ULCERS

The Equine Stomach

by Madalyn Ward, DVM

I was able to attend a fantastic lecture on the equine stomach at the 2003 AAEP convention. A. M. Merritt, DVM, MS shared the latest research on this topic. Here are just a few of the things I discovered. This information is particularly valuable in understanding why the horse is so prone to gastric ulcers and why response to drug therapy can be variable.

Feedstuffs tend to layer in the equine stomach and the layers vary in their pH balance, 7 is neutral, with 1 being most acid and 14 being most alkaline. Coarser materials form a mat in the upper portion of the stomach and mix with saliva to keep the pH in that region around 6 to 7. Medium-density feedstuffs are located in the middle zone of the stomach, and this area has a pH of 4 to 5. High density fluids are in the lower portion of the stomach and the pH is 1 to 2.

The lower portion of the stomach is the glandular portion which is protected from acid by a mucous coating. The upper non-glandular or squamous portion of the stomach is not intended to be exposed to stomach acid. Hydrochloric acid is secreted continually by the parietal cells in the gastric glands which are located in the lower portion of the stomach. The discovery of this layering effect helps explain why exercise tends to increase the frequency of gastric ulcer formation in horses. Monitors implanted in working horses revealed the contractions of abdominal muscles forced the lower pH liquids from the lower portions of the stomach up through the coarser feedstuff layers, exposing the non-glandular portion to the corrosive acids. Even abdominal muscle contraction secondary to anxiety caused the same reaction.

Acid secretion is up or down, and is regulated by food intake. The stomach acts as its own pH meter. It is worth going into some detail as to how this process works so you can understand how different drugs affect stomach acid in the equine. Histamine is a major player in the release of HCL by the parietal cells. By blocking the H2 (histamine 2) receptors, release of stomach acid can be suppressed.

Within the pyloric gland mucosa, unique cell types which are located very close to each other constantly "test" the pH of the stomach and react in such a way as to keep the pH in the correct range.

Antihistamines which block H2 receptors are able to block, at least in part, the release of HCL. Examples of these types of drugs include Cimetidine and Ranitadine. For years these drugs were the mainstay for ulcer treatment in horses. Unfortunately, they were inconsistent in effectiveness because the parietal cells are so sensitive to histamine that frequent large doses were required to control acid secretion. The parietal cells were also able to respond directly to stimulation of the vagus nerve.

The current drug of choice for ulcers in horses is GastrogardT (omeprazole). This drug is a proton pump inhibitor, which means it acts to block acid secretion after all possible stimulators have acted. Like other anti-ulcer drugs, GastrogardT does not address the underlying cause of equine ulcers. This drug can be very effective in treating ulcers but symptoms will often return when treatment is discontinued.

A holistic approach to treating and preventing equine ulcers would include keeping stress to a minimum so horses would not constantly be contracting their abdominal muscles. Feeding free choice hay helps to keep the fibrous mat layer intact in the stomach. Natural products such as aloe vera gel and the herb, slippery elm, can help protect the stomach tissue by forming a gel-like barrier. Feeding a high fat, low carbohydrate feed such as Equine seniorT is much better than high grain diets. Grain is digested in the stomach and increases the production of acid. If one assumes that exercise is going to cause some contact of the acid with sensitive squamous cells, then it make sense to provide nutritional support which maximizes the healing of tissues. Simplexity's blue green algae is one product which gives the body natural vitamins, minerals and antioxidants to support healthy tissues. http://www.holistichorsekeeping.com/algae.html Another good product to support health and healing is Noni Juice. This product contains natural enzymes which are needed in larger amounts when the body is under stress. www.tahitiannoni.com/rodeo I feel all performance horses benefit from these natural products which support health. Based on an understanding of how the equine stomach works, some degree of pathology is almost inevitable in our working horses. Why wait for symptoms to develop?

