

EQUINE GASTRIC ULCER SYNDROME



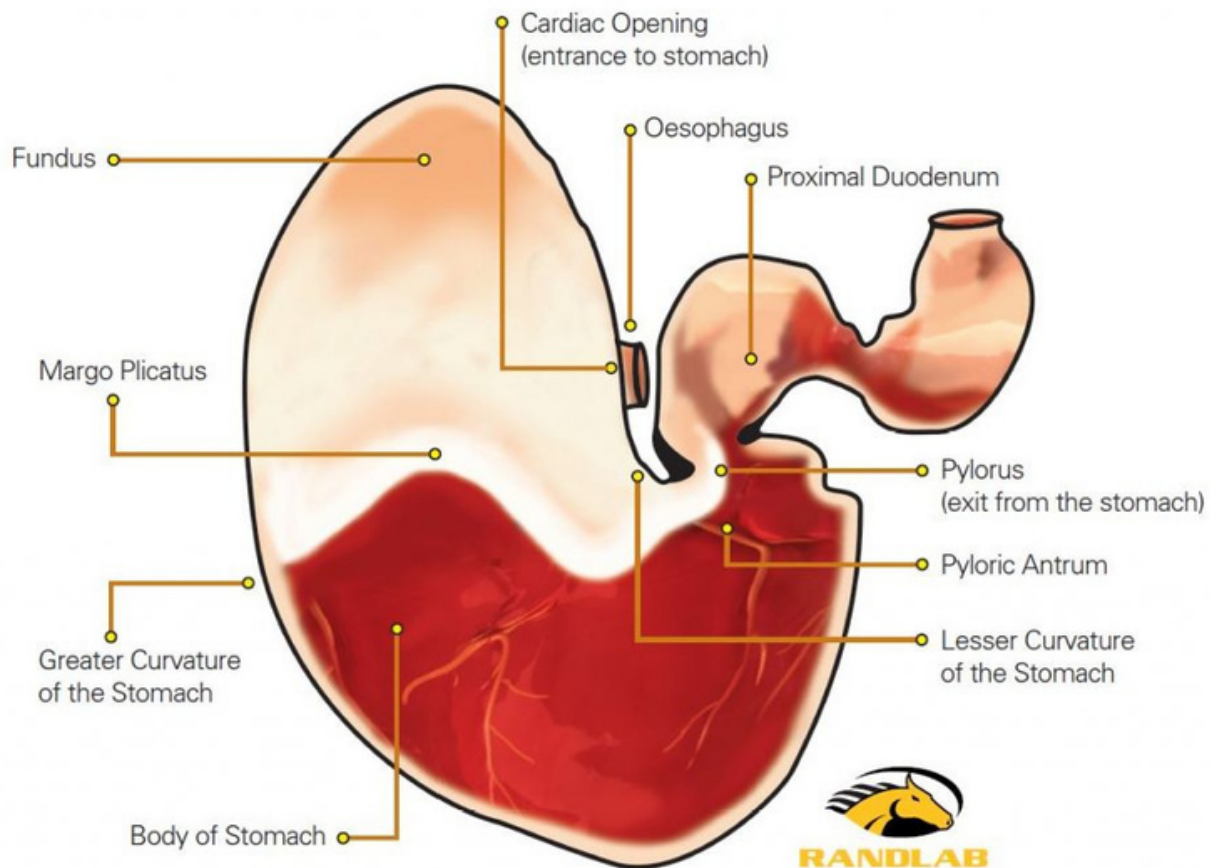
WHAT IS "EQUINE GASTRIC ULCER SYNDROME"?

Equine Gastric Ulcer Syndrome (EGUS) is the term used to describe ulcerative disease of the stomach. It encompasses both Equine Squamous Gastric Disease (ESGD) and Equine Glandular Gastric Disease (EGGD), which specifically describe which area of the stomach is affected. Gastric ulcers can occur in any horse, at any level of training and in a variety of living situations, so they are therefore often considered when a horse displays variable non-specific symptoms.

