Gastric Ulcers: A Pain in the Gut!

By Frank M. Andrews, DVM, MS, DACVIM

Gastric ulcers are common in horses, with a prevalence estimated from 53 to 93%.[1-3] Gastric ulcers can lead to decreased performance, vague clinical signs and may go undiagnosed for months. Gastric ulcers are primarily caused by stomach acids. However, the anatomy of the stomach, diet, restricted feed intake, exercise, stress (stall or transport), and the use of non-steroidal anti-inflammatory agents (NSAIDs) are risk factors for development of gastric ulcers. Because many factors are involved in the cause of gastric ulcers, the term Equine Gastric Ulcer Syndrome (EGUS) was coined in 1999 to describe the condition of erosions and ulcerations occurring in the distal esophagus, non-glandular (NG) and glandular stomach, and proximal duodenum of horses.[4] All ages and breeds of horses are susceptible to gastric ulcers and current therapeutic strategies focus on blocking gastric acid secretion and raising stomach pH. Pharmacologic agents are needed to treat these conditions, however, a comprehensive approach including correcting the underlying cause, environmental management, and dietary manipulation is needed for successful prevention.

PATHOGENESIS

Horses are continuous hydrochloric acid (HCL) secretors, and acid exposure is the primary cause of NG gastric ulcers in horses. Also, performance horses are typically fed low-roughage, high grain diets containing water-soluble carbohydrates (WSCs). A diet high in WSCs provides substrates for gastric fermentation by resident bacteria. Gastric fermentation by-products, such as volatile fatty acids (VFAs), alcohol, and lactic acid (LA), may damage the NG squamous mucosa. Several species of bacteria (*Lactobacillus, Streptococcus, E. coli*) were isolated from the stomach of horses, adding credence to this theory.[5] Previously, an in vivo study in cannulated horses showed that a diet high in WSCs and protein (alfalfa hay/sweet-feed grain diet) produced high VFA concentrations in the stomach.[6] The presence of VFAs (butyric, propionic, and valeric acids), carbohydrate fermentation by-products, and low gastric juice pH were important predictors of ulcer severity. Furthermore, these VFAs have been shown to disrupt chloride-dependent Na transport within the NG mucosa and cause cell swelling and ulceration.[7,8] HCl (gastric juice pH < 4.0) acts synergistically with VFAs on the NG mucosa to cause further damage.

Glandular ulcers are likely caused by a breakdown in the protective factors, such as reduced blood flow, decrease in prostaglandins, and decreased mucus secretion. Stress and the use of NSAIDs are important in causing ulcers in this region.

RISK FACTORS

Exercise Intensity

Horses in training and racing are at high risk of developing NG gastric ulcers.[9] Previously, horses running on a high-speed treadmill showed increased abdominal pressure and decreased stomach volume.[10] Compression of the stomach allowed acids from the glandular mucosa to reflux into the NG region ("acid splash"), leading to NG mucosal injury. Intense exercise likely increases exposure of the NG mucosa to acids, which explains the increased prevalence of gastric ulcers in horses in race training and racing. Furthermore, an increase in serum gastrin concentration occurs during exercise;[11] which stimulates HCl secretion resulting in a lower stomach pH.

Intermittent vs. Continuous Feeding

Horses grazing at pasture have a decreased prevalence of EGUS. During grazing, there is a continuous flow of saliva and ingesta that buffers stomach acid and maintains stomach pH > 4 for a large portion of the day. Conversely, when feed is withheld from horses, before racing or in managed stables, gastric pH decreases rapidly and the NG mucosa is exposed to an acid environment. Intermittent feeding has been shown to cause and increase the severity of NG gastric ulcers, and an alternating feed-deprivation model was developed to produce NG gastric ulcers experimentally.[12] The NG mucosa is the most susceptible to ulceration in horses subjected to intermittent feeding due to its lack of mucosal protective factors. Studies have shown that stomach pH drops 6 h after feeding and dry matter (DM) content decreases 12 h after feeding a mixed-feed diet compared with horses fed a hay diet.[6,13] Thus, horses should be fed hay continuously or every 5 to 6 h to buffer stomach acids.

