Gastric Ulcers- more common than you think...

This condition has been studied intensely over the past few years and is now more commonly referred to as Equine Gastric Ulcer Syndrome or EGUS.

Two syndromes are present, silent and clinical ulcers. Silent ulcers may occur in horses that do not exhibit any clinical signs, however when examining the stomach with an endoscope there are ulcers present.

Clinical signs:

- Grinding of teeth
- Excessive salivation
- Acute colic
- Recurrent colic
- Mild colic
- Dullness
- Decreased water intake
- Poor body condition
- Inability to gain weight/weight loss
- Poor appetite/slow eating
- Poor performance
- Attitude changes
- Anxiety
- Depression
- Temperature changes
- Resentment when tightening girth

Clinical signs are variable and vague; still gastric ulcers are a large problem in sport horses.

Gastric ulcers occur in 90% of sport horses and range from mild to severe forms. The lesions are scored according to the depth and which mucosal lining is involved.

The stomach consists of two areas with different linings. The upper compartment is lined with non-glandular squamous mucosa and the lower compartment is lined with glandular epithelium. The border between these two areas is known as the margo plicatus.

80% of gastric ulcers occur in the upper compartment lined with non-glandular mucosa. The glandular part is the site of gastric fluid production. These fluids include acids and enzymes: hydrogen chloride, pepsinogen and protective fluids such as mucus and bicarbonate.
The adult horse produces about 1.5 litres of stomach fluid per hour, even in the fasting state. This lowers the stomach pH and predisposes to gastric ulcers.

The stomach of a horse takes 30 minutes to empty after a liquid meal and up to 24 hours for a hay-meal. Horses thus need constant roughage to help buffer the acid in the stomach.

**Risk factors in adult horses which may lead to the development of ulcers include:**

- **Intense exercise**
- **Intermittent feeding**
- **Diet**
- **Transport stress**
- **Stall confinement**
- **Nonsteroidal anti-inflammatory drugs**

**Pathogenesis: (How and Why)**
Peptic lesions are produced when hydrochloric acid (HCl) and pepsin secreted by the stomach damage the mucosa of the stomach.

The predominant factor in peptic injury is HCl and the variable degree of susceptibility to HCl injury. This is due to the fact these areas are lined with stratified squamous epithelium which has minimal resistance to peptic injury. The equine glandular epithelium has evolved elaborate mechanisms to protect itself from damage by secreting mucus and bicarbonate.

**Diagnosis**
Presumptive diagnosis is based on clinical syndromes and recognition of clinical signs.

Definitive diagnosis is based on endoscopic exam. This procedure can only be carried out in a specialist facility with a 3m endoscope. There is only one of these scopes available in Cape Town. The procedure involves the horse being starved for a minimum of 6 hours and sedated. A 3m endoscope is then passed from the nostril into the stomach. The stomach is insufflated with air and a water jet is used to clear off feed material adhering to the wall. The stomach wall is then examined for signs of ulcers.

Other diagnostics which may indicate the presence or severity of ulcers include fecal occult blood tests, contrast radiography, abdominal ultrasound and abdominocentesis.

The fecal occult blood test is now available in SA and is a cheaper alternative to endoscopy. The test can be performed at the farm and all that is needed is a faecal sample from the affected horse. The test is called SUCCEED® Equine Fecal Blood Test™ and it is quick and easy to use.

**Treatment:**
Many treatments are available but only a few are proven to be effective in horses.
**Antacids**
Short-lived effect on gastric pH in horses. Difficult to administer antacid in an adequate dose frequently enough to anticipate a prolonged clinical effect which will positively effect ulcer healing.

There is insufficient data available on the affects of antacids in horses.

**Bismuth compounds**
These compounds have little substantial acid-neutralising capacity. Their beneficial effects have been ascribed to gastric cell protection (enhanced secretion of mucus and bicarbonate, inhibition of pepsin activity, and accumulation of bismuth subcitrate preferentially in the craters of gastric ulcers). Although these compounds are often used in the treatment of equine diarrhoea, there is little information about their effects on equine gastric ulcers.

