

Stomach and Colonic Ulcers in the Equine

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Introduction:

It has not been so very many years ago that the term “stomach ulcers” was a relatively unknown term. Now stomach ulcers or Equine Gastric Ulcer Syndrome –EGUS (Andrews et al., 1999), perhaps rightly, has become the new “disease of the year.” Still much about the syndrome is unknown or unrecognized, so the true incidence and ramifications may be significantly higher than previously realized. There are few effective medical treatments available. In the allopathic realm, so much of treatment that is available is expensive. Diagnosis could, until recently, be confirmed only by endoscopic examination. This is a method that is expensive, invasive and even when skillfully applied, may miss ulcers in the distal esophagus, the stomach and proximal duodenum and cannot be used to evaluate the distal area of the duodenum, the small bowel or the large intestine. Diagnosis has to also be based on history, clinical signs, laboratory findings, as well as response to therapy. Current strategies focus on increasing pH to 4.0 or greater by buffering or by inhibiting the production of gastric acid as well as decreasing environmental stress.

As a strong advocate for the need of a more integrative medicine approach, the author encourages practitioners to appreciate and accommodate the best of allopathic medicine integrated with the best of beneficial complementary modalities and systems. To that end, this paper seeks to discuss the use of acupuncture point reactivity as a strong indicator of inflammatory and ulcerative pathology in the GI tract for both fore and hindgut ulcer problems. Acupuncture can also play a role in therapy and like any therapy must be combined with good management counseling.

Incidence of Gastric and Colonic Ulcers:

Gastric ulcers in various studies have shown 90 – 95% of racehorses are afflicted (Murray, 96 – 2000 and Vatisias 199). In studies of performance and show horses the percentages range from 30 to 70%, including dressage horses at about 40% (McClure et al 199, Mitchell 2001), endurance 65 -67 % (Nieto et al, 2004) and western performance 40% (Bertone, 2000). It is important to emphasize that these percentages do not reflect what is occurring in the rest of the GI tract. It is known that ulceration also occurs in the small intestine and the large bowel. In a retrospective necroscopic study of 545 horses, 44% of non-performance and 65% of performance horses had colonic ulcers (Right Dorsal Colitis – RDC).

Causal Factors:

Ulcers have to be considered an iatrogenic/human created problem. This author has always contended that stalls are one of the great injustices to which we have subjected the horse. Now we know that stall confinement alone can lead to ulcer development, sometimes in as little as 24 hours. Typically, accompanying stall confinement are twice daily feedings that often involve high grain rations. It is known that horses continue to secrete acid regardless of whether they are eating or not eating. The stomach secretes from 4 to 60 mmoles of hydrochloric acid per hour in the 1.5 liters of gastric juice produced hourly. Twice daily feeding means that there is a prolonged period of time in which there is no feed in the stomach to neutralize the acid. High grain intakes produce volatile fatty acids that contribute to ulceration. This will be discussed further.

Another key factor for ulcer formation is the frequent administration of non-steroidal anti-inflammatory agents (NSAIDs) such as phenylbutasone and flunixin meglumine (Banamine), as well as the steroid, dexamethasone, all decrease the production of a protective mucous layer in the glandular part of the stomach. NSAIDs play a large role in hindgut ulcers as well as in the glandular portion of the stomach.

Training intensity can be a major factor in ulcer development. The pH of the stomach is lowered with intense training or competition, as for example in endurance, race, or three day event training and competition. Unknown factors during exercise relate to gastric collapse and the “spilling” of acid on the non-glandular squamous portion of the stomach. There may also be significance to decrease motility and delayed gastric emptying in the face of ongoing acid production. Exercise intensity or related stress can also increase ACTH output thereby increasing endogenous steroid production.

The routine need for high levels of electrolyte supplementation in endurance and occasionally in event horses in the speed and endurance phases and electrolyte use during the marathon phase in combined driving may pose an added risk of ulcer formation. (Holcomb et al. AERC presentation 2007)

Signs and Symptoms:

Clinical signs vary with the location of the ulceration and the severity of the lesions. Signs that should alert the owner or clinician of EGUS or Colonic Ulcer Syndrome include:

- Behavioral changes such as resistance, “grouchy” aggressive, depressed or nervous.
- Do not like to be groomed or brushed along the ventral midline
- Slow eating – especially grain... may leave some feed.
- Moderate loss of weight (not more than 10% of body weight) even with increased grain, etc.
- Hair coat may be dull – especially over thorax
- Poor appetite
- Salivation or teeth grinding
- Intermittent low grade chronic diarrhea
- Mild to overt colic after eating or transportation stress

