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Managing glandular gastric disease in horses

Background: Equine glandular gastric disease (EGGD) is an erosive disease of the glandular mucosa of the stomach, which is increasingly recognised as a potential cause of poor performance, girthing pain and behavioural changes in sport horses. Unfortunately, the cause of EGGD has not yet been identified, and thus targeted treatment is not possible. Therefore, current recommendations for treatment and prevention are based on acid suppression and management strategies aimed at mitigating known risk factors.

Aim of the article: This article summarises what we currently know about EGGD and outlines how we can use this information to manage the disease more effectively.

Video
The video mentioned in this article can be viewed with the online version at: bvajournals.onlinelibrary.wiley.com/toc/20427689/2021/43/4

EQUINE glandular gastric disease (EGGD) is an erosive disease of the glandular mucosa, which is increasingly recognised in practice and can be notoriously difficult to treat. Failure to manage this disease effectively is often attributed to the fact that vets do not differentiate between diseases of the squamous and glandular mucosa when considering treatment. Therefore, the terms equine squamous gastric disease (ESGD) and EGGD have been recommended to clearly distinguish the anatomical region of the stomach affected. It is crucial to recognise that these are separate disease entities and that the treatment and management strategies for ESGD cannot be extrapolated to EGGD.

KEY LEARNING OUTCOMES

After reading this article, you should:

- Recognise that equine glandular gastric disease (EGGD) is a separate disease entity from equine squamous gastric disease (ESGD) and that the treatment and management strategies for ESGD cannot be extrapolated to EGGD;
- Know what risk factors have been identified for EGGD and how to implement management strategies to mitigate these risks;
- Recognise clinical signs that may be attributable to EGGD, but understand that they are often non-specific and should therefore be interpreted with caution;
- Be aware of the wide variation in the appearance of lesions on gastroscopy. Be able to describe them and assign them a grade based on their severity;
- Understand that until more is known about the pathogenesis of this disease, first-line treatment is still based on acid suppression. Be aware that response to treatment is unpredictable and that lesions may take several months to heal.

Prevalence

The prevalence of EGGD in adult horses ranges between 47 per cent and 65 per cent in Thoroughbred racehorses; 16 and 33 per cent in endurance horses; 54 and 72 per cent in sport horses; 57 per cent in non-performance horses and 30 per cent in a population of feral horses (ref?). EGGD is often seen concurrently with ESGD in the same horse. EGGD is less common in foals, with only 6 per cent of foals reported to have EGGD in a large postmortem study (ref?).

Pathology

Macroscopic pathology

The equine stomach is comprised of four distinct anatomical regions: dorsal squamous fundus, ventral glandular fundus, cardia and pylorus (Fig 1). Glandular lesions occur most commonly in the pylorus; however, they may occasionally be seen in the ventral fundus and, rarely, the cardia. Small (1 to 2 cm) focal or multifocal, flat or raised lesions are most common, and often lie along the crest of rugal folds (Fig 2). In some cases, these folds may appear hypertrophic (Fig 3). Polypoid or nodular masses are also occasionally seen (Fig 4). Depressed ulcers are rare, but when evident, they represent a severe form of the disease (Fig 5). The surface appearance of these lesions is highly variable and can be described as erythematous, haemorrhagic, and/or fibrinosuppurative (Figs 6–9).

Histology

Hyperaemia and focal erosions are recognised most commonly. Ulceration extending the full thickness of the lamina propria is rare. Therefore, the condition is best described as an erosive gastritis (Martineau and others 2009). Regions of hyperaemia may be histologically normal or may contain areas

of vascular congestion and mild mononuclear inflammatory infiltrate. Erosions vary in depth and are accompanied by a fibrinopurulent exudate and variable amounts of haemorrhage (Fig 10). This is often found in conjunction with a mononuclear cell infiltrate of the lamina propria. Submucosal and muscular fibrosis may be evident in horses with deep ulceration (Bezdekova and others 2020). The raised hypertrophic rugal folds seen in some cases are characterised by hyperplasia of the gastric foveolae, and bear similarities with the hypertrophic folds seen in people with hypertrophic lymphocytic gastritis (Husted and others 2010).

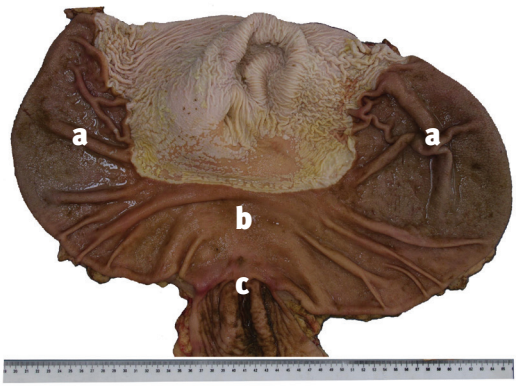

Fig 1: Equine stomach opened along the greater curvature, showing (a) ventral glandular fundus (b) cardia and (c) pylorus. The mucosal lining is divided into a proximal non-glandular and a distal glandular portion by the margo plicatus. The glandular mucosa is red in colour and varies in hue from dark red in the fundus to pale pink in the pylorus. Within these regions a uniform colour is considered normal

Risk factors and aetiopathogenesis

There is no clear relationship between the presence of ESGD and EGGD, and the presence of both conditions concurrently does not indicate that they are associated. The pathogenesis of ESGD is well understood, with a variety of factors associated with intensive management and exercise contributing to a disruption of the normal stratification of gastric pH. This results in exposure of the vulnerable squamous mucosa to acid and leads to ulceration. In contrast, the pathogenesis of EGGD is poorly understood and the mechanisms that lead to the development of this disease have yet to be identified. Similarities exist between EGGD and idiopathic peptic ulcer disease (PUD) in people – in both diseases, the aetiology is unknown and treatment is based on acid suppression.