Diet

Diet has been implicated as a risk factor for EGUS. Serum gastrin concentrations increase significantly in horses fed high-concentrate diets. In addition, as noted previously, concentrate diets are high in WSCs and are fermented by resident bacteria, resulting in the production of VFAs, which, in the presence of low stomach pH (< 4.0), cause damage to the NG mucosa.[7,8] In another study in horses fed a high-protein and high calcium (alfalfa hay/grain) diet showed higher stomach pH values when compared to horses fed a low protein and low-Ca (brome grass hay) diet. Also, horses on the alfalfa hay diet (high-protein, high-Ca) had fewer and less severe gastric ulcers.[6] Feeding alfalfa hay may have some protective effect on the NG mucosa in horses. The protective effects of alfalfa hay fed to exercising horses was confirmed in a more recent study.[14] Furthermore, horses fed mixed feed (128 g of crude protein (CP) and 175 g of crude fiber/kg of DM) for at least 14 d showed increased gastric ulcers in the NG mucosa localized along the Margo plicatus compared with horses fed a hay diet. A diet high in grains (WSCs [sweet feeds]) has been implicated in causing NG gastric ulcers in horses, however alfalfa hay has a protective effect by buffering gastric juice pH and decreasing the effects of VFAs on NG mucosa.

Stall Confinement

Stall confinement has been implicated as a risk factor for EGUS.[15] In that study, 6 of 7 horses housed in stalls had gastric

ulcers, whereas no horse had gastric ulcers after 7 days turn out to pasture. However, in another study, the prevalence of ulcer severity did not differ significantly between horses stabled full-time, horses kept in a stable part-time or horses kept in a pasture full-time.[16] One study in evaluating housing in horses showed that neither proximal stomach nor ventral stomach pH changed significantly in horses housed in stalls alone, housed in stalls with a companion or housed in a grass paddock.[17] However, pH in the proximal stomach was lower during the early morning hours regardless of the housing and feed intake was lowest during these hours. Thus, other factors may play a role in stabled horses that increase the risk of EGUS. Stabled horses are typically fed two large meals daily. These meals are traditionally high in grains and consumed rapidly, which lead to a decrease in saliva production and less buffering of stomach contents.

Non-steriodal Anti-inflammatory Agents

The use of NSAIDs is common in horses presenting with acute abdominal pain. Phenylbutazone or flunixin meglumine are typically given to control pain during a colic episode. These agents have been shown to cause gastric ulcers in horses, but usually at higher than therapeutic doses.[18] Also, the use of NSAIDs in racehorses has not been shown to be a risk factor for EGUS in other epidemiologic studies. Furthermore, NSAIDs are thought to cause more severe ulcers in the glandular mucosa because of their effect on prostaglandin inhibition. Prostaglandin inhibition results in decreased mucosal blood flow, decreased mucus production, and increased HCl secretion. Although prostaglandins are also important in the regulation of acid production and Na transport, it may be their effect on mucosal blood flow that is the most important. Adequate blood flow is necessary to remove hydrogen ions that diffuse through the mucus layer covering the glandular mucosa. Gastric mucosal ischemia may lead to a hypoxia-induced cellular acidosis, release of oxygen-free radicals, phospholipase, and proteases, which may damage the cell membrane leading to necrosis. While NSAIDs are commonly used, they have the potential to exacerbate EGUS in horses with colic and one should use them with caution.

Helicobacter spp. and other Bacteria

Helicobacter spp. has been isolated from man and a variety of animals suffering from gastric ulcers and gastritis. Recently, a new enterohepatic *Helicobacter* species, *Helicobactor equorum*, was isolated from fecal samples of two clinically healthy horses [35].[19,20] Also, Helicobacter equorum DNA was demonstrated in the feces of 2 of 7 (28.6%) foals less than 1 month of age and 40 of 59 (67.8%) foals 1 to 6 months of age. Furthermore, Helicobacter-like DNA was detected in the stomach of 10 Thoroughbred horses in Venezuela.[21] In this study, Helicobacter-like DNA was detected in two out of seven horses with gastric ulcers, three out of five horses with gastritis and five out of six horses with both pathologies and one horse with normal gastric mucosa. Furthermore, ten out of eleven horses infected with Helicobacter had either gastric ulcers or gastritis or both pathologies. However, 39% of the horses in that study did not have gastric lesions, so multiple causes are likely.