- Performance issues such as refusal to jump or want to jump “flat;” endurance horses don’t like to go downhill at speed (viscera mechanically pushing on stomach in both cases)

The pathophysiology in horses and foals stems from “disequilibrium between mucosal aggressive factors (hydrochloric acid, pepsin, bile acids, organic acids) and mucosal protective factors (mucous, bicarbonate)” (Frank Andrews). That is to say that there are likely differences in the ulcer inducing mechanisms in the glandular versus the squamous portions of the equine stomach. Those most prevalent in the nonglandular squamous area are principally due to prolonged exposure to hydrochloric acid, pepsin, bile acids or organic acids. The squamous area that is closest to the margo plicatus is most in contact with the acids and therefore commonly exhibits ulceration. Prolonged exposure to acids in this area is also linked, therefore with more severity.

Diagnostics:

It is considered that gastroscopy is the only accepted means of diagnosing EGUS. It should be noted that most insurance companies won't accept a diagnosis of EGUS unless it is verified by gastroscopy. This requires an endoscope that is approximately three meters in length in order to visualize the proximal duodenum as well as the entire glandular area of the stomach. Foals may be examined with a 2-meter scope. For adequate inspection, the stomach must, obviously, be empty. This requires a 12-hour fast and no water intake for two to three hours prior to endoscopy. Sedation is usually required. Endoscopy cannot, obviously, be used to diagnose hindgut ulceration.

There has been marketed by “Succeed” a useful, in field, occult fecal blood test that can be used as a means of diagnosing both fore and hind gut ulceration. (www.succeedfbt.com) It will not define the severity of ulceration that can be “scored” by number and severity of lesions with gastro-endoscopic examination. The principle of the test is as follows: The test stick contains two strips. They consist of:

Strip A: an antibody to albumin -

Albumin released from stomach will be digested in the proximal small intestine.

A positive fecal test for albumin mean large colon injury since albumen from the stomach (any GI structure proximal to the bile duct) would not make it past the bile duct enzymes in the small bowel

Strip B: an antibody to hemoglobin

Hemoglobin released from stomach will not be digested in small intestine. It is more resistant to the digestive so appearance in feces means bleeding from anywhere in GI tract and the blood may be coming from stomach bleeding.

An “A” strip negative with a B strip positive indicates foregut bleeding

An “A” strip showing a positive with a negative “B” strip is possible with low grade colonic ulcer or a protein losing enteropathy.

Other laboratory markers are non-specific but can still provide some useful data. Horses with gastric ulceration tend to have lower RBC counts and lower hemoglobin values (bottom range of “normal”) than those that do not have ulcers. Additionally, they may be slightly anemic or hypoproteinemic.

Acupuncture “reflex point” Evaluation for EGUS

For the past two years, the author has been tracking cases of EGUS by evaluating the reactivity of a number of acupuncture points on the thorax and abdomen. This is followed by treatment of “command points” on the limbs. The “diagnosis is considered positive if the previously tested points become non-reactive.

In addition to reactive acupuncture points it is very common for ulcer cases to also exhibit significant muscular and myofascial/neuromuscular issues. It is common to see a myofascial/neuromuscular pattern extending from the wither pocket to the thoracolumbar junction. If the examiner traces this “line” with firm finger tip pressure, the horse responds with strong muscular fasciculation and may act out. These same patterns also often occur over the croup muscles and over the cervical portion of the Trapezius muscle.

Myofascial contracture in the wither area often triggers contracture and hypertonicity of the Psoas group of muscles. These muscles are the key stabilizing muscles for the pelvis. Hypertonicity of the Psoas Major and minor as well as of the Iliopsoas muscle results in sacropelvic articulation dysfunction. The articular dysfunction sets up muscle tension and spasms in the muscles of the croup, such as the Middle gluteals, Superficial gluteals, Biceps femoris and sometimes the Semitendinosus and Semimembranosus muscles. Chiropractic issues also arise in the upper thoracic vertebrae as a result of the myofascial-neuromuscular tension.

It has been nothing short of amazing to see that after the acupuncture is performed as a diagnostic tool, *the “body work issues” quickly resolve, or at least, ameliorate without performing any direct or specific bodywork.* Further bodywork, chiropractic and acupuncture may, and certainly should, be performed to enhance resolution and improve the well being of the horse. It is also noteworthy that *chronic body pain is a stressor* and may be a factor in ulcer production. A cycle is created that feeds upon itself to worsen the condition.