The glandular mucosa is fundamentally different from the squamous mucosa in that it is normally exposed to a highly acidic environment. As such, EGGD is likely to result from a breakdown of the normal defence mechanisms that protect the mucosa from acidic gastric contents. This is supported by the fact that glandular lesions respond less favourably to acid suppression compared with

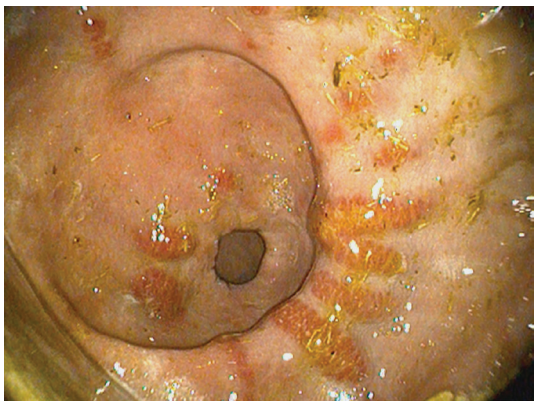

Fig 2: Multifocal, raised, erythematous/fibrinosuppurative lesions. Several lesions can be seen on the crest of rugal folds

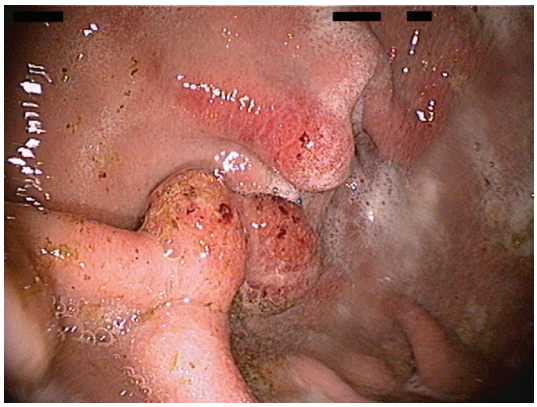

Fig 3: Multifocal to diffuse, raised, erythematous/haemorrhagic lesions with associated hypertrophy of the rugal folds

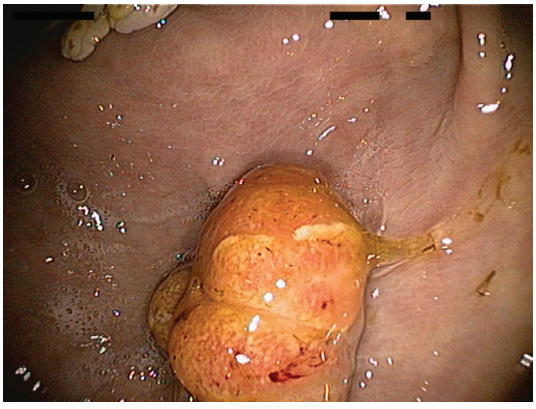

Fig 4: Focal, polypoid (nodular), haemorrhagic/fibrinosuppurative lesion

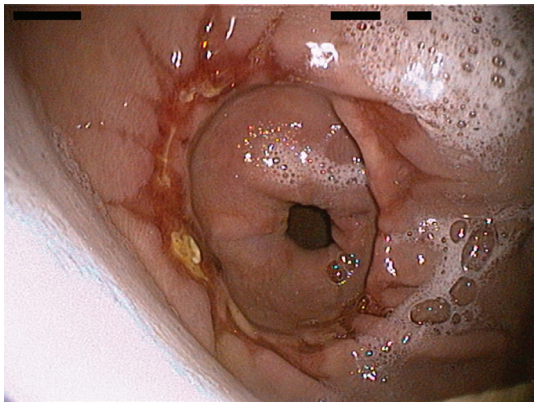

Fig 5: Locally extensive, depressed (ulcerative), haemorrhagic/fibrinosuppurative lesion