Once gastric ulcers are present, other bacteria have been implicated in inhibiting ulcer healing. Bacteria, including *E. coli*, were cultured from the stomach of horses.[5] In rats, which have a compound stomach similar to horses, *E. Coli* administered orally rapidly colonized acetic acid-induced gastric ulcers and impaired healing.[22] Oral antibiotic treatment with streptomycin and/or penicillin suppressed bacterial colonization of the ulcer and accelerated ulcer healing. Also, oral administration of lactulose resulted in an increase *Lactobacillus spp.* growth and colonization of the ulcer bed. Accelerated ulcer healing was seen in the rats compared to placebo treated controls. Thus, bacterial colonization of gastric ulcers in the stomach of horses may delay ulcer healing and in this case treatment with antibiotics may be indicated.

CLINICAL SIGNS

Clinical signs associated with EGUS are often vague and include partial anorexia, mild colic, dull and/or rough hair coat, weight loss, poor performance, change in behavior, and halitosis. Ulcers are more common in horses showing clinical signs. For horses with a client complaint of conditions associated with gastric ulcers, or showing subtle signs of poor health, gastric ulcers were identified in 88-92% compared to 37-52% identified in horses not showing clinical signs. In addition to an increased prevalence of ulcers in clinically affected horses, the severity of ulceration is correlated with the severity of the symptoms.

DIAGNOSIS

Diagnosis of EGUS requires a thorough history, identification of risk factors, physical examination, and a minimum data base. However, gastroscopy is the only definitive diagnosis for gastric ulcers. Standing gastroscopy procedures have been described in detail elsewhere in the literature and require at least a 2 meter endoscope to visualize the NG mucosa and Margo plicatus and a 2.5m to 3m endoscope to visualize the pylorus and proximal duodenum in most adult horses.[4] Use of a gastric ulcer scoring system allows clinicians to compare gastroscopic findings, monitor ulcer healing, and evaluate efficacy of treatment.

Currently there are no hematologic or biochemical markers to diagnose EGUS. However, a recent report showed that horses with gastric ulcers had lower RBC counts and hemoglobin concentrations than horses that did not have gastric ulcers.[23] Some horses with EGUS might have a decreased PCV or total solids over time, but these values are rarely outside normal reference range. Other presumptive diagnostic testing includes a sucrose (table sugar) permeability test, where urine sucrose is measured as an indicator of ulcer severity.[24] Sucrose appears in the urine because it is absorbed through gastric ulcers and if the urine concentration is ≥ 0.7 mg/ml, then the sensitivity and specificity is 83% and 90%, respectively, to detect ulcers in horses. Unfortunately, use of high performance liquid chromatography (HPLC) is required to measure sucrose concentrations.

Fecal occult blood testing (FOBT) has been shown to be helpful in the diagnosis of gastric ulcers.[25] Since this early study, a new fecal occult blood test (SUCCEED™ Equine Fecal Blood Test, Freedom Health LLC., Aurora, OH) was developed to aid in the diagnosis of gastric ulcers. The positive and negative predictive value of the FOBT for the diagnosis of gastric ulcers was good, according to the package insert. The test utilizes specific equine monoclonal antibodies to both albumin and hemoglobin in an easy to use kit. This test should be used

as part of a complete work-up and not as a stand-alone test. False positive FOBT may result if there was a recent rectal examination, rectal biopsy or other rectal trauma, as well as any protein losing enteropathy.

When gastroscopy is not available and ulcers are strongly suspected, it may be worthwhile to start empirical treatment and observe for resolution of clinical signs. If the horse does not respond to treatment, further evaluation is indicated.

