be tricky. Adequate sedation, appropriate restraint with the neck in flexion, and deploying the water valve above the arytenoid cartilages to encourage swallowing is usually sufficient for safe passage. Further sedation should be considered if the patient starts to chew. The second scenario is if the endoscope stops progressing forward at the distal tip, and the insertion tube of the endoscope retroflexes into the pharynx. This should be identified if the image becomes static (the distal tip is not moving), and yet the clinician is continuing to pass the endoscope. This can be avoided by careful monitoring of the image as the endoscope is passed.

What if the lesions are worse or no better on repeat gastroscopy?

First, it is important to check if the original presenting clinical signs have improved or not. It is also important to check owner compliance with management alterations, dosing amount, timing of medication in relation to nutrition and exercise, and administration of medication. Actual roughage intake vs roughage being provided should be measured.

Squamous gastric disease

Omeprazole can be switched from an oral form to injectable to improve bioavailability. If an injectable form is being used, dosing intervals could be reduced from 7 to 5 days. Alternatively, esomeprazole (Sundra et al, 2024) or the addition of histamine receptor antagonists could be considered. Aim for peak acid suppression at the time of greatest risk – administer omeprazole followed by a small feed 30 minutes later (1–2 litres chaff or palatable hay) before exercise (Vokes et al, 2023).

Glandular gastric disease

Clinicians could switch from oral omeprazole/sucralfate to injectable omeprazole, or misoprostol +/- sucralfate. In cases refractory to treatment for 3 months, biopsy should be performed. There is a small subset of cases of glandular disease that may respond to glucocorticoids (Rendle et al, 2018). This may be because of the inflammatory nature of the glandular lesions, which are typically lymphoplasmacytic. In humans and dogs, this infiltrate is associated with inflammatory bowel disease, although that has not been demonstrated in horses (Banse and Andrews, 2019). Glucocorticoids have been shown to slow ulcer healing in rats as a result of the inhibition of prostaglandin; therefore, they are not a first choice for non-responders. Instead, it should be ensured underlying stressors have been tackled. The benefit of long-term omeprazole in refractory cases is unclear if an initial response has not been seen.

Can horses with gastric ulcer syndrome return to their previous level of exercise?

Yes, but ideally exercise practices are altered to try to prevent recurrence of gastric ulcers or treatment failure.

Squamous gastric disease

High intensity exercise is a risk for developing squamous gastric disease. Ideally, horses with squamous gastric disease are not exercised intensely (trot or faster) for more than 40 minutes daily. Exercise would be done in the afternoon when the most roughage has been consumed according to the natural circadian rhythm of foraging. If using oral omeprazole, exercise should be undertaken following administration and a small feed (Vokes et al, 2023).

Glandular gastric disease

Regular exercise is a risk for developing glandular gastric disease. It is recommended that horses with glandular gastric disease have two – possibly three – rest days spaced out within a week. Behavioural stress should also be minimised by reducing the number of riders and handlers (Rendle et al, 2018).

Can non-steroidal anti-inflammatory drugs be safely administered for other pathologies in horses with a history of gastric ulcers?

Non-steroidal anti-inflammatory drugs increase the risk of gastric ulceration (Richardson et al, 2018; Flood and Stewart, 2022). The pathophysiology of this is poorly understood, as prostaglandin at the mucosal level has been shown to be unchanged in horses with non-steroidal anti-inflammatory drug-induced disease (Pedersen et al, 2018). It is worth noting that the studies demonstrating the ulcerogenic capabilities of non-steroidal anti-inflammatory drugs used doses higher than those typically used in practice (MacAllister et al, 1993; Martinez et al, 2016). On this basis, some authors believe the increased risk of glandular disease when non-steroidal anti-inflammatory drugs are used at therapeutic doses is overestimated (Vokes et al, 2023). However, therapeutic doses of non-steroidal anti-infammatory drugs have been shown to cause squamous and glandular gastric disease in fasted horses, a scenario which is not uncommon in hospitalised horses. Suxibuzone, a prodrug for phenylbutazone, had less ulcerogenic effects than phenylbutazone when administered orally at equimolecular doses in one study (Monreal et al, 2004), but another found no difference (Andrews et al, 2009). Richardson et al (2018) showed increased risk of glandular gastric disease with phenylbutazone