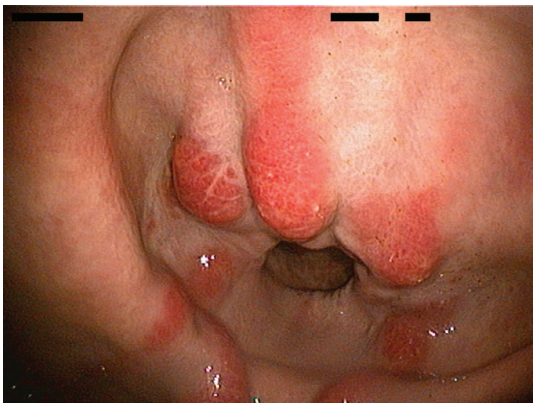

Fig 6: Multifocal, raised, erythematous lesions

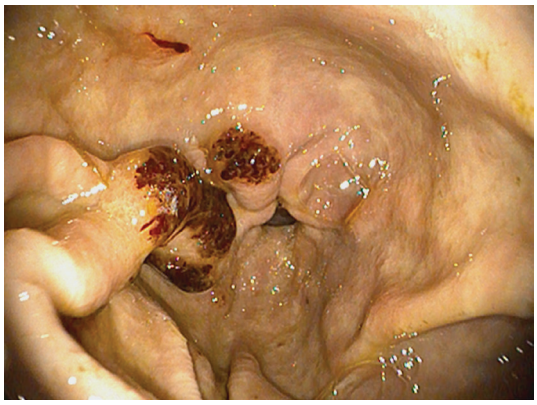

Fig 7: Multifocal, raised, haemorrhagic lesions

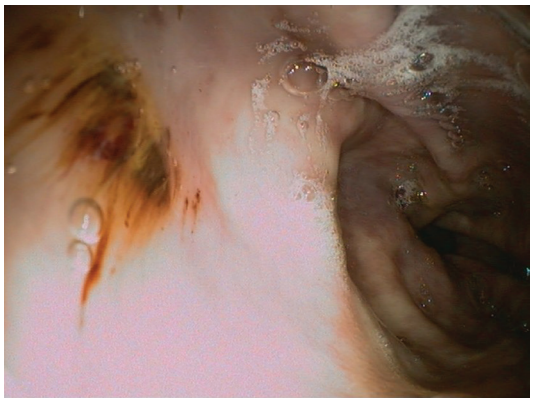

Fig 8: Focal, flat, haemorrhagic/fibrinosuppurative lesion

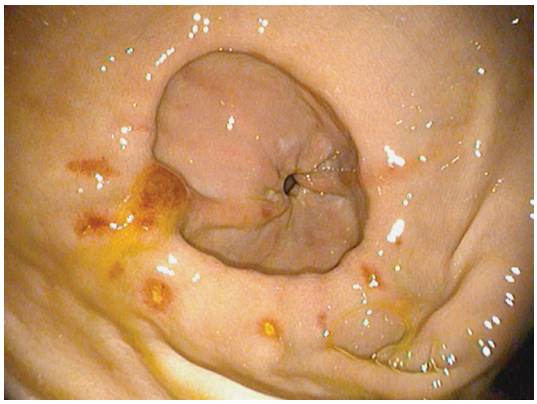

Fig 9: Multifocal, flat, erythematous/fibrinosuppurative lesions

squamous lesions. Therefore, while acid injury is unlikely be the primary cause of EGGD, a low pH may perpetuate mucosal damage and inhibit mucosal healing.

Stress

There is a growing body of evidence to suggest an association between stress and EGGD. Horses with EGGD have greater increases in endogenous cortisol in response to novel stimuli and in response to exogenous administration of adrenocorticotropic hormone (ACTH), suggesting increased susceptibility to stress (Scheidegger and others 2017). Other studies have provided additional indirect evidence of an association between EGGD and stress. Reported risk factors that may cause stress include the trainer, the number of handlers and riders, and lack of experience in competition horses.

Stress is a recognised risk factor for peptic ulcer disease in people and has been shown to decrease gastric mucosal prostaglandin E2 production. This is thought to result in increased acid secretion, altered mucosal blood flow, decreased mucus production, and cytokine-mediated impairment of mucosal defences. This compromises the gastric barrier, leading to increased susceptibility to acid injury and a reduced regenerative capacity. Therefore,

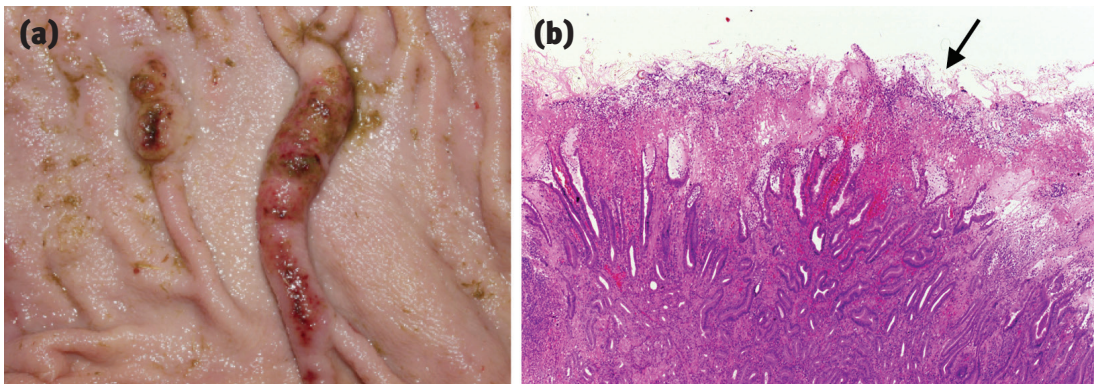

Fig 10: (a) Postmortem specimen of the glandular mucosa in the region of the pylorus obtained from the stomach of a horse with equine glandular gastric disease. Characteristic lesions are visible along the crest of rugal folds. (b)Histological examination confirmed an erosive gastritis with superficial haemorrhage and fibrinopurulent exudate (arrow?). Picture: H. Martineau, xxx

stress may be a factor in the perpetuation, if not the initiation, of EGGD.

Exercise

There is evidence to suggest that exercise may be a risk factor for EGGD. Horses that exercise for more than four to five days a week are more likely to develop EGGD, and it is not the intensity of exercise, but rather the total amount of exercise that increases the risk of EGGD (Pederson and others 2018). Exercise has been associated with an increase in gastric permeability in people and dogs. The increase in gastric permeability during exercise is likely due to splanchnic hypoperfusion and subsequent intestinal ischemia that damages gastric epithelial cells and ultimately compromises the gastric barrier. This results in increased exposure to luminal acid and ultimately leads to gastric injury.