MANAGEMENT

The mainstay of pharmacologic treatment of EGUS is to suppress hydrochloric acid (HCl) secretion and increase stomach pH. Because of the high recurrence rate, effective acid control should be followed by nutritional and dietary management strategies to prevent ulcer recurrence.

Pharmacologic Therapy

Omeprazole

Omeprazole paste (4 mg/kg, PO, q24h; Gastrogard®, Merial Limited, Duluth, GA) significantly inhibits gastric acid secretion for 24 hours in horses.[26] In an acid environment omeprazole is activated to a sulphenamide derivative and binds reversibly to the H+/K+ ATPase in parietal cells and inhibits transport of hydrogen ions into the stomach.[27] Because of its effect on the cell, omeprazole is often called a proton-pump blocker and the effect on gastric acid secretion is dose and time dependent. Omeprazole is metabolized in the liver and excreted in urine and bile, and significant liver disease may affect the metabolism of the drug. Omeprazole has been shown to be an effective treatment for EGUS at a dose of 4 mg/kg orally once daily.[28] Omeprazole has been shown to be superior to ranitidine in healing and preventing gastric ulcers in training and racing horses.[29] Furthermore, there was improved performance, weight gain, attitude, appetite, and appearance after treatment. A second study found that 99% of spontaneous ulcers in adult horses and foals more than 4 weeks-of-age were improved with 86.7% healed with omeprazole treatment.[30] Omeprazole also increased the rate of ulcer healing in horses removed from race training.[31] The effectiveness of GastroGard® can be evaluated by aspirating gastric juice from the biopsy chamber of the endoscope or a nasogastric tube and measuring pH. A gastric juice pH > 4.0, 18 hours after omeprazole administration, is considered effective.

An FDA-approved IV formulation is not currently available, however a recent study showed that omeprazole sodium (0.5 mg/kg, IV; Premier Pharmacy Labs, Inc., Weeki Wachee, FL) increased mean gastric juice pH of greater than 4.0, 1 hour after administration.[32] This omeprazole compound might show promise for treatment of EGUS in horses with dysphagia, gastric reflux, or other conditions that restrict oral intake of omeprazole paste. However, due to the variability of acid suppression after the first dose, a loading dose (1.0 mg/kg, IV, Q24h) was recommended.

Histamine Type-2 Receptor Antagonists

Cimetidine

Cimetidine has been used since the early 1980s to treat and prevent ulcers in horses and foals, but there is little scientific evidence in the veterinary literature showing that it has efficacy in the treatment of EGUS. The author cannot recommend cimetidine for treatment or prevention for EGUS.

Ranitidine

Ranitidine hydrochloride is four times more potent than cimetidine.[33] When given orally (6.6mg/kg, PO, q8h), ranitidine suppresses acid output and maintains a median stomach pH of 4.6.[34] Ranitidine (6.6 mg/kg, po, q8h) was able to successfully limit ulcer development in a feed deprivation model (Table 1). Lower doses (4.4 mg/kg, PO, q8h) given orally are ineffective for treatment of EGUS. The recent availability of the generic ranitidine has made this drug popular in treating EGUS. While ranitidine has been the most studied, other H2 antagonists have been evaluated experimentally and may allow for less frequent dosing and more effective acid suppression.

Coating or Binding Agents

Sucralfate and bismuth subsalcyclate are two compounds that bind to stomach ulcers and promote healing. Sucralfate is a hydroxyl aluminum salt of sucrose octasulfate and binds to the negatively charged particles in the ulcer bed, buffering HCL by increasing bicarbonate secretion, stimulating prostaglandin production, and adhering to the ulcer bed.[35] In the stomach, sucralfate is converted to a sticky amorphous mass, which is thought to prevent diffusion of HCl into the ulcer. In a clinical trial in horses, sucralfate (22 mg/kg, PO, q8h) did not improve healing in 6- and 7-month-old foals and therefore alone may not be beneficial in treatment of EGUS. However, it may be effective when used in conjunction with acid suppressive therapy or in the treatment of colonic ulcers.