KEY POINTS

- Equine gastric ulcer syndrome is the most common disease of the equine stomach, seen in up to 100% of some equine populations.
- Equine gastric ulcer syndrome has been defined as two separate syndromes: equine glandular gastric disease and equine squamous gastric disease.
- Gastroscopy remains the best method of identifying and classifying gastric ulcers.
- Prevention is ideal where possible, and the mainstay of treatment for gastric ulcers is omeprazole.
- Challenges faced by clinicians managing horses with gastric ulcers include determining the correct drug dose, use of nutraceuticals and maintaining freedom from gastric ulcers once resolved.

(4.4 mg/kg) over firocoxib (0.1 mg/kg). Considering the evidence, the author believes that non-steroidal anti-inflammatory drugs can be used in cases with previous gastric ulcer syndrome given the widespread benefit of their use. However, suxibuzone is a reasonable choice for oral medication over phenylbutazone where possible, and care should be taken when using non-steroidal anti-inflammatory drugs in fasted horses.

Should omeprazole be used prophylactically?

Omeprazole has historically been prescribed prophylactically in three clinical situations: hospitalised foals, horse with gastric ulcers receiving non-steroidal anti-inflammatory drugs and times of perceived stress in horses with a history of gastric ulcers. Routine use of omeprazole in neonatal foals has been shown to increase the risk of diarrhoea (Furr et al, 2012) and is no longer recommended. However, the high incidence of gastric ulcers in older foals and weanlings (Murray, 1999) supports prophylactic use during hospitalisation.

Omeprazole to prevent non-steroidal medication induced ulceration is common in humans on long-term aspirin or non-steroidal medication (Kinoshita et al, 2018). However, one study has found an increased incidence of serious gastrointestinal complications in horses receiving non-steroidal medications and omeprazole in combination (Ricord et al, 2021), so this cannot be recommended.

Omeprazole is often used at times of increased risk in cases with a history of gastric ulcers. This seems sensible given the known number of environmental stressors, particularly with glandular gastric disease. However, it should be a considered choice, used at times of stress rather than continuously, as prolonged use of omeprazole is not without risk. Rebound hyperacidity is more marked with longer courses of omeprazole because of the elevation in gastrin levels during omeprazole treatment (Helgadóttir et al, 2021). There is a theoretical increased risk of fracture as a result of changes in calcium absorption (Flood and Stewart, 2022; Vokes et al, 2023), and reduced efficacy is a concern with long-term use. One study demonstrated a reduced area under the curve and maximal concentration on day 29 over day 1 (Di Salvo et al, 2017), and a second showed decreased squamous gastric disease prevention in horses treated for 90 days (Kerbyson et al, 2016).

Conclusions

Equine gastric ulcer syndrome is a highly prevalent disease. Gastroscopy is the gold standard technique for diagnosis and monitoring, but improvement in clinical signs and possible histopathology of biopsies should be taken into consideration when managing glandular gastric disease. Omeprazole remains the cornerstone of pharmaceutical therapy, but emphasis should be placed on altered management. Attention should be given to omeprazole dosing practices and formulations and careful consideration given to its prophylactic use. Continued research into equine glandular gastric disease in the future should continue to guide veterinary management of these cases, and clinicians should try to stay in touch with current recommendations as they may change with further understanding. EQ

Conflicts of interest

The author declares that there are no conflicts of interest.

