Equine Gastric Ulceration Syndrome – is feeding the key?

Definition

Equine Gastric Ulceration Syndrome (EGUS) is the general term used to describe ulceration of any part of the epithelial tissue of the stomach. In recent years the Equine Gastric Ulcer Council has promoted the sub-division of EGUS into two categories which more accurately describes where the ulcers occur. The first describes ulceration of the non-glandular upper part of the stomach and is called Equine Squamous Gastric Disease (ESGD). The second describes the ulceration occurring in the lower glandular part of the stomach so is termed Equine Glandular Gastric Disease (EGGD) (Sykes, et al., 2015). Ulceration has also been known to occur at the end of the oesophagus and in the upper regions of the duodenum (Andrews *et al.,* 1999). The consensus statement on EGUS published 8 years ago, further divided equine squamous gastric disease into primary disease which occurs in horses with an otherwise normal GIT and secondary disease which occurs in horses with pyloric stenosis. At present equine glandular gastric disease is less well categorised and just reflects the region where it occurs in the stomach. (van den Boom, 2022).

ESGD is categorised using a scoring system recommended by the EGU Council with a range of 0 – 4. The scoring is based on lesion number, size and depth of any lesions (Table 1).

Table 1. Scoring system recommended by the EGU council when describing ulceration of the non-granular mucosa of the equid stomach, adapted from Sykes et al. (2015)

Score	**Description of the squamous mucosa**
0	The epithelium is intact and there is no appearance of hyperkeratosis
1	The mucosa is intact, but there are areas of hyperkeratosis
2	Small, single or multifocal lesions
3	Large single or extensive superficial lesions
4	Extensive lesions with areas of apparent deep ulceration

Currently little data is available on EGGD so grading is not yet standardized. Until this condition is better understood a specific hierarchical scoring system is not recommended. Instead veterinarians are encouraged to describe ulcer presence or absence, their appearance, and where they are located.

Incidence of EGUS

Although 22% of feral horse populations suffer from EGUS (Ward et al., 2015), the incidence is higher in our domesticated population (see Table 2). This is primarily due to management and feeding cereal-based concentrates. Diagnosing EGUS was problematic until the arrival the 2m gastroscope with research commencing only in 1989 (Murray et al., 1989). Further gastroscopy developments, have now described ulceration in all regions of the stomach and it is generally accepted that ESGD is high in most equine disciplines.

Table 2. The incidence of Equine Squamous Gastric Disease in horses in different disciplines in the UK

% of population	**Discipline**	**Source**
80-100	Thoroughbred racehorses in training	Vatistas et al. (1999); Bell et al. (2007)
87	Standardbred trotters	Rabuffo et al. (2002); Jonsson and Egenvall (2006)
66-93	Endurance	Rabuffo et al. (2002); Jonsson and Egenvall (2006).
17-58	Show and sports horses	Murray et al. (1989); McClure et al. (1999) Luthersson et al. (2009).
37-59	Leisure	Murray et al. (1989); McClure et al. (1999) Vatistas et al. (1999)
11	At home leisure	Chameroy (2006)

Recent work has demonstrated that EGGD is also present in both feral and domesticated horse populations. Ward et al. (2015) found EGGD at a rate of 30% in feral horse populations, while others have reported incidence in our domesticated horses of between 35-72% across different equine disciplines and populations. (Luthersson et al., 2009, 2019; Sykes et al., 2015).

Causes of equine gastric glandular disease

The causes of EGGD are yet to be fully elucidated. Currently failure of the normal gastric glandular mucosal defence mechanisms is thought to be the major cause (Sykes et al., 2015). Several studies have implicated stress (Scheidegger et al., 2017 and Sykes et al., 2019) but it is not yet known if

there is any link to feeding. Further details on EGGD can be found in van den Boom (2022) and Rendle, et al., (2018).