Bismuth containing compounds may have a coating effect similar to sucralfate. Additionally it will inhibit the activation of pepsin and increase mucosal secretion. A compound containing 26.25 g of bismuth failed to raise stomach pH in 5 horses.[36] Bismuth subsalicylate may be converted to sodium subsalicylate in the gastrointestinal tract, which may cause gastric irritation. Also, salicylates, similar to aspirin, decrease prostaglandin secretion and may further compromise an already damaged mucosa. Thus, compounds containing bismuth are not recommended for treatment of EGUS.

Synthetic hormones

Misoprostol, a synthetic PGE 1 analogue, is effective in the treatment of gastric and duodenal ulcers in man. Acid suppression, increased mucosal blood flow, increased bicarbonate secretion, and increased mucosal restitution are mechanisms of misoprostol. Misoprostol (5 mcg/kg, PO) was shown to increase gastric juice pH and inhibited gastric acid secretion for 8 hours after administration. Misoprostol is contraindicated in pregnant and nursing horses due to its effect on increasing uterine contractions. Although no reports of side effects have been reported in horses, I have noticed some horses with mild colic signs 30 to 60 minutes after administration.

Prokinetic Agents

Prokinetic agents are a valuable adjunct therapy in horses with EGUS and when there is adynamic ileus and gastroduodenal reflux. Bethanechol (0.25 mg/kg, IV) and erythromycin lactobionate (0.1 and 1.0 mg/kg, IV) have been shown to increase solid phase gastric emptying.[37] No adverse effects were seen in healthy horses, however other forms of erythromycin can cause fatal colitis at antimicrobial doses. Both prokinetics increase gastric emptying versus saline, but bethanechol appeared to be superior in increasing solid-phase

gastric emptying, where as erythromycin increased liquid-phase gastric emptying. Bethanechol is a synthetic muscarinic cholinergic agent that is not degraded by acetylcholinesterases. The only side effect of the bethanechol administration was increased salivation. Other authors have recommended a dose of 0.025 – 0.030 mg/kg subcutaneously every 3-4 hours followed by oral maintenance therapy of 0.3 – 0.45 mg/kg 3-4 times daily. It is also possible that gastroduodenal reflux may worsen after treatment in patients with a proximal small intestinal obstruction.

Duration of Pharmacologic Treatment

It is difficult to predict how long a NG or glandular gastric ulcer will take to heal, but the initial recommended treatment time for most antiulcer medications is at least 28 days. However, management changes in addition to pharmacologic therapy can affect healing ulcers. For example, in horses with gastric ulcers induced by feed-deprivation, the ulcers were healed or nearly healed in horses after 9 days of pasture turnout,[31] whereas, omeprazole-treated Thoroughbred horses kept in training took longer to heal. Furthermore, pastured horses with spontaneous occurring ulcers showed 86% healing after 28 days of treatment.[30] We recommend endoscopic examination in horses with gastric ulcers after 14 days of omeprazole treatment *(Figure 1a and 1b)*. More than half of the gastric ulcers healed in 14 days during the FDA-Approval clinical trial.[28] This could save the client money and if the ulcers have healed, the dose could be reduced (1 mg/kg, PO, q24h) to prevent recurrence while the horse remains in race training. In addition, adding alfalfa hay to the diet might potentiate the effect of omeprazole on increasing gastric juice pH and facilitate healing. If healing has not occurred after 28 days of omeprazole treatment, then further workup may be indicated. It should be noted that clinical signs might resolve before complete ulcer healing has taken place. Ranitidine should be used a full 28 days or longer (>40 days) as it is not as effective in ulcer healing compared to omeprazole treatment.[29]

Environmental, Nutritional and Dietary Management

Pharmacologic therapy may be necessary to heal both NG and glandular gastric ulcers in horses. Once pharmacologic therapy is discontinued ulcers will return within several days if management changes are not instituted. Environmental, nutritional, and dietary management can be initiated during therapy to help facilitate ulcer healing and prevent ulcer recurrence. Adding alfalfa hay to the diet to buffer gastric acid can be helpful. Also, pasture turnout to facilitate continuous eating will help saliva production. Several treatment and preventative strategies for working horses are listed in Table 2.