References

Ahmadnejad M, Jalilzadeh-Amin G, Sykes BW. Prophylactic effects of glycyrrhiza glabra root extract on phenylbutazone-induced equine glandular gastric disease (EGGD). J Equine Vet Sci. 2022;118:104088. https://doi.org/10.1016/j.jevs.2022.104088

Andrews FM, Buchanan BR, Smith SH, Elliott SB, Saxton AM. In vitro effects of hydrochloric acid and various concentrations of acetic, propionic, butyric, or valeric acids on bioelectric properties of equine gastric squamous mucosa. Am J Vet Res. 2006;67(11):1873–1882. https://doi.org/10.2460/ajvr.67.11.1873

Andrews FM, Reinemeyer CR, Longhofer SL. Effects of top-dress formulations of suxibuzone and phenylbutazone on development of gastric ulcers in horses. Vet Ther. 2009;10(3):113–120

Argenzio RA. Comparative pathophysiology of nonglandular ulcer disease: a review of experimental studies. Equine Vet J Suppl. 1999;(29):19–23. https://doi.org/10.1111/j.2042-3306.1999.tb05163.x

Banse HE, Andrews FM. Equine glandular gastric disease: prevalence, impact and management strategies. Vet Med (Auckl). 2019;10:69–76. https://doi.org/10.2147/VMRR.S174427

Becht JL, Byars TD. Gastroduodenal ulceration in foals. Equine Vet J. 1986;18(4):307–312. https://doi.org/10.1111/j.2042-3306.1986.tb03638.x

Böhm S, Mitterer T, Iben C. The impact of feeding a high-fibre and high-fat concentrated diet on the recovery of horses suffering from gastric ulcers. Pferdeheilkunde. 2018;34(3):237–246. https://doi.org/10.21836/PEM20180304

Bonilla AG, Hurcombe SD, Sweeney RW, Hewetson M, Mudge MC. Small intestinal segmental volvulus in horses after gastroscopy: four cases (2011–2012). Equine Vet Educ. 2014;26(3):141–145. https://doi.org/10.1111/eve.12100

Busechian S, Conti MB, Sgorbini M et al. A comparison of the efficacy of two omeprazole formulations in the treatment of equine gastric ulcer syndrome in racehorses: a blinded, randomized clinical trial. J Equine Vet Sci. 2023;126:104296. https://doi.org/10.1016/j.jevs.2023.104296

Bush J, van den Boom R, Franklin S. Comparison of aloe vera and omeprazole in the treatment of equine gastric ulcer syndrome. Equine Vet J. 2018;50(1):34–40. https://doi.org/10.1111/evj.12706

Coleman MC, Slovis NM, Hunt RJ. Long-term prognosis of gastrojejunostomy in foals with gastric outflow obstruction: 16 cases (2001–2006). Equine Vet J. 2009;41(7):653–657. https://doi.org/10.2746/042516409x424126

Crumpton SM, Baiker K, Hallowell GD, Habershon-Butcher JL, Bowen IM. Diagnostic value of gastric mucosal biopsies in horses with glandular disease. Equine Vet J. 2015;47(S48):9. https://doi.org/10.1111/evj.12486_18

Di Salvo A, Busechian S, Zappulla F et al. Pharmacokinetics and tolerability of a new formulation of omeprazole in the horse. J Vet Pharmacol Ther. 2017;40(4):348–355. https://doi.org/10.1111/jvp.12371

Elfenbein JR, Sanchez LC. Prevalence of gastric and duodenal ulceration in 691 nonsurviving foals (1995–2006). Equine Vet J Suppl. 2012;(41):76–79. https://doi.org/10.1111/j.2042-3306.2011.00449.x

Ferrucci F, Zucca E, Croci C, Di Fabio V, Ferro E. Treatment of gastric ulceration in 10 standardbred racehorses with a pectin-lecithin complex. Vet Rec. 2003;152(22):679–681. https://doi.org/10.1136/vr.152.22.679

Flood J, Stewart AJ. Non-steroidal anti-inflammatory drugs and associated toxicities in horses. Animals (Basel). 2022;12(21):2939. https://doi.org/10.3390/ani12212939

Furr M, Cohen ND, Axon JE et al. Treatment with histamine-type 2 receptor antagonists and omeprazole increase the risk of diarrhoea in neonatal foals treated in intensive care units. Equine Vet J Suppl. 2012;(41):80–86. https://doi.org/10.1111/j.2042-3306.2011.00499.x

