Differential Diagnosis and Treatment of Gastric vs. Colonic Ulcers
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Ulcers or Syndrome?

Veterinarians by now are quite familiar with gastric ulcers in horses. Studies have shown that 90% of racehorses and almost 60% of performance horses have stomach ulcers (McKenzie 2003). Gastric ulcers can be visualized with a three-meter endoscope, but lacking that, most veterinarians resort to symptomatology. Symptoms associated with ulcers include poor performance, inappetance, attitude changes. This grouping of symptoms defines Equine Gastric Ulcer Syndrome, commonly called EGUS (Cohen 2003).

Good medical practice encourages us to concentrate not just on the symptoms, but rather on the disease state(s) leading up to them. For gastric ulcers, that disease state is called gastritis or sometimes gastroenteritis, when the intestine is also involved.

Our 2003 study of 180 performance horses at a Texas abattoir showed us that as well as gastric ulcers, horses are afflicted with colonic ulcers (Pellegrini 2005). We found 87% of the horses had gastric ulcers and 63% had colonic ulcers. The overlap of 54% indicates that over half of the horses necropsied had both colonic and gastric ulcers.

In keeping with our discussion of gastric ulcers and reflecting the large co-occurrence of the two ulcer types, we propose the nomenclature Equine Colonic Ulcer Syndrome (ECUS), whose underlying disease is colitis.

EGUS (Gastritis)

EGUS is a well-known issue in the equine world, but despite years of study, there remain many uncertainties. Do all the lesions have clinical importance and significance? What is the etiology of any given ulcer? Several theories have been put forward to describe possible causative agents, including NSAIDs (anti-prostaglandins for pain), exercise and performance (sloshing stomach acids) (Merritt 2003), high cortisol levels (stress from trailering, stalling, performing etc.), stomach hyperacidity (intermittent feeding), hard feed (excessive carbohydrates), bacteria (Helicobacter Equorum) and parasites (bots).

Despite the reasonable nature of these possible causes, it is still difficult to establish which set of these is affecting any particular horse. Nevertheless, EGUS is widely and routinely suspected in performance horses.
(Cohen 2003) (Durham and White 2008), and some practitioners feel it may be over-diagnosed and over-treated, e.g. in the case of poorly performing horses with an uncertain diagnosis.

**Colonic Ulcer Syndrome (Colitis)**

In contrast to EGUS, colonic ulcer syndrome is little-known in the field, despite nine years of accumulating evidence. Due to the difficulty of visualization, the main data have come from necropsies. As a consequence, many veterinarians are not familiar with colonic ulcers or the colitis that may give rise to them often without any specific symptoms.

The exception is right dorsal colitis (RDC) that is known to be associated with NSAID administration (Cohen 2003). However, our studies have shown that ulcers can exist in quadrants other than the right dorsal region, and include several different types.

Some of the possible causes are the same as for EGUS, although the bacteria and the parasites in the hindgut are different from the foregut. These similarities reinforce the strong overlap between the two ulcer types.

**Bacteriology**

From 111 horses in a Canadian abattoir in 2003, we were able to collect 159 tissue samples for histopathology and luminal swabs for culturing, from both normal and ulcerated tissue.

The ecology of equine intestinal bacteria is largely unknown, but our studies have shown that dozens of species exist in each horse and can be cultured or detected histopathologically. We tested for 55 different strains of bacteria. Some of the most prevalent bacteria include:

In this chart, abundance is computed from growth of the cultured bacteria according to a scale from 0 to 3:

1. none detected
2. light colony growth
3. moderate growth
4. abundant growth

This numbering system should be understood as a rough approximation, as "abundant growth" is not exactly three times greater than "light growth". Nevertheless such a system allows us to make numerical comparisons that are useful and otherwise unobtainable. The growth was averaged over both ulcerated and normal tissue and shows that E. coli and enterococcus are the most abundant of the bacteria we tested for, more than double the abundance of any of the other species.

We then looked at differences between ulcerated and normal tissue. To see which species were most associated with one condition or the other, we subtracted the averages of ulcerated abundance from normal abundance and took the absolute values of those differences to show which species changed the most in moving from a normal to an ulcerated condition. Here are the associations we found, expressed as a percentage change over the total range (0-3):

Note that E. coli, the most abundant species, is strongly associated with normal tissue, with other species seemingly less so. For ulcerated tissue, we found the following associations:
Note that Streptococcus viridans, which shows the greatest change from normal to ulcerated tissue, is eighth on the overall abundance list. Thus, some of the biggest changes we noted in ulcerated tissue were associated with fairly underrepresented species, perhaps due to enhanced toxicity.

It must be stressed that these associations are not known to be causal. At this point, it is not known if the bacteria are implicated in the development of ulcers or merely opportunistic.

**Differential Diagnosis (using SOAP)**

Differentiating foregut versus hindgut lesions or recognizing their coexistence allows for a more targeted treatment (Cohen 2003). Not only are these two very different environments in terms of flora, pH, enzymes and mechanics, but they may have very different etiologies and thus different treatments.

Every day we use the mnemonic of SOAP to guide us in our diagnostic effort. SOAP has four components: Subjective, Objective, Assessment and Plan. We take each in turn:

**Subjective**

We start with subjective clinical signs and/or reported anamnesis, including poor body condition, partial anorexia/poor appetite, girthiness, attitude changes, poor performance/poor training (Durham and White 2008) and altered fecal consistency (diarrhea).