Ulcers

by Madalyn Ward, DVM

Much of the attention on treating ulcers has been focused on a new and expensive drug to treat ulcers. While I am not an advocate of treating ulcers with drugs, I am excited to see more focus put on this common condition. As many of my clients know, I put a huge emphasis on the digestive tract for a wide variety of complaints.

Many recent magazine articles are discussing specifically ulceration of the equine stomach. These ulcers can be easily visualized with the aid of a two-meter long flexible endoscope. I would like to suggest that ulcers or erosions are just as likely to occur throughout the intestinal tract. Breakdowns in the intestinal lining leads to a serious condition known as leaky gut syndrome. More on that later.

Gastric Ulcers

The lining of the horse's stomach is made up of a glandular and non-glandular (squamous) portion. The glandular portion covers the lower two-thirds while the squamous mucosa covers the upper one third.

Most ulcer problems will develop in the non-glandular lining because it has little protection against the acid which is produced in the lower portion of the stomach. The exceptions to this are the ulcers caused by toxicity from non-steroidal anti-inflammatory

drugs such as ButeT and BanamineT. These ulcers occur in the glandular portion of the stomach because NSAIDs block the release of prostaglandin. Prostaglandin is one of the agents the body depends on to protect the glandular lining of the stomach from the hydrochloric acid and pepsin that it secretes.

Clinical Signs

Both adult horses and foals are susceptible to gastric ulceration. In the adult horse, clinical signs include diminished appetite, colic, frequent pawing, weight loss and poor performance. A classic sign is a horse who eats the grain portion of his diet slowly if at all, but eats hay readily. Horses with gastric ulcers will often be back sore since the acupuncture association point for the stomach is in the muscle adjacent to the spine at the level of the thoraco lumbar junction. Clinical signs in foals include colic, pot bellied appearance, teeth grinding, and excessive salivation.

Causes

Pastured, grazing horses rarely develop gastric ulcers, however when these same horses are brought into stalls and intermittently fasted they develop ulcers with in five to seven days. Thoroughbred racehorses in training appear to be the most at-risk population, which has led researchers to conclude strenuous exercise may cause a temporary decrease in blood supply to the stomach. Another possible explanation for the increased incidence in racehorses is management. Most racehorses in training stand in a twelve by twelve stall for twenty-three hours a day with little to no contact with other horses. It is also common practice to feed racehorses large portions of grain and a minimum amount of hay. Even if hay is offered free choice, horses will eat less of it if the grain portion of the diet is meeting their caloric needs. It is also common practice to give racehorses frequent doses of NSAIDs both for injury and "prophylactically". All these factors alone or combined may account for the increased incidence of gastric ulcers in racehorses.

Researchers rule out nervous stress as a cause for ulcers, but I disagree. My experience has been that I see more ulcers in horses who do not like their jobs or are challenged beyond their capabilities. Often horses are not overfaced physically but mentally, especially where showing or competing is involved. On the opposite side, boredom can create stress in a type A horse who likes to compete. Physiological stress definitely contributes to ulcer formation. Injury or any kind of chronic disease, especially if pain is involved, sets a horse up for ulcers.

Treatment

Conventional treatment for gastric ulcers involves neutralizing the normal stomach acid or blocking its production. I have several problems with this approach. First of all, the problem is not an excess production of acid. Horses produce a small amount of acid continually because they are designed to eat continually. So blocking acid production is a Band-Aid solution when what we really need to do is reevaluate management. Secondly, we are doing the horse a huge disservice by artificially lowering the pH in his digestive tract. The higher pH is harmful to the natural bacterial flora and allows for the overgrowth of pathogenic organisms. This sets the horse up for dysbiosis or leaky gut syndrome. Also digestion is impaired if acid is not present in adequate amounts to breakdown the food. This allows undigested starches to be dumped into the large

intestine which has a bacterial population designed for fiber digestion. Ulcers in humans have been linked to the overgrowth of the pathogenic bacteria helicobactor pylori, but so far no specific bacteria have been linked to the formation of ulcers in horses. Instead of chemical protective agents, I use two ounces of an aloe vera gel drink two to three times a day mixed with two teaspoons of ground slippery elm bark. I mix these together in a 60cc syringe and give orally five to ten minutes before feeding. The aloe vera aids in healing of the stomach lining and the slippery elm is a mucilaginous herb which protects and soothes the stomach lining allowing it to heal. I also give SimplexityT blue green algae at the rate of one to three tablespoons, a day to provide additional nutrition and help heal the ulcers.