The points used for “diagnosis” can also indicate whether there is inflammatory response in the hindgut and/or small intestine. Another key point worthy of emphasis is that *the discovery of these musculo/myofascial and “bodywork issues should create awareness that EGUS should be ruled out.*

Indicator Points for GI inflammatory conditions such as EGUS:

The author's preference is the use the tip of a needle cap to evaluate the level of reactivity of the points. The flat side of a needle cap is also used to apply pressure to ascertain muscle and myofascial sensitivity. The key is to apply a consistent pressure on all points tested. *With excess pressure any point will react.*

The first "indicator" is marked reactivity in the distal third of the Brachiocephalicus muscle. Along its ventral border lies the Stomach Meridian. A sharp fasciculation or spasm is found bilaterally when EGUS exists. If the response is unilateral there is more likely a chiropractic, or a muscle issue. If that area (region of ST 10 – 11) is found to be reactive, check BL 20 and BL 21, the Back Shu Points for Spleen and Stomach. If these are also reactive, proceed to LIV 13, the "alarm point" for the spleen. Also proceed to check the Stomach "alarm point" ST 12, as well as ST 14, and the Large Intestine "alarm point," ST 25. Also include evaluation of BL 25 and BL 27 – Back Shu points for the Large Intestine and the Small Intestine.

All points should show the approximately the same degree of reactivity *bilaterally*. If points are only unilaterally reactive, consider the "diagnosis" to be suspect and inconclusive. By evaluating points related to Small and Large Intestine, ulcers sites in those segments should be considered.

"Diagnosis" cannot stop with just the above information. The next step is to treat command points on the limbs to see if all points (and perhaps the afore mentioned body soreness issues) are ameliorated. Many of the cases with EGUS that the author has worked on are young horses in race training, and that entail concern for my safety and welfare. They are often not amenable to needle acupuncture of the limbs – especially medial aspects of the hind limbs. Fortunately, low-level laser or LED units (photonic therapy units) work very well and can be more safely applied than needles.

Points Treated For "Confirmation of a Tentative Diagnosis of EGUS:

For the sake of brevity, the action and reasons for the use of each point will not be covered in this paper. The author refers the reader to any good acupuncture text. The points listed are a "cookbook" formula and can certainly be modified and/or enhanced for specific cases – especially if acupuncture is chosen as a therapeutic modality.

Master Points for "diagnostic" treatment:

ST 36
SP 6
LI 4
PC6

Other useful points for "diagnostic" treatment

SP 4
GB 34
LIV 2
LI 11

If a good response is elicited, the case for acupuncture “diagnosis is enhanced. One can then decide to use conventional EGUS therapy to further prove the diagnosis and resolve the case (or resort to endoscopy for confirmation). Correlation with cases that have received endoscopy has been approximately 85% accurate. If it is feasible to see the patient on a regular basis for follow-up acupuncture (for four to six treatments) good results can be achieved. *Often the client can be taught to monitor the indicator points. Thereby, they can monitor progress during ongoing therapy or be aware of the recurrence of ulcers.* For initial healing, therapeutic doses of brand name omeprazole (GastroGard or UlcerGard) can be very helpful.

No treatment can be successful, long term, without addressing the environmental and human induced causes. Those involved in holistic forms of medicine certainly recognize the need to treat the “root.”

Conventional Therapy:

The goals are to remove or ameliorate the predisposing factors and decrease acid production and/or neutralize the acids. It is also desirable to have medications that minimize the frequency of dosage to ensure more likely client compliance. The current mainstay of therapy is omeprazole. The only FDA approved drug at this time is GastroGard or UlcerGard by Merial LTD. It is an acid/proton pump that can cut acid production by as much as 90 percent for up to 27 hours when administered once daily. Omeprazole is difficult to compound while maintaining potency. Consequentially, questionable efficacy has made generics a poor choice. Potency has run around 30 percent in most that have been tested. Though some tested have shown about 70 percent potency, the generics are a “caveat emptor (Nieto et al, 2002, Orinsi et al, 2003).

Other products may have value in prevention or maintenance regimens. These include the H-2 antagonists (histamine inhibitors, such as *Ranitidine (Zantac)* at a dose of 6.6 mg/kg every 8 hours or 11.0 mg/kg every 12 hours. Another H2 antagonist is “*Cimetidine*” (Tagamet). In tests, Cimetidine has performed poorly. H-2 antagonists act to suppress acid secretion without completely blocking acid production. It has been reported that *Sucralfate* is more effective when used with HE antagonists (Hinchcliff et al). Coating agents, such as Bismuths (*Pepto Bismol*) have shown no significant value in therapy (Picavet, 2002).