Dietary causes of equine squamous gastric disease

Essentially, ESGD occurs because the squamous mucosa is exposed to acid which erodes the surface causing ulceration. Two acids are implicated, hydrochloric acid, which is continually secreted from the glandular mucosal gastric pits (Luthersson and Nadeau, 2013), and plays an essential role in protein digestion and pathogen control in the stomach; and lactic acid and volatile fatty acids, which come directly from fermentation and are the end products of microbial degradation (Andrews et al, 2017). and volatile fatty acids in the stomach should be relatively limited if the horse is fed as it's digestive anatomy dictates (ie trickle-fed high amounts of slowly degraded fibrous feedstuffs). Trickle feeding prevents over-filling of the stomach and maintains the flow of digesta into the small intestine. Mean retention time of feed in the stomach and small intestine should be quite short (2–3 hours; Chaucheyras-Durand et al, 2022), resulting in limited time for significant fermentation to occur, thus acid accumulation in the stomach **should** not be an issue.

Poor dietary choices and feeding management, such as meal feeding high-starch / high-sugar diets can initiate undesirable rapid fermentation and indeed slow stomach emptying. Such a situation leads to high lactic acid and VFA production which can be EGUS inducing (Andrews et al., 2017; Jonsson & Egenvall, 2006, Murray and Schusser,1993, Luthersson, et al., 2009).

The potential for stomach bacteria to produce lactic acid and VFAs is well documented. Julliand and Grimm, (2016) reported high concentrations of the anaerobic bacteria and lactate utilizers, known degraders of starch, can reach up to 10 million colony-forming units per ml of stomach contents, producing 8mmol/L of acid after a meal. Although this capacity for fermentation in the stomach can be activated, it is important to emphasise that high starch/sugar- low fibre diets are neither necessary nor desirable and will be detrimental to gastric health and overall well-being.

The other situations that increase mucosa : acid interaction, is when the diet is low in fibre or when the stomach is empty. Murray and Schusser, (1993) reported stomach pH in horses continually fed Timothy hay was 3.1 while those that were fasted had pH of 1.5. Meal-feeding cereal-based diets can often result in periods when the stomach is empty thus inadvertently increasing acid contact with the mucosa. Periods of 6 hours or more between feeds is associated with EGUS (Luthersson, et al., 2009). Furthermore, low fibre diets which often accompany high-starch, high-sugar diets, lack

the necessary fibre to form a 'fibre mat' on top of the digesta which can help reduce acid splash during exercise (Lorenzo-Figueras and Merritt, 2002).

Alfalfa hay or chaff is alkaline in nature, contains ulcer-protective ingredients such as calcium and protein, and has been shown by Bauerlain et al (2020) to be ulcer-reducing in adult horses. However, a study by Fedtke et al (2015) cast doubt on the suitability of alfalfa chaff as a fibre feed for newly-weaned foals. In the study, newly-weaned foals fed chaff had an increased ulceration score post diet, particularly around the pylorus region of the stomach, compared with those fed hay. Newly-weaned foals are known to exhibit significant levels of squamous gastric disease, and all foals in this study did show increased squamous gastric disease after weaning. However, the foals in this study fed 3 times per day with either molassed or unmolassed alfalfa mixed in with 0.9 kg oats (ie, 2.7 kg oats per day) had significantly more lesions compared with those fed 2.7 kg oats in 2 or 3 meals per day plus ad libitum hay. The authors attributed the increased lesions to the rough nature of the alfalfa stalks which caused an abrasive action to an already compromised organ. This may have been the case, but it is just as likely that the foals chewed the chaff extensively, creating small particle size (which can be ulcer-promoting) which mixed with oats created ideal conditions for ulceration. Furthermore, the difference in the hay vs alfalfa chaff groups could be attributed to the tricklefeeding nature of the hay diet, which may have had a less detrimental effect on gastric health than the meal feeding of the alfalfa chaff.

Equine squamous gastric disease is highly prevalent in weaned foals, and sympathetic feeding during weaning should be the aim to avoid extending the duration or severity of squamous gastric disease. Modifications such as short-term reduction in cereal feeding and taking particular note of constant access to fibre and promoting chewing will help to promote a speedy recovery from post-weaning equine squamous gastric disease. If owners are concerned about feeding alfalfa chaff to foals, the benefits from this feed can still be gained by feeding alfalfa pellets (Vondran et al, 2016). These could be used as a full or partial replacement for cereals immediately after weaning until improvement in squamous gastric disease scores are seen.