**Sucralfate**
Sucralfate is a hydroxy aluminium salt of sucrose octasulphate. At a pH<4 this complex forms a sticky viscid gel. This gel adheres to epithelial cells and, with more affinity, to the base of ulcer craters. Once the gel is established and adhered to the ulcer crater it is difficult to wash out. This binding to ulcer craters is thought to represent the main therapeutic action. Sucralfate needs to be administered at 20-40mg/kg every 8 hours.

There is a scarcity of data available on its use in horses. Based on the lack of efficacy on stratified squamous ulcers, sucralfate is not recommended as sole therapy in the treatment of EGUS involving only the non-glandular portion of the stomach. The effect of sucralfate on duodenal or glandular gastric ulcers in horses has not been studied.

**Histamine H2 receptor antagonists**
Histamine H2 receptor antagonists were the first drugs reported to be used in the treatment of EGUS. Their primary effect is a dose dependent inhibition of gastric acid secretion. These compounds are highly selective with little or no effect on histamine H1 receptors. Commonly available H2 receptor antagonists include cimetidine, ranitidine, nizatidine and famotidine. Ranitidine must be administered at 8 hour intervals to be effective.

The potency of these is significantly different. Although there is ample evidence that the histamine H2 receptor antagonists have a significant effect on intragastric pH in horses and foals, scientific evidence that these compounds enhance ulcer healing in horses is lacking.

**Proton pump inhibitors**
These agents are potent and highly specific inhibitors of gastric acid secretion and possess a novel mechanism of action: the inhibition of hydrogen-potassium adenosine triphosphatase
(H+, K+-ATPase). This enzyme is the hydrogen ion pump in gastric parietal cells and is believed to be the terminal step in the acid secretory pathway.

Omeprazole and lansoprazole are the currently available proton pump inhibitors.

Omeprazole, the first to be developed, has been shown to decrease gastric acid secretion in horses and lots of other species. It has a prolonged antisecretory effect, allowing for once-daily dosing and has been shown to have significantly greater effect than placebo on healing of NSAIDs induced gastric ulcers in horses. Healing is time and dose dependent.

Studies on the effects of orally administered omeprazole on equine gastric acid secretion indicate that 3-5 days may be necessary for omeprazole to reach steady state and maximum antisecretory effects. Acutely symptomatic or painful horses/foals may require initial parenteral administration of omeprazole or a histamine H2 receptor antagonist for more rapid effect.

**Synthetic prostaglandin**

Prostaglandins inhibit the secretion of gastric acid, stimulate the secretion of mucus and bicarbonate, and provide cytoprotection for the gastric mucosa. Possible complications of prostaglandin treatment include diarrhoea, abdominal pain and abortion. There is insufficient data to recommend the use of prostaglandin for the treatment of gastric ulcers in horses at this time.

**Commerically available products in South Africa that are recommended for use in horses include:**

**Sucralfate**, trade name **Ulsanic**, available at 1g/5ml in 250ml bottles. The dose for an average size horse will be 55ml 3 times a day. This can be used in conjunction with a proton pump inhibitor but is not effective for long enough if used alone.

**Ranitidine** is available as 300mg tablets as **Histak** or **Ultak**. The average horse will need 110 tablets dosed orally 3 times a day. This is impractical and very costly.

**Omeprazole** is available as oral paste in the form of **GastroGard** or **Omepracote**.

**Prevention**

Negative factors have to be eliminated. Having fresh food available (day and night) seems to be very important especially when in box stalls. Training has to be very progressive.
Recurrent fasting, pain and long term treatment with NSAIDs are risk factors. Good observation and preventative treatment should prevent ulceration or allow one to recognize them at an early stage.

**Conclusion**
Gastric ulcers can present in many forms. Treatment is critical to healing these ulcers and preventing the progression of clinical signs.

The best diagnostic technique is endoscopy but this is an invasive and expensive procedure. The faecal occult blood test can therefore be considered as a more practical alternative.

The gold standard treatment is omeprazole at 4mg/kg for curative treatment and then at 2mg/g for preventative doses.