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Equine Gastric Ulceration Syndrome (EGUS) is a common problem in all types of horses working in all disciplines. An ongoing Internet-based survey by Merial Animal Health in the UK using an online owner information derived risk assessment tool suggests that one third of pleasure horse owners in the UK think that their horse has a high risk of having EGUS based on assessment of management, diet and workload. A further 21% feel there horses are at medium risk. These data in themselves warrant taking EGUS seriously in animals beyond the more typically affected racehorse and high performance Sport Horses. The prevalence varies with the management and the nature of work in a particular horse or group. However the effect of having gastric ulcers on an individual horse is quite variable and not predictable. This presentation will address normal gastric structure, physiology and function, the pathophysiology of EGUS, its prevalence, clinical signs including effects on performance, diagnosis and some brief notes on management.

GASTRIC ANATOMY, PHYSIOLOGY AND FUNCTION

The upper 50% of the equine stomach is lined by a stratified squamous epithelium (“squamous” mucosa) and the lower half by a complex compound glandular epithelium (“glandular mucosa”). There are some structural and functional variations between the cardiac, fundic and pyloric regions of the glandular mucosa but their clinical relevance is unclear. A thick mucus curtain coats the glandular mucosa and has a critical barrier function to protect the underlying mucosa. Through a complex mixture of mucins, lipids, trefoil proteins and bicarbonate ion trapping a pH gradient is created with the highest pH (6.5) immediately adjacent to the mucosal surface and the lowest at the luminal surface (1.5). The squamous mucosa has minimal barrier protection that is limited to a thin mucus layer. Adequate glandular mucosal perfusion and prostaglandin levels are critical to the health and integrity of this region. The glandular mucosa continuously secretes gastric juice irrespective of food intake (1.5L/h). An H+/K+ ATPase “proton” pump in the parietal cells within the gastric glands continuously secretes hydrogen ions that are delivered via acid fingers or tunnels through the mucus layer into the lumen combining with chloride ions to produce HCl. With every H+ ion a HCO3− ion is produced and delivered to the mucosal surface in capillary blood flow where it is incorporated into the mucus barrier. Acid production is enhanced by histamine release stimulated by gastrin release during feeding, exercise and periods of stress. Prostaglandin E-regulated glandular mucosal blood flow is therefore critical to the integrity of this tissue. Normally stratification of food (solid and liquid) within the stomach is important for protection of the squamous mucosa which is highly susceptible to acid injury. A mat of low density fibrous material (pH 6-7) floats on a medium density mat (pH 4-5) which in turn sits on top of a high density highly acidic (pH 1-2) liquid layer within the glandular body.
The normal pH at the margo plicatus is pH 4.1 rising to pH 5.5 in the squamous fundus. The squamous mucosa has minimal protection against peptic acid injury, ulcerates and heals rapidly but has a huge proliferative healing ability such that even the most severe ulcers will heal without any evidence of scarring. The glandular mucosa ulcerates and heals much more slowly.

PATHOPHYSIOLOGY OF MUCOSAL INJURY

Squamous injury in predominantly due to acid injury through inappropriate exposure to HCl, and to a lesser extent pepsin (cleaved from pepsinogen when pH <4), bile acids (excess pyloric bile reflex) and VFA and lactic acid (carbohydrate fermentation).

Glandular mucosal injury occurs when normal barrier function fails (reduced mucosal perfusion, reduced bicarbonate delivery, mucus layer failure). This can include COX 1 inhibition by systemic NSAID administration as well as direct topical toxicity by oral NSAIDs. There is some evidence that bacterial infection (an equine *Helicobacter spp*. variant and others) may be involved in glandular mucosal injury and the propagation of squamous injury. Starch fermenting bacteria occur in the equine stomach producing lactic acid which can exacerbate the effects of HCL. When squamous ulcers develop, mucosal bacterial populations reduce in diversity and increase in numbers. Moreover 4 weeks of antibiotic therapy alone can result in both symptomatic relief and significant healing in squamous mucosal ulcers in horse in simulated race training. Thus the case for at least a contribution by bacteria to the pathophysiology of EGUS is now established.

RISK FACTORS FOR GASTRIC ULCERATION

1. **Exercise** – Ulcer prevalence and severity increases with increased training activity and declines but does not disappear if animals are removed from training. Speeds greater than trot lead to a fall in pH in the upper, squamous region due to liquid acid reflux/splashing/compression from the glandular fundus. This is exacerbated in starved animals. pH begins to rise within 5 minutes of stopping exercise. Treadmill exercise at gallop results in an increased number and severity of squamous ulcers in comparison to maximal exercise in trotters. Serum gastrin increases during exercise. Elite endurance horses have a high prevalence of ulcers during the competition season that falls in the off season.