High amounts of straw fed to adult horses can also have this physical effect in the stomach. Luthersson et al. (2009) reported a 4.5-fold increase in mucosal ulceration when straw was fed as the sole forage. Straw in limited amounts (0.25kg DM/100kg BW) can be a useful 'filler' for overweight horses but its low nutritional content and limited fermentation in the hind gut, can pre-dispose to impaction colic, so it's use should be limited.

There are several other widely accepted management-related pre-disposing factors for EGUS including: sudden changes in diet (Andrews et al., 2017; Luthersson et al., 2009), stress, electrolyte administration (Holbrook et al., 2005), fasting, starvation, administration of non-steroidal anti-inflammatory drugs (Fennell & Franklin, 2009) and restricting access to water which increases the risk of developing EGUS by 2.5 times. At present unequivocal data to support an un-disputed link of all of these with ESGD is lacking and as such they maybe stimulants for some horses but not for others.

There is clear evidence that keeping horses at pasture reduces, stress, stereotypic behaviours and allows expression of innate behaviours such as foraging (Mach et al., 2020). However, there is no evidence to support that this management system alone will be an effective ESGD prevention strategy. Although the gastric pH of horses kept at pasture has been noted to be higher at 4 than when horses are stabled for long periods, if horses are trained and fed cereals while at pasture the incidence of EGUS can still be high.

Symptoms, Clinical signs

Symptoms of EGUS are multifactorial but only the following ones have a well-supported association with ESGD:

- Colic, where a reported association between ESGD and colic was found in 83% of horses with recurrent episodes of colic (Vatistas et al., 1999;, Murray et al., 1989; Dukti et al., 2006)
- Poor performance in Thoroughbred racehorses (Vatistas et al 1999) and Standardbred racehorses, (Jonsson and Egenvall, 2006) are associated with the presence of gastric ulcers, independent of their severity or the number of ulcers.

Other symptoms reported from a range of individual studies are: weight loss, poor appetite, poor coat, diarrhoea, girthing issues and reluctance to work.

While many of these might be caused by squamous gastric disease, there is a lack of data to support unequivocal association which makes them unreliable diagnostic tools. Crib biting, a common stereotypy has been linked with squamous gastric disease (Luthersson and Nadeau, 2013) but to date, little data has been gathered to link the onset of crib biting with squamous gastric disease. The presence of crib biting in horses is more likely because of stress associated with inappropriate management regimes, rather than the presence of ulcers per se (Daniels et al, 2019; Sykes et al, 2019). The suggestion that crib biting is performed to relieve stomach discomfort is highly tenuous. Crib biting does not increase saliva production (Houpt, 2012) so it is highly unlikely that stomach pain relief is the stimulation for this behaviour. If any relationship does exist, it is more likely that the

action of crib biting stimulates the vagus nerve, which in turn stimulates gastrin production (from parietal cells) and therefore increases gastric secretions.

Diagnosing equine squamous gastric disease

The only sure way of diagnosing ESGD is to perform gastroscopy (Andrews *et al.,* 1999). However, gastroscopy is invasive and the horse needs to be starved for 12 hours prior to the procedure which can be ulcer inducing in itself. Loss of mucosal integrity can be seen during gastroscopy but grading lesions tends to be lower than grades given on necroscopy, thus correlating lesion grade with clinical signs can be challenging (van den Boom, 2022). This makes linking symptoms to ulceration difficult, so veterinarians need to take account of gastroscope results, on-set of clinical signs, individual horse activity and history when reaching a diagnosis.

An objective system of scoring some of the behavioural issues such as girthing behaviour, behaviour suggesting the horse cold-backed and reluctance to work, alongside recording empirical data on weight loss and feed intake might help in the selection of more accurate indicative symptoms as diagnostic features for EGUS.

Several non-invasive tests on the market have attempted to use parameters such as faecal albumin or haemoglobin as indicators of EGUS but no data has been reported to support their accuracy in diagnosing ESGD. (Sykes et al., 2014).