The equine stomach is divided into two major areas: the **squamous** (non-glandular) region and the **glandular** region. The squamous region is an extension of the oesophagus and does not have a mucous barrier protecting the mucosa from gastric acid. Therefore, it is this region that most often becomes inflamed and eroded when exposed to gastric acid for a prolonged period of time with up to 80% of gastric ulcers being classified as ESGD. The glandular region secretes a protective layer of mucus and bicarbonate to buffer and protect the mucosa as it is always exposed to gastric acid. Ulcers form in this region when the barrier 'fails'. The **margo plicatus** is the structure that forms the border between these two areas (non-glandular and glandular) and is where most ulcers are identified.

EQUINE GASTRIC ULCER SYNDROME

THE EQUINE STOMACH - ANATOMY



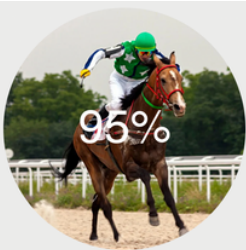
CONTRIBUTING FACTORS

- **Feed:** Many horse owners feed two daily feeds with a prolonged time between meals, which contributes to increasing the amount and acidity of the gastric fluid. Furthermore, high grain diets increase production of Volatile Fatty Acids (VFAs) that further decreased the fluid pH.
- **Stress** can be induced in horses for a variety of reasons and is totally dependent on the individual. Ultimately stress contributes to EGUS through elevated blood cortisol levels. for example, studies have demonstrated that just a few hours of transport can be enough to induce gastric ulceration in some horses.

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CONTRIBUTING FACTORS

- **Age:** Foals are susceptible to gastric ulcers as they secrete gastric acid from 2 days of age, the gastric mucosa is not fully developed, and long period without feeding reduces buffering effect of milk.
- **Exercise** is known to increase gastric acid production, decrease blood flow to the gastrointestinal tract and increase intrabdominal pressure. This results in gastric acid being pushed to the upper squamous portion of the stomach and with repeated exposure can result in damage.
- **Stomach size:** The equine stomach is proportionally small compared to other species. Therefore, they cannot handle large amounts of food and are built to graze continuously. This means the stomach naturally produces a steady flow of gastric acid during the day, which ideally should be buffered by saliva and a high roughage diet. However, many current management practices and feeding regimes often do not take this into consideration and therefore predisposes horses to developing ulcers.
- **Medications:** Chronic administration of non-steroidal anti-inflammatories (bute, flunixin) are non-selective and whilst reducing inflammation they also decrease production of the protective mucus layer by the glandular region and contribute to ulcer formation.



Racehorses in Training



Endurance Horses



Standardbred Racehorses



English Sports Horses



Western Sports Horses



Broodmares



Backyard Ponies

EQUINE GASTRIC ULCER SYNDROME

CLINICAL SIGNS

The clinical signs of a horse with gastric ulcers may include:

- Poor appetite*
- Dullness
- Attitude changes
- Decreased/poor performance
- Reluctance to train/jump
- Poor body condition
- Poor hair coat
- Weight loss
- Excessive recumbency
- Low-grade colic
- Loose faeces
- Teeth grinding
- Difficult to handle when tightening girth
- Behavioural changes when riding – eg. Bucking, pig rooting, reluctance to increase speed (canter)



*Horses with ulcers may walk away from their food when it first hits the stomach due to discomfort, slow to eat or may not finish their meals with a preference for roughage (hay).

Severity of clinical signs is often not associated with severity of gastric ulcers and a seemingly healthy horse can have gastric ulcers.