2. **Intermittent feeding** – Starvation for a 24 h period disrupts gastric stratification and results in increased bile reflux through the pylorus which has a synergistic effect on acid injury to the squamous mucosa. Repetitive periods of starvation and feeding can result in severe ulceration in some animals within 4 days. Feed deprivation models that readily induce squamous mucosal injury do not produce glandular ulcers.

3. **High concentrate diets** – Induce gastric acid secretion via gastrin, increases gastric fluidity leading to greater mixing of upper and lower content strata. Carbohydrate...

THREE TYPES OF GASTRIC ULCERATION ARE RECOGNIZED

1. Primary squamous ulceration
   a. Driven by excess acid exposure
   b. Most common form of ulceration in may observational studies

2. Primary glandular ulceration
   a. Failure of mucosal defences
   b. Less common in most observational studies

3. Secondary squamous ulceration
   a. Excess acid exposure due to impaired gastric emptying
   b. Physical or functional obstruction of gastric outflow or proximal duodenum.
   c. Rare
fermentation in the stomach increases the concentration of highly lipid soluble VFAs. Studies in Denmark showed that a high starch diet (>1g/Kg body weight [BW] per meal increased the risk of ulcers 2 fold. But periods of forage deprivation increased the risk of ulcers 4 fold. Coarse, higher fibre feed requires more mastication resulting in greater salivary bicarbonate buffering. Therefore regular or continuous access to roughage is critical for maintaining gastric mucosal integrity.

4. Stabling, transport, changes in social grouping and other stressors – Individual stable confinement, light exercise and concentrate feeding of previously grouped yearlings resulted in significant ulceration by 2 weeks which progressed over the 10 week study period. In Australia Thoroughbreds were turned out to pasture for 6 weeks after completion of a simulated race training model of gastric ulceration. Existing ulcers resolved in 65% but new superficial ulcers developed in 42%. Ulceration is common (50%) in non-working donkeys at post-mortem following death or euthanasia due to disease or injury. Although intuitively maintenance at pasture should result in a reduced EGUS prevalence this is not the case if other stressors such as training continue. Choice for an individual animal as to whether they have access to a stable, a yard or pasture may be more important than our perception that pasture turnout is the best environment for the modern horses

5. NSAID drugs – due to blockade of prostaglandin-mediated COX-1 effects particularly affecting the glandular mucosa and combinations or stacking NSAIDs rapidly cause severe squamous ulceration.

6. Systemic illness may result in significant ulceration possibly associated with changes to intestinal mucosal blood flow.

7. Stress, stereotypies & crib-biting in yearlings - crib-bitters in young stock (foals, weanlings and yearlings) have more ulceration and cribbing may reduce with treatment. In adults that are long term established crib-biters it is rare to find EGUS except in sudden onset or exacerbation cases.

PREVALENCE

Many studies from around the world have demonstrated a high prevalence of Equine Gastric Ulceration Syndrome within performance horse populations (see table below). Some of these studies have reported an increasing prevalence and ulcer grade with increasing intensity and duration of training. Although the prevalence of ulceration is lower amongst pleasure horses and those working at lower speeds it still quite widespread and in some individuals is unexpectedly severe even if other risk factors are absent. Unfortunately few of these prevalence studies have accurately documented the severity or grade of the ulceration as well as their presence. There was a paucity of information regarding prevalence of EGUS amongst horse populations in the UK and few if any studies had attempted to address the effect of EGUS on performance. Anecdotal evidence would support improved performance following treatment of moderate to severe ulceration but is much less clear with lower grades of ulceration.

<table>
<thead>
<tr>
<th>Discipline/ Type</th>
<th>Squamous %</th>
<th>Glandular %</th>
</tr>
</thead>
<tbody>
<tr>
<td>TB Racing (H K)</td>
<td>80</td>
<td>-</td>
</tr>
<tr>
<td>TB Retired (H K)</td>
<td>52</td>
<td>-</td>
</tr>
<tr>
<td>TB Racing (USA)</td>
<td>100</td>
<td>51</td>
</tr>
<tr>
<td>TB Training (USA)</td>
<td>93</td>
<td>51</td>
</tr>
<tr>
<td>STB Racing (CAN)</td>
<td>56-93</td>
<td>↑ with training</td>
</tr>
<tr>
<td>STB Racing (SWE)</td>
<td>70</td>
<td>↑ with training</td>
</tr>
<tr>
<td>TB Racing (NZ)</td>
<td>88</td>
<td>Training no effect</td>
</tr>
<tr>
<td>H/ Dressage (USA)</td>
<td>60</td>
<td>-</td>
</tr>
<tr>
<td>Endurance (USA)</td>
<td>57-93</td>
<td>27 (Bleeding)</td>
</tr>
<tr>
<td>Show Horses (USA)</td>
<td>58</td>
<td>1</td>
</tr>
<tr>
<td>Eventers</td>
<td>75</td>
<td>-</td>
</tr>
<tr>
<td>Broodmares at grass</td>
<td>70</td>
<td>57</td>
</tr>
<tr>
<td>Foals</td>
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DATA FROM THE AUTHOR’S PRACTICE

Retrospective data from animals presented over a 13 month period were collated. As the animals were selected by Owner/Trainer/Vet they represent a biased population for prevalence studies.