Treatment of equine squamous gastric disease

As most ESGD will require veterinary intervention for initial healing, drug therapy that centres around reducing the acid : mucosa interaction is favoured. Omeprazole is the drug of choice as it has been proven to reduce acid secretion. The buffered /coated form of omeprazole, GastroGarda at a dose of 4 mg/kg once daily for 28 days, consistently produces ESGD healing rates of 70–77%. (Andrews et al., 1999).

Several alternative treatments for EGUS have been fully reviewed by van den Boom (2022) and while B-vitamins and organic acids, sea buckthorn berries and antiacid seem to give symptomatic relief; others such as a combination of an antacid (magnesium hydroxide), a pectin-lecithin complex and Saccharomyces cerevisiae have shown promise as prophylactic agents for both ESGD and EGGD.

Prevention of equine squamous gastric disease

As poor feeding management is one of the major causes of ESGD, a good feeding regime will be the best way to prevent it. Appropriate feeding will depend on the activity and level the horse is

performing at. However, in all cases quality fibre should be the basis of the diet and other nutrient sources (fat and cereals) should only be added when energy demands are at a level that cannot be met by fibre alone. If diets are constructed with a strong base of quality fibre (a minimum of 1% bodyweight per day and preferably 1.5%), then behavioural needs will also be met and this should help to reduce general stress, as well as reducing ulcer formation.

Although high forage diets are strongly associated with low levels of ESGD, it is essential to note that feeding high levels of forage will not prevent ESGD if high levels of cereals and sugars are also fed. Galinelli et al. (2021) reported that horses fed diets that were higher than 1g/kg bodyweight per meal of starch and sugar still presented with ulcers despite being fed >2% dry matter forage/ kg bodyweight per day. However this study also demonstrated the importance of adequate forage in the diet, as out of a group of 48 horses, 27 with ESGD and 21 controls, 21% of the ESGD horses had inverted cereal : forage ratios and 86% of that group had forage intakes lower than 1.5% of bodyweight/day. Some of the horses i.e., 82% of clinical ESGD cases, were fed diets that contained more than 2g of sugars and starch/kg BW per day, and in 41.5% of the diets, the sugars and starch provision was higher than 2g/kg BW per meal, which is well above current recommendation for diet formulations. EGUS free horses were also fed an average of 3 (SD ± 0.3) meals per day, while the EGUS sufferers received only 2 meals/day, suggesting that feeding management plays a role.

As ESGD is present in feral horses, there will be horses that are fed high-forage low cereal diets that still present with ESGD. Indeed Galinelli et al .(2021) reported that 17% of all horses that presented with EGUS had sugar and starch intakes below 1g/kg BW/meal. This suggests for some horses the main cause of ESGD was other than diet formulation.

Conclusions

Equine squamous gastric disease is highly prevalent in the horse population and is associated with a range of negative welfare conditions such as colic, weight loss, poor appetite, girthing discomfort, poor coat and diarrhoea. Equine squamous gastric disease is best diagnosed using a combination of gastroscopy and case history, as other currently available tests are unreliable. The cause of the ulceration is acid erosion of the squamous epithelial tissue, so strategies to reduce the production and interaction of acid with the mucosa should be a priority when preventing and treating equine squamous gastric disease. While many management factors are postulated to play a role in ulcer formation, the main proven initiators are meal-feeding high amounts of starch and sugar, underfeeding fibre and long periods without feed. To minimise equine squamous gastric disease, all

horses should be fed 1.5% of their body weight in dry matter per day as fibre. When energy demands dictate that cereals are needed in the diet they should be given in several small feeds over the course of the day, and water should always be available ad libitum both in the stable and at pasture.

Key points:

1. Provide continuous access to forage (grass or hay) ensuring a minimum intake of 1.5 kg DM / 100 kg BW/day
2. Add quality fibre to the ration before adding cereals and fat
3. Give cereals in small meals i.e., 4-6 times /day and no more than 1g starch/kg BW/meal
4. Supply water ad libitum
5. Give electrolytes in feed rather than in water

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