Sucralfate is a coating agent that forms a protective gel type of coating over ulcers and although it may not have a direct healing action allow the body to heal the ulcers. *Sucralfate* requires dosing every 6 to 8 hours. It may have particular value in treating hindgut ulcers occurring in the right dorsal colon (RDC syndrome).

Another proprietary compound that the author has had good success with, especially as maintenance level therapy is “*Ulc-Rid*.” It is an all-natural product containing herbs and probiotics (sold by Equine Nutritional Consultants LLC in Versailles KY - <equineconsultant@earthlink.net>. It needs to be administered bid and is a liquid formulation that is best given as a drench, though clients do report success with “top

dressing.” Other “stomach soothing” agents have been favored by competitors in eventing, endurance and show. Such products include papaya (Natural Plan Stomach Soother – Healthmate Products, Inc. www.stomachsoother.com), or aloe-vera juice. This group of products may best be utilized for prevention during transportation and competition.

Another product of interest is a product called “Pronutrin” marketed by Boehringer Ingelheim. Though it is not yet marketed in the USA it shows promise. It is a pectin-lecithin complex. Lecithin forms a highly hydrophobic protective layer over the ulcerated area and thus reinforcing acid-repelling properties of phospholipids of the squamous mucosa (Lichtenberger 1996). The other component, Pectin, when in the presence of low pH, turns into a gel that can bind the bile acids produced in the glandular stomach and duodenum. Pectin also helps stabilize the protective mucus and increase the buffering capacity of the stomach contents. This results in a higher pH post prandially.

It is important to note that the lecithin used is stabilized with a hydrophilic polymer to prevent its breakdown into Lyso-lecithin. Lyso-lecithin is harmful to mucosal cells. *For this reason pure lecithin is contraindicated in therapy of GI ulceration.*

The product is currently available in Europe and South Africa. It is a pelleted feed that can be fed as a “top-dressing.” Dosage is 50 mg/100 kg of body weight divided into a bid dose. It should be fed for 4 to 6 weeks. It is likely best used as a preventative or a maintenance product after a seven to 10 day period of an omeprazole product.

It is also noteworthy in studies where alfalfa is fed instead of grass hays that there is a lower incidence of gastric ulcers. This may be related to the higher calcium and protein levels (Robinson 2003). Alfalfa has been shown to buffer gastric contents. A partial ration of alfalfa and grass hay may be beneficial. Changes should, of course be made gradually.

Right Dorsal Colitis (Colonic Ulcer Syndrome)

The significance of Ulcerative colitis has been alluded to in the early paragraphs of this paper. It perhaps occurs less frequently but can have more severe clinical signs. Colonic ulcers are probably associated with PG-2 (Prostaglandin) inhibition by the administration of NSAID drugs or by the stress related production of endogenous corticosteroids. Signs can include mild or intermittent colic, lethargy, poor appetite. In more severe forms complete anorexia, fever and diarrhea may be seen accompanied by dehydration, ventral edema and weight loss.

Diagnosis is difficult. Negative gastroscopy, in the face of ulcer signs and behavior, may lead one to a presumptive diagnosis of colonic ulcers. The occult fecal blood test marketed by “Succeed” may prove to have value. Ultrasound may demonstrate mural thickening of the right dorsal colon. Acupuncture “indicator points” also may be helpful in arriving at a presumptive diagnosis of Ulcerative Colitis.

Treatment requires cessation of the administration of NSAIDS. It is also helpful to offer small but frequent feedings and reduce gut fill to allow the colon to rest. Decreasing or elimination of dry hay and substituting Purina Senior. – The product is a pelleted (alfalfa based) complete feed. It provides 30 percent fiber and can work well to reduce gut fill. Small frequent feedings are indicated. Grazing for 15-minute intervals four times a day may help maintain weight. A switch to a complete feed should be made over a weeks time to allow the gut to accommodate the change. The complete feed program may need to be continued for three or four weeks.

Products such as *Sucralfate* at a dose rate of 22 mg/kg every 6 to 8 hours to form a proteinaceous bandage may be very helpful. Once bound to the ulcer crater they may stimulate local prostaglandin production and that action may exert a cytoprotective effect on the colon mucosa. (F. Andrews, Proceedings of the Kentucky Equine Research Conference, 2008)

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Conventional Diagnostic and Treatment Flow Chart – Courtesy of Dr. Drank Andrews