DIAGNOSIS

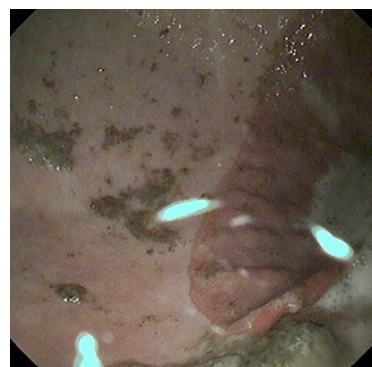
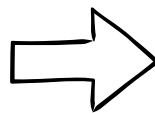
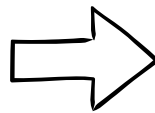
A definitive diagnosis can be achieved by visualization of the entire stomach via a minimally invasive **gastroscopic examination**. The patient is required to fast for at least 12 hours and have water withheld for 4 hours prior to scoping to allow full examination of the empty stomach. Under sedation, an endoscope is passed through their nose into their stomach. This allows the veterinarian to directly examine the stomach mucosa with particular attention to the location and severity of ulcers (graded from 1 to 4). Ideally, a gastroscopy should be performed prior to beginning treatment to determine the degree of ulcers and to prescribe appropriate treatment, duration and management changes. Thereafter, repeat gastroscopies determine that the ulcers have healed and should at least be performed prior to discontinuing any treatment.

EQUINE GASTRIC ULCER SYNDROME MEDICATION

- **Omeprazole** (Ulcershield or Gastrozol) acts by inhibiting the production of gastric acid by blocking the acid pump, therefore increasing the pH of the gastric fluid to keep it above pH 4. It is available as a once daily oral paste, granules and injectable forms and should be given an hour prior to food.
- **Ranitidine** (Ulcerguard) is another product used to reduce gastric acid production by blocking the acid receptor. It is also available as an oral paste and ideally needs to be given three times a day with horses removed from training to be effective.
- **Sucralfate** (Carafate) can be used in conjunction with the above treatments and/or when treating ECGD as it binds to the gastric glandular mucosa and is thought to promote healing.
- **Antacids** are of limited use in the treatment of gastric ulcers in horses as they must be administered in high volumes every 2 hours to neutralize stomach acid.

Case Example

This mare had recently moved to a new property and began showing signs of discomfort, including girthiness and wood chewing. A gastroscope revealed grade 2 ulcers on the greater curvature and grade 2-3 ulcers on the lesser curvature of her stomach. She was treated with a course of omeprazole, and a follow-up gastroscope at the end of treatment confirmed that her gastric ulcers had completely healed.



EQUINE GASTRIC ULCER SYNDROME

PREVENTION

- Prevention of gastric ulceration involves addressing underlying causative factors:
- **Frequency feeding** or keeping your horse on pasture with access to roughage 24/7. This will buffer gastric fluid and stimulate saliva production (saliva acts as a buffer to gastric acid).
- **Reduce** the amount of grain and concentrate feeds.
- **Addition of Lucerne hay** (helps to create a physical barrier within the stomach to stop acid splashing on the non-protected area of the stomach and also buffers acid).
- **Reduce use of non-steroidal anti-inflammatories** and if necessary use a safer, more-selective options (eg. meloxicam) or supplement with Sucralfate while treating with non-steroidals.
- **Limit stressful situations** (eg. travelling, separation).
- **When stabled allow horses to see other horses** and have access to roughage (either 24/7 or via frequent small feeds).
- **Feed lucerne hay before travelling and riding** to reduce the gastric acid 'splashing' onto the glandular regions of the stomach.
- **Wet down hay** (for glandular disease only).
- **Add oils into the feed** – this helps to line the stomach – make sure to introduce to diet slowly, up to half a cup of oil a week for a 500kg horse and no more than 2.5 cups of oil a day for a 500kg horse that isn't in excessive amounts of work (as a general rule).
- **Consider maintenance supplements such as 'Gastro-Protect'.**

It is important to recognise that EGUS is a multifactorial syndrome that occurs due to a wide variety of individual and environmental factors. Diagnosis and treatment is integral to improving the patient's comfort and quality of life as well as reduce reoccurrence in the future. Prevention, however, should not rely solely on continuous medical treatment for EGUS and it is necessary for owners to address other areas that could be adding the overall clinical presentation.

**If you have any questions or concerns please contact us on
0412 619 740.**