NSAIDs

NSAIDs are a well-recognised risk factor for gastric ulceration in many species; however, there is currently no evidence to suggest that administration of these drugs at recommended therapeutic doses increases the risk of EGGD or ESGD in horses. Inappropriately high doses have been reported to cause erosions and ulceration of the glandular mucosa, but lesions were not seen when NSAIDs were administered at recommended therapeutic doses for up to 15 days (Andrews and others 2009).

Bacteria

The role of bacteria in the pathogenesis of EGGD is unclear. *Helicobacter pylori*, a bacterium usually found in the stomach, has not been consistently identified in horses with EGGD and is unlikely to be a primary causative agent. Similarly, other gastrically adapted bacteria are unlikely to play a primary role in the initiation of EGGD. However, opportunistic secondary bacterial infections (eg, *Escherichia fergusonii*) may be a factor in the perpetuation of EGGD in isolated cases, and detection of large numbers of any bacteria in biopsies obtained from mucosal lesions should be cause for concern, as this is not a normal finding in the equine glandular stomach (Husted and others 2010).

Inflammatory bowel disease and gluten sensitivity

Crohn's disease is a subcategory of inflammatory bowel disease (IBD) in people, which causes inflammation and ulceration of the intestine. The terminal ileum and colon are most affected, but the disease can affect any part of the intestine, including the stomach. Increased gastric permeability has been reported in patients with Crohn's disease, suggesting involvement of the stomach in a high proportion of patients with this disease. The mechanism for this increased permeability is related to inflammatory changes and their associated effects on gastric barrier. There is currently no evidence to suggest that IBD in horses affects the stomach; however, there are similarities in the histological appearance of lesions seen in horses with IBD and those seen in the glandular mucosa of horses with EGGD. Therefore, it is possible that EGGD may be a manifestation of IBD, and further investigation to confirm this association and to understand the underlying pathophysiological mechanism is warranted.

Coeliac disease is a genetic disease associated with a gluten intolerance, which results in severe villous atrophy of the small intestine that responds to gluten exclusion. People with coeliac disease have also been reported to have increased gastric permeability, and it has been suggested that this may be due to the presence of a concurrent lymphocytic gastritis. Gluten sensitive enteropathy has been described in horses, and an association between gluten sensitivity and EGGD has been suggested (van Proosdij and others 2020).

Clinical signs

Clinical signs that may be attributed to EGGD include:

- Changes in temperament (including nervousness and aggression);
- Acute or recurrent colic (often manifested as mild postprandial abdominal discomfort);
- Reduced appetite, altered eating patterns and unexplained weight loss;
- Poor performance and changes in rideability (including reduced willingness to work and reluctance to go forward);
- Cutaneous sensitivity (manifested as biting of the flanks or resentment of girthing, grooming, leg aids or rugging).

Perhaps the most intriguing clinical sign that is recognised almost exclusively in horses with EGGD is cutaneous sensitivity. The most likely explanation is that of a viscerosomatic reflex (referred pain), in which localised visceral stimuli in the stomach produce patterns of reflex activity and hyperaesthesia in segmentally related somatic structures, such as the skin. This abnormal behaviour appears to resolve following treatment.

Delayed gastric emptying has been reported in horses with chronic severe pyloric ulceration and is associated with a poor prognosis in young horses (Bezdekova and others 2020).

Diagnosis

Gastroscopy is currently the only reliable method for definitive diagnosis of EGGD in horses. Endoscopically guided biopsies are rarely indicated, but if more severe pathological changes are suspected, or if cases are refractory to treatment, then biopsies should be considered.

Table 1: Descriptive terminology for equine glandular gastric disease

Type	Descriptive terminology
Anatomical region affected	Cardia, fundus, pylorus (antrum or sphincter)
Distribution	Focal, multi-focal, or diffuse (locally extensive)
Topography	Flat, raised, nodular or depressed
Appearance	Erythematous, haemorrhagic or fibrinosuppurative

Table 2: Equine Gastric Ulcer Council's five-point ordinal grading system for equine glandular gastric disease

Grade	Appearance of the glandular mucosa
0	The epithelium is intact and there is no appearance of erythema
1	The epithelium is intact, but there are areas of erythema
2	Small, single, or multifocal lesions
3	Large single or extensive superficial lesions
4	Extensive lesions with area of apparent deep ulceration

Gastroscopy technique

Food should be withheld from the horse for 16 hours and water for two hours before gastroscopy. Following completion of fasting, the horse is sedated and a 3 m video endoscope is passed into the stomach via the ventral nasal meatus and the oesophagus. The stomach is then distended by insufflation with air through the biopsy channel of the endoscope until the squamous and glandular mucosae are visible. To ensure proper identification of all mucosal defects, gastric contents should be rinsed from the mucosa with tap water flushed through the biopsy channel. At the conclusion of the examination, the stomach should be deflated by suctioning air through the biopsy channel. When performing gastroscopy, it is important to visualise the entire stomach, including the pylorus and proximal duodenum, as lesions in these regions are easily overlooked. The procedure is demonstrated in Video 1.