Prevention

Recognize the horses need to eat continuously. Provide hay free choice and use lowernutrition grass hay if your horse is an easy keeper. If grain must be fed in large amounts, divide feedings so that no more than three pounds is given at any one time. Match your horse with a job he enjoys and is well suited for. Provide as much exercise and entertainment as possible. Provide Probiotics on a daily basis if your horse is in training or confined due to injury or illness. Avoid frequent or long term use of non-steroidal antiinflammatory agents.

The Latest on Ulcers

by Madalyn Ward, DVM

At the 2006 Holistic Veterinary Conference, Dr. Scott Carter gave a lecture on gastric and colonic ulcers. So much of the research has focused on gastric ulcers and their treatment but Dr. Carter also shared research on colonic ulcers. A study of the postmortem findings in 500 horses revealed that 63% of performance horses had colonic ulcers.

We have come to understand many of the causes of gastric ulcers, such as infrequent feedings, high starch diets, and exercise causing the acid in the lower protected portion of the stomach to move into the upper, non protected portion. We also know the connection of non-steroidal anti-inflammatory drugs which decrease prostaglandin production and therefore decrease the production of protective mucous to line the intestinal walls. For gastric ulcers, treatments have been mostly focused on buffering the stomach acid or decreasing its production. Dr. Carter's research indicates that while these treatments have been somewhat effective in treating and preventing stomach ulcers they have actually increased the imbalance in the rest of the digestive tract and in many cases been responsible for increasing the presence of colonic ulcers.

To understand what is happening we need to review the function of the stomach and small intestine in the digestive process. The glandular portion of the stomach in the horse produces acid on a continuous basis. It also produces digestive enzymes and mucous

which protects it from the actions of the acid and digestive enzymes. The equine stomach is small and food does not normally stay in the stomach long before it is passed to the small intestine. The small intestine is also relatively short and food moves quickly to the large intestine where fermentation of fiber occurs.

The digestion of starch occurs in the stomach and small intestine by the actions of stomach acid and digestive enzymes produced in the stomach, and pancreatic enzymes released into the small intestine. Bacteria in the small intestine also contribute to the correct breakdown of starch. All of this starch digestion is highly dependant on the correct pH. The problem with the current treatments for ulcers is that the focus is on decreasing or buffering stomach acid which raises the pH in the stomach and small intestine. When the pH is raised to above four the digestive enzymes stop functioning and the population of healthy starch digesting bacteria in the small intestine decreases. Now undigested starch is moving into the small intestine with few beneficial bacteria. There is always a small number of harmful bacteria lurking about, without healthy bacteria to keep them in check and a higher pH environment, they thrive and they begin to ferment rather than digest the starch. The small intestine is not designed for fermentation and the harmful substances produced damage the intestinal lining producing ulcerations. The toxins produced by fermentation of starches greatly stress the liver when they are picked up by the blood vessels going from the intestines to the liver. Some of these toxins also find their way into the bloodstream directly through damage to the intestinal lining.

Now we have the remaining starch that was not digested or fermented being dumped into the cecum portion of the large intestine. The cecum is designed for the fermentation of fiber, not starch, and it also depends on pH for proper function. The lactic acid produced by the starch fermentation lowers the pH in the large intestine which kills the beneficial fiber fermenting bacteria and protozoa. Continued production of lactic acid and other harmful substances effectively shuts down fiber digestion and causes ulcerations and damage to the intestinal lining of the cecum and colon.

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