Table 1. Drug Therapy for Treatment of EGUS

Drug	Dosage	Dosing Interval	Route of Administration
Omeprazole	1.0 mg/kg	Intravenously	Q 24 hrs
Omeprazole	4 mg/kg	Orally	Q 24 hrs
Omeprazole (prevention)	1 mg/kg	Orally	Q 24 hrs
Ranitidine	1.5 mg/kg	Intravenously	Q 6 hrs
Ranitidine	6.6 mg/kg	Orally	Q 8 hrs
Famotidine	0.3 mg/kg	Intravenously	Q 12 hrs
Famotidine	2.8 mg/kg	Orally	Q 12 hrs
Misoprostol	5 mcg/kg	Orally	Q 8 hrs
Sucralfate	20-40 mg/kg	Orally	Q 8 hrs
AlOH/MgOH antacids	30g AlOH/15 g MgOH	Orally	Q 2hrs
Bethanechol	0.025 – 0.30 mg/kg	Subcutaneous	Q 3-4 hrs
Bethanechol	0.3-0.45 mg/kg	Orally	Q 6-8 hrs
Erythromycin lactobionate	0.1 – 1.0 mg/kg	Intravenously	Undetermined

*Cimetidine: Not effective for treatment of EGUS

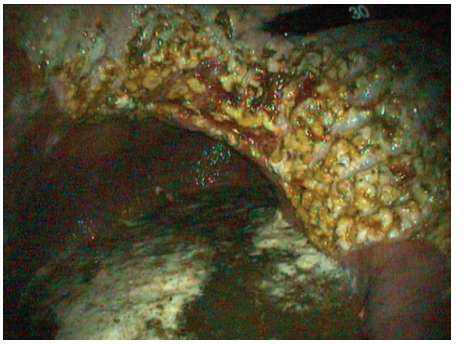

Figure 1a. Severe gastric ulcers (Grade 3/3) in the lesser curvature of a horse after stall confinement and intermittent bolus feeding.

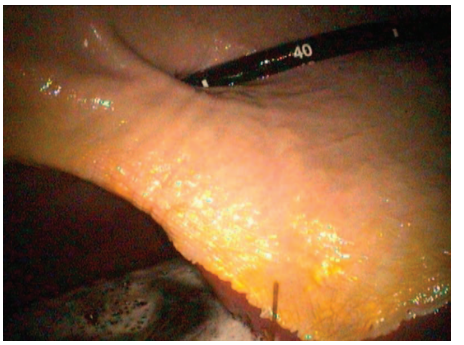

Figure 1b. The lesser curvature in the same stomach (Grade 0/3) after treatment (4 mg/kg, GastroGard, orally, Q24h, 14 days). There is mild hyperkeratosis still present, but no gastric ulcers.

Antibiotics vs. Probiotics

Helicobacter pylori and other *Helicobacter spp*. have not been shown to be a cause of EGUS, although *Helicobacter equorum*, a urease positive bacteria, was recently isolated from feces, and stomach mucosa of horses and foals.[20] Also, a large population of diverse acid-tolerant bacteria (*E. coli, Lactobacillus and Streptococcus spp*.) were isolated from the gastric contents of horses fed various diets.[38] In rats, which have a compound stomach similar to horses, bacteria (*E. Coli*) rapidly colonized acetic acid-induced stomach ulcers and impaired ulcer healing.[22] In that study, oral antibiotic treatment

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Table 2 – Treatment/Preventative Strategies

STRATEGY	Exercise Regimen	Grass hay vs Alfalfa Hay	Husbandry	Intermittent vs Free choice feeding	Pharmacotherapy
Strategy 1	Race training / Intensive training	Alfalfa	Stall confinement	Free Choice	Gastroguard 4mg/kg 28 days then 2/mg/kg maintenance
Strategy 2	Race training / Intensive training	Alfalfa	Stall confinement	Free Choice	Ranitidine 6.6 mg/kg PO TID for treatment and maintenance
Strategy 3	Race training / Intensive training	Alfalfa	Stall confinement	Free Choice	Antacid 30/15 g Al/Mg before exercise and in evening
Strategy 4	Moderate training, Show/performance horses	Alfalfa	Stall confinement with limited pasture turnout	Free choice when in stall	Same as 1 & 2, or treat with omeprazole or ranitidine 3 days before and throughout show
Strategy 5	Brood mare, trail horse, pleasure horse	Either	Maintained on pasture	On pasture	Not necessary except when diagnosed with ulcers. Treat for 14 days and reevaluate ulcers