Grading

Until recently, the use of a hierarchical grading system similar to that used for ESGD was considered inappropriate for EGGD – there is a wide variation in the gross appearance of these lesions, and therefore the relative severity and clinical importance of a particular lesion type is unknown. Instead, the use of descriptive terminology with a clear distinction of the anatomical region affected, distribution, topography and the surface appearance of the lesions has been recommended (Rendle and others 2018) (Table 1). The problem with this approach is that it is difficult to describe the lesions to owners without the use of a simple grading system that provides an estimate of severity. A reliable grading system is also important for comparison of lesions when assessing response to treatment, particularly if more than one person in the practice is performing the gastroscopy. With these limitations in mind, the interobserver reliability of this descriptive system was recently assessed and was found to be poor (Tallon and Hewetson 2021).

An alternative is to use an ordinal grading system based on lesion depth, size, and number, similar to that which was proposed by the Equine Gastric Ulcer Council for ESGD (Wise and others 2021a) (Table 2). This has advantages as it has been used for many years and is familiar to owners and equine practitioners. Clinical experience would suggest that glandular lesions tend to improve in depth, size and number with treatment, irrespective of lesion type. Therefore, the grading system can easily be applied to the glandular mucosa (Fig 11), and in a recent

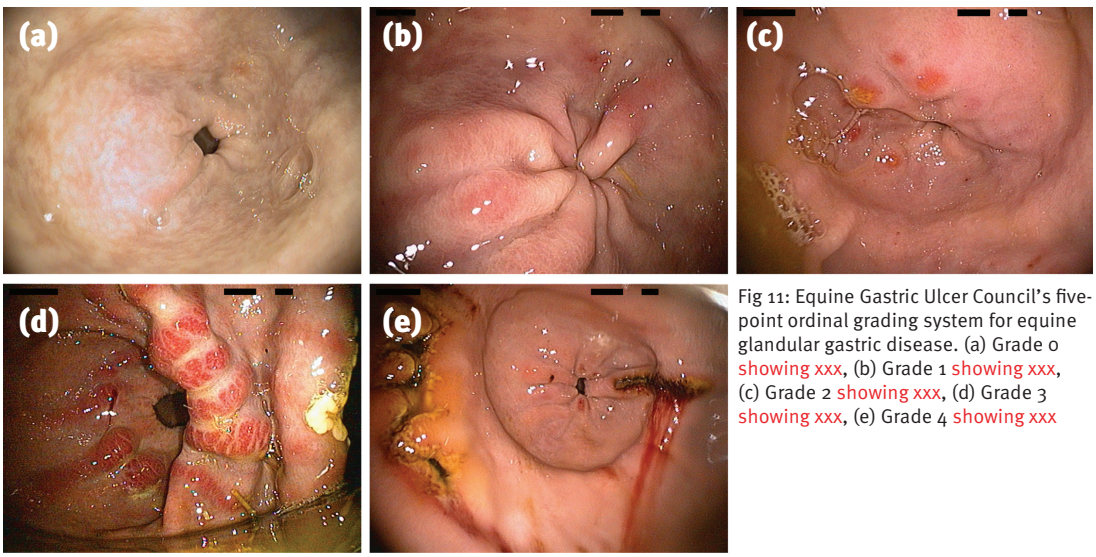

Fig 11: Equine Gastric Ulcer Council's five-point ordinal grading system for equine glandular gastric disease. (a) Grade 0 showing xxx, (b) Grade 1 showing xxx, (c) Grade 2 showing xxx, (d) Grade 3 showing xxx, (e) Grade 4 showing xxx

study, the intraobserver and interobserver reliability for EGGD was found to be acceptable, regardless of experience (Wise and others 2021a). The grading system should not be used as an alternative to a detailed description of the lesions, but rather as an additional tool to estimate lesion severity when assessing response to treatment and communicating findings to the owner.

Management

When do you treat?

With an increase in public awareness of EGGD and its popularity as a 'catch-all' diagnosis for poor performance in sport horses, an increasing number of vets are electing to treat horses on an empirical basis, without the benefit of a definitive diagnosis. There is currently very little evidence to suggest a direct cause-and-effect relationship between clinical signs and the presence or severity of glandular lesions in horses; although it has been reported that treatment of EGGD is associated with improved performance (Varley and others 2019), this relationship is unlikely to be linear or temporally consistent. Therefore, clinical signs should be interpreted with caution, and treatment should always be based on endoscopic evidence of EGGD.

What do you treat?

A wide variety of lesion types are seen in horses with EGGD and there is currently no evidence to suggest a correlation between lesion type and histological severity. Therefore, the relative clinical significance of a particular lesion type is unknown, and all lesion types should be treated. However, mucosal erythema (ie, grade 1 lesions) is an exception, as the underlying mucosa may be histologically normal in some cases (Martineau and others 2009). Interpretation of these lesions is further complicated by the fact that assessment of mucosal colour is subjective and may be affected by light and colour settings on the endoscope. Mucosal blanching may also be misinterpreted as regions of erythema. This appears as an intermittent reticulated pattern (Fig 12) in the region of the pylorus, and is a normal finding associated with pyloric peristalsis. Therefore, the decision to treat mucosal erythema should be based on clinical judgement.

How do you treat?

Although acid is unlikely to be directly implicated in the development of lesions, a low pH exacerbates mucosal injury and inhibits healing, hence the adage 'no acid no ulcer'. Therefore, the primary goal of treatment in EGGD is acid suppression. Secondary goals include encouraging mucosal healing, restoring normal gastric barrier function and treating underlying inflammation when relevant. The efficacy of acid suppressants for the treatment of EGGD can be assessed by demonstrating an increase in pH in the ventral stomach and an improvement in lesion score and/or appearance. In people with PUD, it is recommended that the intra-day percentage of time that gastric pH is maintained above 4 should be greater than 66 per cent to facilitate healing – this is likely to be similar in horses.