Galinelli N, Wambacq W, Broeckx BJG, Hesta M. High intake of sugars and starch,

low number of meals and low roughage intake are associated with equine gastric ulcer syndrome in a Belgian cohort. J Anim Physiol Anim Nutr (Berl). 2021;105(Suppl 2):18–23. https://doi.org/10.1111/jpn.13215
Gough S, Hallowell G, Rendle D. A study investigating the treatment of equine squamous gastric disease with long-acting injectable or oral omeprazole. Vet Med Sci. 2020;6(2):235–241. https://doi/org10.1002/vms3.220
Gough S, Hallowell G, Rendle D. Evaluation of the treatment of equine glandular gastric disease with either long-acting-injectable or oral omeprazole. Vet Med Sci. 2022;8(2):561–567. https://doi.org/10.1002/vms3.728
Helgadóttir H, Lund SH, Gizurarson S, Waldum H, Björnsson ES. Pharmacokinetics of single and repeated oral doses of esomeprazole and gastrin elevation in healthy males and females. Scand J Gastroenterol. 2021;56(2):128–136. https://doi.org/10.1080/00365521.2020.1859610
Hepburn RJ. Treatment of ulceration of the gastric glandular mucosa: retrospective evaluation of omeprazole and sucralfate combination therapy in 204 sport and leisure horses. Presented at the 11th Internation Equine Colic Research Symposium, Dublin, 7–10 July 2014
Hewetson M, Sykes BW, Hallowell GD, Tulamo RM. Diagnostic accuracy of blood sucrose as a screening test for equine gastric ulcer syndrome (EGUS) in adult horses. Acta Vet Scand. 2017;59(1):15. https://doi.org/10.1186/s13028-017-0284-1
Hewetson M, Venner M, Volquardsen J et al. Diagnostic accuracy of blood sucrose as a screening test for equine gastric ulcer syndrome (EGUS) in weanling foals. Acta Vet Scand. 2018;60(1):24. https://doi.org/10.1186/s13028-018-0377-5
Kerbyson NC, Knottenbelt DK, Carlsake HB et al. A comparison between omeprazole and a dietary supplement for the management of squamous gastric ulceration in horses. J Eq Vet Sci. 2016;40:94–101. https://doi.org/10.1016/j.jevs.2016.02.228
Kinoshita Y, Ishimura N, Ishihara S. Advantages and disadvantages of long-term proton pump inhibitor use. J Neurogastroenterol Motil. 2018;24(2):182–196. https://doi.org/10.5056/jnm18001
Kranenburg LC, van der Poel SH, Warmelink TS, van Doorn DA, van den Boom R. Changes in management lead to improvement and healing of equine squamous gastric disease. Animals (Basel). 2023;13(9):1498. https://doi.org/10.3390/ani13091498
Lamglait B, Vandenbunder-Beltrame M, Trunet E, Lemberger K. Description of gastric ulcers and of their suspected, associated risk factors in deceased wild equids at the Réserve Africaine de Sigean, France (2010–2016). J Zoo Wildl Med. 2017;48(3):668–674. https://doi.org/10.1638/2016-0249.1
Lehman ML, Bass L, Gustafson DL, Rao S, O'Fallon ES. Clinical efficacy, safety and pharmacokinetics of a novel long-acting intramuscular omeprazole in performance horses with gastric ulcers. Equine Vet Educ. 2021;34(11):573–580. https://doi.org/10.1111/eve.13546
Lewis S. Gastric ulceration in an equine neonate. Can Vet J. 2003;44(5):420–421
MacAllister CG, Morgan SJ, Borne AT, Pollet RA. Comparison of adverse effects of phenylbutazone, flunixin meglumine, and ketoprofen in horses. J Am Vet Med Assoc. 1993;202(1):71–77
Martinez JR, Zuluaga AM, Silveira GE. Effects of corn oil on the gastric mucosa of horses with induced ulcer. Rev Colomb Cienc Pecu. 2016;29(2):138–148. https://doi.org/10.17533/udea.rccp.v29n2a07