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suppressed bacterial colonization of the ulcer and markedly accelerated ulcer healing. Also, trimethoprim suphadimidine or a probiotic (*Lactobacillus agilis, L. salivarius, L. equi, Streptococcus equinus, S. bovis*) administered orally to horses decreased the number and severity of ulcers compared to untreated controls.[39] These data suggest that the resident stomach bacteria are important in maintenance and progression of NG gastric ulcers in horses and treatment with antibiotic and/or probiotics might facilitate ulcer healing or prevent ulcers from recurring. Antibiotics and/or probiotics administration might be used in horses with chronic non-responsive gastric ulcers as an adjunct to therapy.

Seabuckthorn Berries and Pulp

Figure 2. Seabuckthorn Berries (*Hippophae rhamnoides*) growing in Tibet.

There is an increasing interest in the use of herbs and berries that have therapeutic application in man and animals. Berries and pulp from the seabuckthorn plant (SBT; Hippophae rhamnoides) are high in vitamins, trace minerals, amino acids, antioxidants and other bioactive substances (Figure 2). SBT berries and pulp have been used successfully to treat mucosal injury. In addition, SBT have been shown to successfully treat and prevent acetic acid-induced gastric ulcers in rats.[40] In a recent study, a SBT preparation (3 ounces fed twice daily; SeaBuck™ Complete, Seabuck LLC, Midvale, UT) administered to horses, prevented an increase in number and severity of gastric ulcers after feed-deprivation.[41] Although, SBT did not significantly (P=0.06) decrease NG gastric ulcer scores, compared to untreated controls, gastric ulcer scores in 7/8 SBT-treated horses either stayed the same or decreased compared to just 2/8 of the untreated controls. SBT may be used as an adjunct therapy.

Pectin, Lecithin and Antacid Supplements

There are many supplements on the market containing pectin, lecithin and antacid (calcium carbonate, sodium bicarbonate). The author recently evaluated a supplement containing pectin, lecithin and antacids (Egusin® SLH and 250; Centaur Corporation, Oakland Park, KS).[42] These products, when mixed with sweet feed (4 ounces, twice daily) and fed to horses, prevented NG gastric ulcers from increasing in response to feed deprivation, but required 35 days of feeding to see an effect, which is consistent with results from previous studies.[43]

The author (FMA) also performed a small study with an antacid preparation containing calcium carbonate (4 ounces, top-dressed on grain, q12h; Neigh-Lox®, Kentucky Performance Products, LLC, Versailles, KY). Nonglandular gastric ulcer scores were not significantly different when compared to controls after 3 weeks of feeding.[44] However, gastric juice pH remained ≥ 4 for 2 hours after feeding. Also, in an *in vitro* Ussing chamber study, this supplement when added to VFA-acid damaged NG mucosa resulted in recovery of sodium transport. These data suggest that calcium carbonate preparations may have some efficacy in maintaining mucosal integrity, but may need to be fed more frequently than twice daily to improve gastric ulcer scores.

Oils (corn oil, rice bran oil)

Ponies, with gastric cannulae, fed corn oil (45 ml, orally, once daily) by dose syringe had significantly lower gastric acid output and increased prostaglandin concentration in gastric juice.[45] Corn oil supplementation could be an economical approach to the therapeutic and prophylactic management of glandular ulcers in horses, especially those associated with the use of NSAIDs. In contrast to the previous study, corn oil, refined rice bran oil and crude rice bran oil (8 ounces, once daily, mixed in grain) showed no effect on NG ulcer scores after six weeks of feeding.[46] Thus, dietary oils might be an adjunct therapy in horses to prevent glandular ulcers.

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