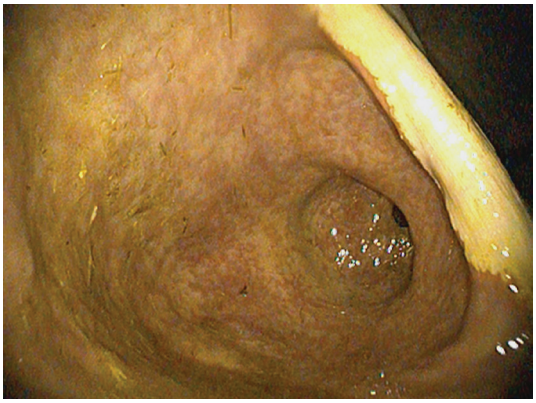

Fig 12: Blanching of the glandular mucosal in the region of the pylorus. This should not be confused with erythematous lesions

The two drugs of choice for acid suppression in horses are omeprazole and misoprostol. Neither of these drugs hold a veterinary licence specifically for the treatment of EGGD in the UK. Oral omeprazole is licenced for the treatment of ESGD; however, there is no market claim of its efficacy in the management of EGGD. Therefore, selection of a first-line drug for EGGD should be done under the prescribing cascade. Table 3 outlines the approximate doses of drugs used to treat equine glandular gastric disease.

Omeprazole

Omeprazole is a substituted benzimidazole that suppresses gastric acid secretion by specific inhibition of the parietal cell H+/K+ ATPase pump. Binding is irreversible and so the drug has a long duration of action.

Several formulations of omeprazole are available, including intravenous, intramuscular and oral preparations. Intravenous omeprazole has been shown to effectively inhibit gastric acid secretion and increase pH for up to 24 hours (ref?), but it is not practical for long-term management of EGGD. Oral preparations are used most commonly due to their ease of administration. Omeprazole is acid labile and so protection from the acidic environment of the stomach is necessary. The two most common formulations are buffered and enteric-coated omeprazole. Enteric-coated omeprazole has been shown to have increased bioavailability when compared with buffered omeprazole, and a dose as low as 1 mg/kg is effective at inhibiting acid secretion (Wise and others 2021b). Omeprazole should be administered on an empty stomach, as food may impact the bioavailability and therefore the efficacy of the drug. This can be achieved practically by administering it first thing in the morning at least one hour before feeding. This

Table 3: Approximate drug doses used to treat equine glandular gastric disease

Drug	Dose	Route	Frequency	Comments
Intravenous omeprazole	0.5 mg/kg	IV	Q 24 hours	Not practical
Buffered omeprazole	4 mg/kg	PO	Q 24 hours	Administer on empty stomach
Enteric coated omeprazole	1–4 mg/kg	PO	Q 24hours	Administer on empty stomach
Long-acting injectable omeprazole	4 mg/kg	IM	Q seven days	Risk of injection-site reactions
Misoprostol	5 µg/kg	PO	Q 12 hours	Concerns over client safety
Sucralfate	12 mg/kg	PO	Q 12 hours	Questionable efficacy

IM Intramuscularly, IV Intravenous, PO Orally, Q Every

ensures the stomach will be empty; horses eat very little during the night even if they have access to forage.

Both formulations have been shown to increase intragastric pH; however, their efficacy for treating EGGD is poor, with published healing rates as low as 9 to 32 per cent (ref?). The reason for this is unclear, but may be related to regional differences in acid suppression in horses that naturally consume high roughage diets. It has been shown that the percentage of intra-day time that pH was >4 in the ventral stomach of these horses was as low 30 to 40 per cent on day five following daily administration of a buffered formulation of omeprazole at a dose of 4 mg/kg (Sykes and others 2017a); this is clearly well below the threshold for healing reported in people.

An alternative approach is to use intramuscular omeprazole. A long-acting preparation has recently been reported to be more effective than oral omeprazole in increasing the pH in the ventral stomach (Sykes and others 2017b), and has the added advantage of achieving effective acid suppression without the necessary dietary modifications that are required when using oral formulations. A dose of 4 mg/kg has been shown to inhibit acid secretion for up to seven days and so can be conveniently administered at weekly intervals. Large-scale efficacy of this formulation for the treatment of EGGD has yet to demonstrated; however, Sykes and others (2017b) reported 75 per cent healing in 12 horses after two weeks, and there are anecdotal reports of healing rates as high as 86 per cent after four weeks of treatment (Rendle, unpublished data). The formulation is oil based and should be administered by deep intramuscular injection into the neck or gluteals. Transient non-painful swellings associated with drug administration have been reported.

Misoprostol

Misoprostol is a synthetic prostaglandin E1 analogue that is used to treat chronic erosive gastritis in people and has recently been advocated for the treatment of EGGD. Misoprostol inhibits acid secretion via direct stimulation of prostaglandin E1 receptors on parietal cells and exerts a cytoprotective effect on the gastric mucosa, which may be of additional benefit in the treatment of EGGD. Stimulation of mucus and bicarbonate secretion from non-parietal cells helps maintain the adherent mucus gel layer and the mucus-mucosal pH gradient, thus ensuring a neutral pH at the mucosal surface. Furthermore, regulation of mucosal blood flow ensures adequate oxygen delivery and rapid elimination of hydrogen ions that may have diffused through to the lamina propria. Misoprostol also has anti-inflammatory effects and has been shown to inhibit tumor necrosis factor-α (TNFα) production in equine leucocytes.