Horses were divided into groups by discipline as racehorses or pleasure / private low-mid grade performance horses.

All animals underwent overnight (16-18 h) starvation with withdrawal water 3 h before examination. Animals were housed on a bed that they would not eat or were muzzled.

The grading systems for ulceration of both squamous and glandular mucosae were as published by the Equine Gastric Ulcer Council (1994). Grades 2, 3 & 4 were considered significant.

Grade 0: Intact epithelium, no erythema & hyperkeratosis
Grade 1: Areas of erythema &/or hyperkeratosis, mucosa intact
Grade 2: Small single or multifocal lesions
Grade 3: Large, single or multifocal or extensive multifocal lesions
Grade 4: Extensive lesions with areas of apparent deep ulceration

EGUS in 90 Racehorse presented with suspected clinical disease
Prevalence 92%, mean ulcer grade 2.7, mean ulcer grade of significant ulceration (grades 2-4) 3.0

EGUS in 120 pleasure/performance horses presented with suspected clinical disease
Prevalence 60%, mean ulcer grade 1.6, mean ulcer grade of significant ulceration (grades 2-4) 2.4

Therefore racehorses are more likely to have ulceration than pleasure/low-mid grade performance horses and that ulceration is likely to be more severe.

ARE CRIB BITERS AT HIGHER OF EGUS RISK THAN NON-CRIB BITERS?

In a separate group EGUS in 25 adult crib biters as the only presenting sign were compared to the pleasure horse population. Prevalence 72%, mean ulcer grade 2.0, mean grade of significant ulceration 2.6 vs. Prevalence 60%, mean ulcer grade 1.6, mean ulcer grade of significant ulceration (grades 2-4) 2.4

DOES EGUS CAUSE RECURRENT COLIC?

In most studies colic is reported to be a rare presenting sign in animals with EGUS. EGUS in 25 recurrent colics vs general population. Prevalence 68%, mean ulcer grade 2.1, mean grade of significant ulceration 2.7 vs Prevalence 60%, mean ulcer grade 1.6, mean ulcer grade of significant ulceration (grades 2-4) 2.4. It is more likely that EGUS is a common secondary effect in animals under stress associated with recurrent episodes of colic than a primary driver of recurrent abdominal pain in all but a small number of cases.

CLINICAL SIGNS IN ADULT HORSES

Often signs are vague but include picky appetite, not eating up (“leaving”), failure to maintain adequate body condition – often these signs appear and/or progress as training intensity, speed and workload increase. Low grade recurrent colic (? post-prandial) is uncommon but we may be poor at identifying the behavioural changes associated with low grade “gastric” pain. Poor performance, behavioural changes and sour, poor jumping may all be associated with increasing workload and likely reflect pain related to ulcers. Changes in temperament and reduced ridden performance are currently the commonest reasons for gastroscopy in non-racehorse in my practice. When low grade EGUS is identified, especially glandular mucosal injury (grade 2/4) a trial course of acid suppression (7-14 days, 4 mg/kg daily as Gastrogard) can help to clarify.
the significance of the findings. Some individuals will show dramatic responses to treatment with marked improvements in appetite, temperament and performance. This improvement may be noted as little as 2-5 days into treatment as ulcers cease to be bathed in acid gastric contents. There is marked individual variation in both susceptibility to ulceration and the correlation between grade (severity and distribution) and clinical signs particularly in sport horse.

EFFECTS ON PERFORMANCE

The effect of EGUS on performance has often been questioned and there is little data on this but the responses to treatment seen in both race and sport horses suggest a significant effect on performance in many horses especially those with moderate to severe (grade 3&4/4) EGUS. A small study at Bristol University looked at the pre and post EGUS diagnosis and treatment performance of 4 racehorses with grade 3 and 4/4 ulcers where EGUS was the only clinical abnormality found during a detailed poor performance. A significant improvement in winnings and ratings was documented. An unpublished study routinely screening elite sport horses in the Team GB team squad found ulcers in 80% of the horses. Of these half had some sort of recognizable clinical sign although the effect was always low grade. The other half were asymptomatic. The ulcers were treated in all cases and of the asymptomatic group 50% showed an improvement in performance and consistency no doubt due to elimination of low grade pain.

References upon request.