Monreal L, Sabaté D, Segura D, Mayós I, Homedes J. Lower gastric ulcerogenic effect of suxibuzone compared to phenylbutazone when administered orally to horses. Res Vet Sci. 2004;76(2):145–149. https://doi.org/10.1016/j.rvsc.2003.10.004
Muñoz-Prieto A, Cerón JJ, Rubio CP et al. Evaluation of a comprehensive profile of salivary analytes for the diagnosis of the equine gastric ulcer syndrome. Animals (Basel). 2022;12(23):3261. https://doi.org/10.3390/ani12233261
Muñoz-Prieto A, Contreras-Aguilar MD, Cerón JJ et al. Changes in calprotectin (S100A8-A9) and aldolase in the saliva of horses with equine gastric ulcer syndrome. Animals (Basel). 2023;13(8):1367. https://doi.org/10.3390/ani13081367
Murray MJ. Gastroduodenal ulceration in foals. Equine Vet Educ. 1999;11(4):199–207. https://doi.org/10.1111/j.2042-3292.1999.tb00948.x
Murray MJ. How to perform gastroduodenoscopy. AAEP Proceedings. 2002;48:282–286
Nadeau JA, Andrews FM, Patton CS, Argenzio RA, Mathew AG, Saxton AM. Effects of hydrochloric, valeric, and other volatile fatty acids on pathogenesis of ulcers in the nonglandular portion of the stomach of horses. Am J Vet Res. 2003;64(4):413–417. https://doi.org/10.2460/ajvr.2003.64.413
Nicol CJ, Davidson HP, Harris PA, Waters AJ, Wilson AD. Study of crib-biting and gastric inflammation and ulceration in young horses. Vet Rec. 2002;151(22):658–662. https://doi.org/10.1136/vr.151.22.658
Pagan JD, Hauss AA, Pagan EC, Simons JL, Waldridge BM. Long-chain polyunsaturated fatty acid supplementation increases levels in red blood cells and reduces the prevalence and severity of squamous gastric ulcers in exercised Thoroughbreds. J Am Vet Med Assoc. 2022;260(S3):S121–S128. https://doi.org/10.2460/javma.22.06.0275
Patino JJ, Velez SA, Martinez JR. Ethological, endocrinological and gastroscopic evaluation of crib-biting Colombian Creole horses. J Vet Behav. 2020;40(5):92–97. https://doi.org/10.1016/j.jveb.2020.10.005
Pedersen SK, Cribb AE, Read EK, French D, Banse HE. Phenylbutazone induces equine glandular gastric disease without decreasing prostaglandin E2concentrations. J Vet Pharmacol Ther. 2018;41(2):239–245. https://doi.org/10.1111/jvp.12464
Rebhun WC, Dill SG, Power HT. Gastric ulcers in foals. J Am Vet Med Assoc. 1982;180(4):404–407
Rendle D, Bowen I, Brazil T et al. Recommendations for the management of equine glandular gastric disease. UK-Vet Equine. 2018;2(Supp 1):2–11. https://doi.org/10.12968/ukve.2018.2.S1.3
Richardson LM, Whitfield-Cargile CM, Cohen ND, Chamoun-Emanuelli AM, Dockery HJ. Effect of selective versus nonselective cyclooxygenase inhibitors on gastric ulceration scores and intestinal inflammation in horses. Vet Surg. 2018;47(6):784–791. https://doi.org/10.1111/vsu.12941
Ricord M, Andrews FM, Yñiguez FJM et al. Impact of concurrent treatment with omeprazole on phenylbutazone-induced equine gastric ulcer syndrome (EGUS). Equine Vet J. 2021;53(2):356–363. https://doi.org/10.1111/evj.13323
Shawaf T, El-Deeb WM, Elgioushy M. The contribution of specific and nonspecific biomarkers in diagnosis of equine gastric ulcer syndrome (EGUS) under field condition. J Equine Vet Sci. 2020;84:102853. https://doi.org/10.1016/j.jevs.2019.102853
Spanton J, Smith L, Mair TS. Is serum amyloid A elevated in horses with equine gastric ulcer syndrome? Equine Vet Educ. 2019a;32(11):16–19. https://doi.org/10.1111/eve.13145
Spanton J, Smith L, Mair T. A clinical audit of the prevalence of colic in the 48 hours after gastroscopy in 436 horses. Equine Vet Educ. 2019b;32(11):12–15. https://doi.org/10.1111/eve.13144