In a recent comparative study that investigated the efficacy of misoprostol for treatment of EGGD, glandular lesions healed in 72 per cent of horses, and the drug was shown to be superior to a combination of omeprazole and sucralfate (Varley and others 2019).

The drug is rapidly absorbed following oral administration, and a single dose of 5 µg/kg has been shown to inhibit acid secretion for up to eight hours and maintain gastric pH above 3.5 for up to five hours. The effect of food on misoprostol absorption remains to be elucidated. Transient abdominal discomfort may occur for up to 30 mins following drug administration and horses may develop bouts of mild, self-limiting diarrhoea which resolves with cessation of drug therapy. Misoprostol can cause abortion in women, and so it is important to inform owners of the risk and provide appropriate guidance regarding handling and storage of the drug.

Sucralfate

Sucralfate is a basic aluminium salt of sulphated sucrose that reacts with hydrochloric acid in the stomach to form a viscous paste that selectively binds to ulcerated tissue. As such, it provides a protective barrier at the ulcer surface, preventing further injury from acid, pepsin and bile. Sucralfate is also thought to stimulate production of prostaglandin E2 and epidermal growth factor,

thus enhancing blood flow and promoting reepithelialisation.

There are no reports on the efficacy of sucralfate monotherapy for the treatment of EGGD, but it has been suggested that the efficacy of oral omeprazole for the treatment of EGGD may be improved by combining it with sucralfate. The efficacy of this combination was investigated in a recent prospective study, but unfortunately the outcome was poor, with reported healing rates of 20 per cent (Varley and others 2020). Therefore, combination therapy is of questionable additional benefit, especially considering the improvements seen with intramuscular omeprazole monotherapy.

Glucocorticoids

Some horses with EGGD will not respond satisfactorily to acid suppression alone, and there is anecdotal evidence to suggest that, in a small subset of cases, the judicious use of glucocorticoids may be beneficial. The rationale for the use of glucocorticoids is based on similarities in the histological appearance of lesions seen in horses with IBD and those seen in horses with EGGD. Glucocorticoids should be used in conjunction with acid suppression and should not be a first-line treatment for EGGD.

Antimicrobials

There is no evidence to suggest that antimicrobials are indicated as a first-line treatment for EGGD; however, opportunistic bacteria may have the capacity to colonise the damaged mucosa and inhibit healing in a small proportion of cases. Therefore, biopsies of mucosal lesions should be obtained in horses that are refractory to treatment. The presence of large numbers of any bacteria may be suggestive of an opportunistic secondary bacterial infection and the judicious use of antimicrobials in such cases may be warranted.

How long do you treat for?

In contrast to ESGD, where healing is usually complete within two weeks of initiating treatment, the rate of healing in EGGD is less predictable and initial treatment should be continued for at least four weeks before gastroscopy is repeated. The goal of treatment should be resolution of clinical signs and a normal appearance to the gastric mucosa on gastroscopy. Unfortunately, up to 25 per cent of horses with EGGD will not respond within four weeks of initiating treatment (Varley and others 2019), and in some cases healing may take many months. The reason for this is unclear, but is likely to be due to variations in the chronicity of disease