These signs are often the first clue that gastritis or colitis may be impacting the health and performance of the horse (Merritt 2003), but they are frustratingly non-differential.

**Objective**

We can then list the objective observations, using strictly measurable data after TPR (temperature, pulse and respiration rate) using three-meter endoscopy, rapid antibody tests (lateral flow immunoassay) and pH meters.

Note that there are fewer objective measures available to the veterinarian. Three-meter endoscopes are expensive and require extensive training to master. Unfortunately, endoscopes are not useful in visualizing colonic lesions, as horses can't tolerate the total evacuation of the hindgut. Rapid antibody tests are just recently becoming available, and they promise to distinguish foregut and hindgut lesions using albumin as a proxy for hindgut lesions (foregut bleeding produces albumin also, but it is digested by stomach acids and enzymes). In our work we have also used pH meters to help recognize hindgut acidity, commonly thought to result from excess carbohydrates reaching the colon. Although highly variable, pH can be a useful adjunct to other measures. Further tests may involve CBC and chemistry, fecal egg count, etc.

**Assessment**

Based on the subjective and objective observations, the veterinarian can make an assessment, including differential diagnoses. For GI problems, the differential diagnosis is between lesions in the foregut, hindgut or both. Foregut issues include EGUS – stomach ulcers, gastritis, neoplasia, parasitism and anterior enteritis. Hindgut issues include colitis (Colonic ulcers), intestinal traumas (rectal tears, traumatic events), parasitism, neoplasia and intestinal inflammatory disease.

The differential diagnosis can be problematic; clinical signs of colitis (colonic ulcers) and EGUS can overlap and these two conditions can also occur concomitantly (Cohen 2003). Our studies have shown that 60% of gastric ulcers are accompanied by colonic ulceration (colitis).

The etiology of these diagnoses can be broken down into infectious and non-infectious. The infectious agents include salmonella, clostridium, lawsonia, streptococcus and other infectious agents (see bacteria charts). Non infectious causes can include eosinophilia, lymphocytosis, plasmacytosis, and inflammatory bowel disease. Note that these non-infectious causes may be a response to the aforementioned infectious agents or parasites.

**Plan**
Further investigation may be needed to refine the differential diagnosis, but if a clear distinction can be made, there are two possible treatment plans, depending on whether a foregut or hindgut issue is suspected. In the following sections, we look at possible treatments for both.

EGUS Treatment

Pharmaceutical
Typically, and especially when fast results are desired, the treatment will be pharmaceutical. Among the drugs available for foregut treatment include proton pump inhibitors (omeprazole) such as GastroGard® and UlcerGard® from Merial, H2 Antagonists (All off-label, with uncertain absorption) such as Cimetidine, Ranitidine and Famotidine, and Fenbendazole – Weak PPI (anthelmentic) and mucosal protectants, such as sucralfate.

Husbandry
Longer term, and especially for preventive purposes, better husbandry is recommended. This includes turn out, multiple feedings (4 daily, at least) and dietary modification such as adding alfalfa and reducing hard feed.

Over-the-counter (OTC) Products
In addition to husbandry and pharmaceuticals, there are alternatives in the form of nutraceuticals and natural remedies. These include Antacids (temporarily palliative for 30 to 90 minutes) such as Neigh-Lox®, Mylanta® Maximum Strength and Maalox®. Herbals (richer in anecdote than science, may exacerbate lesions) include devil’s claw, licorice and mint.

Colonic/Colitis Treatment

For hindgut problems, the treatment is usually quite different and varies according to etiology and diagnosis. Unfortunately, the etiology of colonic ulceration is poorly understood. However, it is possible to list some common diagnostic differentiators, such as symptomatic vs. asymptomatic, infectious vs. non-infectious, febrile vs. afebrile, immune-mediated, neoplastic and parasitic.

Pharmaceutical
As with EGUS, there are pharmaceuticals, but fewer of them, including antibiotics, immunomodulators, anti-inflammatory agents, steroids, non-steroidal anti-inflammatory (but not for RDC) and mucosal protectants such as sucralfate.

Husbandry
Good husbandry can help to minimize hindgut issues. The guidelines are similar to the guidelines for EGUS, namely turnout, multiple feedings and dietary modification.

OTC Products
There are few products on the OTC market that are designed specifically to target the hindgut. As more veterinarians become familiar with Colonic Ulcer Syndrome, we expect new products to fill this niche. Among them are probiotics and prebiotics as well as natural supplements or nutraceuticals including psyllium, Succeed® DCP from Freedom Health LLC, Equine GI Support™ from MD’s Choice Nutritional Products and Bio-Sponge from Platinum Performance, Inc..

Conclusions

Equine ulcers are endpoints of a disease stage, typically gastritis or colitis (colonic ulcers) (Andrews 2009). These lesions and ulcers represent a syndrome that leads to poor performance and impaired health. Although EGUS is well-documented, the existence of equine colonic ulcers is only beginning to be appreciated. Colitis is a syndrome of its own, producing mild to severe ulcers and presenting multiple symptoms. In our view colitis is vastly under-diagnosed (unless showing a very acute course), while EGUS is possibly over-diagnosed and over-treated. Techniques to better differentiate a GI diagnosis are essential for proper treatment of these two very different ailments. One way is to test feces for the presence of albumin and hemoglobin, which helps to distinguish the two sources of GI bleeding. Further research is needed to understand more about the unique etiology and pathology of colitis. We hope that our research will lead to improved diagnostics, treatments, palliatives and preventives.
References