Stucchi L, Zucca E, Serra A et al. Efficacy of the administration of a natural feed supplement in the management of equine gastric ulcer syndrome in 7 sport horses: a field trial. Am J Anim Vet Sci. 2017;12(3):104–110. https://doi.org/10.3844/ajavsp.2017.104.110
Sundra T, Kelty E, Rendle D. Five- versus seven-day dosing intervals of extended-release injectable omeprazole in the treatment of equine squamous and glandular gastric disease. Equine Vet J. 2024;56(1):51–58. https://doi.org/10.1111/evj.13938
Sykes BW, Sykes KM, Hallowell GD. Efficacy of a combination of apolectol, live yeast (saccharomyces cervisiae) and magnesium hydroxide in the management of equine gastric ulcer syndrome in Thoroughbred racehorses: a blinded, randomized, placebo-controlled clinical trial. J Equine Vet Sci. 2014;34(11–12):1274–1278. http://doi.org/10.1016/j.jevs.2014.09.006
Sykes BW, Hewetson M, Hepburn RJ, Luthersson N, Tamzali Y. European College of Equine Internal Medicine consensus statement – equine gastric ulcer syndrome in adult horses. J Vet Intern Med. 2015a;29(5):1288–1299. https://doi.org/10.1111/jvim.13578
Sykes BW, Sykes KM, Hallowell GD. A comparison of three doses of omeprazole in the treatment of equine gastric ulcer syndrome: a blinded, randomised, dose-response clinical trial. Equine Vet J. 2015b;47(3):285–290. https://doi.org/10.1111/evj.12287
Sykes BW, Underwood C, Greer R, McGowan CM, Mills PC. Pharmacokinetics and bioequivalence testing of five commercial formulations of omeprazole in the horse. J Vet Pharmacol Ther. 2016;39(1):78–83. https://doi.org/10.1111/jvp.12240
Sykes BW, Underwood C, Greer R, McGowan CM, Mills PC. The effects of dose and diet on the pharmacodynamics of omeprazole in the horse. Equine Vet J. 2017;49(4):525–531. https://doi.org/10.1111/evj.12630
Sykes BW, Bowen M, Habershon-Butcher JL, Green M, Hallowell GD. Management factors and clinical implications of glandular and squamous gastric disease in horses. J Vet Intern Med. 2019;33(1):233–240. https://doi.org/10.1111/jvim.15350
Tamzali Y, Marguet C, Priymenko N, Lyazrhi F. Prevalence of gastric ulcer syndrome in high-level endurance horses. Equine Vet J. 2011;43(2):141–144. https://doi.org/10.1111/j.2042-3306.2010.00129.x
Traub-Dagartz J, Bayly W, Riggs M, Thomas N, Pankowski R. Exsanguination due to gastric ulceration in a foal. J Am Vet Med Assoc. 1985;186(3):280–281
Ward S, Sykes B, Brown H, Bishop A, Penaluna L. A comparison of the prevalence of gastric ulceration in feral and domesticated horses in the UK. Equine Vet Educ. 2015;27(12):655–657. https://doi.org/10.1111/eve.12491
Wise JC, Wilkes EJA, Raidal SL et al. Interobserver and intraobserver reliability for 2 grading systems for gastric ulcer syndrome in horses. J Vet Intern Med. 2021;35(1):571–579. https://doi.org/10.1111/jvim.15987
Varley G, Bowen IM, Habershon-Butcher JL, Nicholls V, Hallowell GD. Misoprostol is superior to combined omeprazole-sucralfate for the treatment of equine gastric glandular disease. Equine Vet J. 2019;51(5):575–580. https://doi.org/10.1111/evj.13087
Venner M, Lauffs S, Deegen E. Treatment of gastric lesions in horses with pectin-lecithin complex. Equine Vet J Suppl. 1999;(29):91–96. https://doi.org/10.1111/j.2042-3306.1999.tb05178.x
Vokes J, Lovett A, Sykes B. Equine gastric ulcer syndrome: an update on current knowledge. Animals (Basel). 2023;13(7):1261. https://doi.org/10.3390/ani13071261