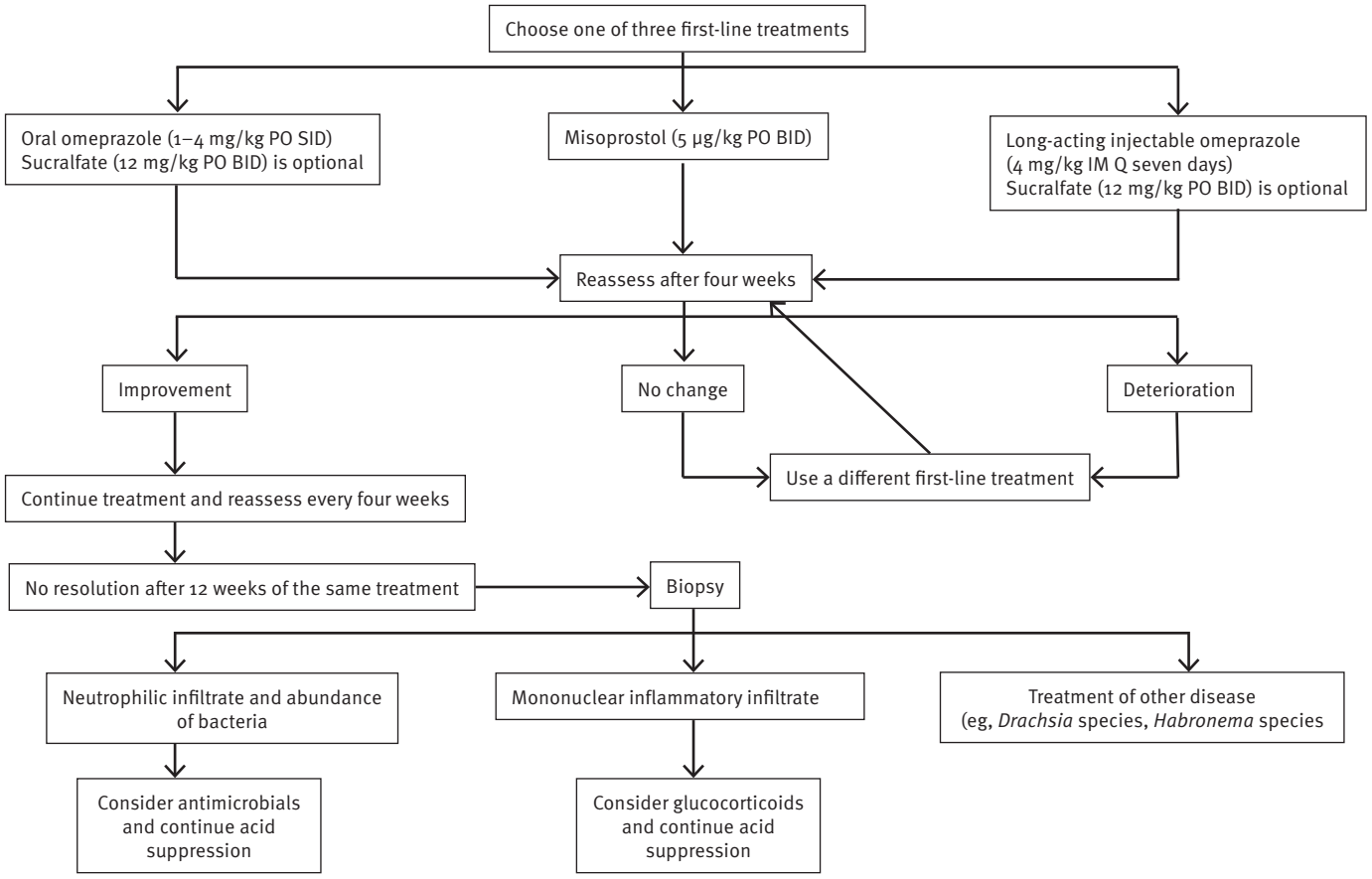

Fig 13: Recommendations for the management of horses with equine glandular gastric disease that do not heal after four weeks of treatment. Adapted from Rendle and others (2018). BID Twice a day, PO Orally, SID Once a day, Q Every

and/or the severity of underlying inflammatory process. Horses with hypertrophic rugal folds (Fig 3) can be expected to take even longer to respond to therapy. In severe cases, the pylorus will be permanently disfigured despite mucosal healing. This is likely due to submucosal and muscular fibrosis, and may result in pyloric dysfunction and delayed gastric emptying (Bezdekova and others 2020).

If there is no improvement in lesion appearance after four weeks, an alternative first-line treatment should be selected. If there is improvement but healing is not complete, then the same treatment should be continued. Irrespective of the treatment used, gastroscopy should be repeated at monthly intervals to assess progress. If there is no improvement after three months, further management should be based on the histological appearance of mucosal lesions obtained from endoscopically guided biopsies. A useful algorithm to guide management of horses with EGGD that do not heal after four weeks of treatment is outlined in Fig 13. Once lesions have healed, treatment can be stopped abruptly. There is no evidence of a rebound increase in acid production in horses once treatment has ended.

Prevention

It is impossible to recommend targeted preventive strategies for EGGD, as the underlying pathogenesis of this disease has yet to be elucidated. However, management changes that mitigate exposure to known risk factors would seem logical. These include minimising changes to routine, avoiding potential stressors, and implementing rest days during the week for horses participating in strenuous activities. There are currently no commercial supplements on the market that have evidence-based claims of efficacy for prevention of EGGD. However, supplementation with oils containing linoleic acid (eg, 45 ml of rapeseed or maize oil daily) may be of benefit, as this has been demonstrated to decrease acid output and increase production of prostaglandin E2.

Summary

EGGD is increasingly recognised in equine practice. It is important to distinguish this disease from ESGD as there are significant differences in the aetiopathogenesis and treatment. The cause of EGGD has not yet been identified, but it is likely to result from a breakdown of the normal defence mechanisms that protect the mucosa from acidic gastric contents. Risk factors that have been identified include stress and exercise frequency, but there is ongoing research in this area. Clinical signs are non-specific and gastroscopy should always be performed to confirm the disease. Current recommendations for treatment and prevention are based on acid suppression and management strategies to mitigate stress and fatigue. Response to treatment is unpredictable and lesions may take several months to heal.

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SELF-ASSESSMENT: MANAGING GLANDULAR GASTRIC DISEASE IN HORSES

This self-assessment quiz can be completed by accessing the online version of this article at bvajournals.onlinelibrary.wiley.com/toc/20427689/2021/43/4

1. **In what region of the stomach are lesions associated with equine glandular gastric disease (EGGD) seen most commonly?**
   a) Dorsal fundus
   b) Ventral fundus
   c) Cardia
   d) Pylorus

2. **Which of the following risk factors have been associated with EGGD?**
   a) Restricted access to turnout
   b) Intense exercise
   c) Lack of experience
   d) Limited access to water

3. **Clinical signs that may be attributable to EGGD include which of the following?**
   a) Bruxism
   b) Stereotypical behaviour
   c) Poor hair coat
   d) Girthing pain

4. **Which of the following drugs should not be used as a first-line treatment for EGGD?**
   a) Oral omeprazole
   b) Ranitidine
   c) Long-acting intramuscular omeprazole
   d) Misoprostol

5. **What is the appropriate course of action for a horse with EGGD that has not responded to treatment with oral omeprazole after four weeks?**
   a) Continue with oral omeprazole
   b) Stop oral omeprazole and start treatment with misoprostol
   c) Continue with oral omeprazole but add sucralfate
   d) Continue with oral omeprazole but add antimicrobials

Answers: (1) d, (2) c, (3) d, (4) b, (5) b