Review

The Current Status of Equine Gastric Ulcer Syndrome in Horses - Focusing on Diagnosis, Treatment and Prevention

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Review

The Current Status of Equine Gastric Ulcer Syndrome in Horses – Focusing on Diagnosis, Treatment and Prevention

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Simple Summary: Mucosal diseases of the horse's stomach are described as Equine Gastric Ulcer Syndrome (EGUS). The equine gastric mucosa consists of two different parts (keratinised and glandular), disease of these is now named Equine Squamous Gastric Disease (ESGD) and Equine Glandular Gastric Disease (EGGD). There are many studies investigating the causes, diagnostic methods, therapy and prevention of mucosal disease of both parts. Over the last years, ESGD and EGGD have been the focus of multiple studies. The main aim of this paper is to provide an overview of the latest knowledge on diagnosis, treatment and prevention of gastric ulcers in horses.

Abstract: The term Equine Gastric Ulcer Syndrome (EGUS) has been used since 1999. As there are important differences, the terms Equine Squamous Gastric Disease (ESGD) and Equine Glandular Gastric Disease (EGGD) have been introduced in 2015. Risk factors like dietary management, training regimens and drug side effects, as described for NSAIDs, predispose horses to ESGD or EGGD. To date, gastroscopy remains the gold standard for a reliable diagnosis. However, there are new diagnostic approaches using less invasive biomarkers. The proton pump inhibitor omeprazole is currently recommended by the American College of Veterinary Internal Medicine and the European College of Equine Internal Medicine (ACVIM/ECEIM) for the treatment of both ESGD and EGGD. Omeprazole per os is much less effective in treating EGGD, so it is often combined with sucralfate, which forms a protective mucosal barrier to shield it from the gastric acid. Intramuscular application of omeprazole has been shown to increase efficacy in EGGD therapy. Alternative non-pharmacological treatments have been investigated due to the high cost of approved omeprazole, long-term adverse effects in humans and the restriction on omeprazole use in some equestrian sports. An update on diagnosis, treatment and prevention of ESGD/EGGD is the focus of this review.

Keywords: equine gastric ulcer syndrome; equine squamous gastric disease; equine glandular gastric disease; horse; stomach; diagnosis; treatment; prevention

1. Introduction

Equine Gastric Ulcer Syndrome (EGUS) is the umbrella term used for diseases of both mucosal layers of the stomach. The dorsal part of the stomach is covered by squamous epithelium and the ventral part by glandular epithelium, which secrete hydrochloric acid, pepsinogen, histamine, mucus and sodium bicarbonate [1]. Currently, there is no clear relationship between the presence of ESGD and EGGD [2].

Primary ESGD is a result of acid exposure and secondary a direct result of delayed gastric emptying [2,3]. The equine gastric squamous epithelium is susceptible to acid injury, as it is poorly perfused and lacks a mucus-bicarbonate layer to protect it [4]. Some protection is provided by the thick keratinized epithelium together with the presence of high electrical resistance, tight epithelial junctions and an osmophilic phospholipid surfactant-like layer [2,5]. However, endogenous hydrochloric acid (HCl) and also other synergistic gastric components such as short chain fatty acids

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(SCFAs), lactic acid and bile salts can erode the outer keratinized layers and lead to disruption of the electrical properties of the transport cells [6]. Hyperkeratosis (thickening of the stratum corneum) also appears to be a response to excessive acid exposure [7]. Risk factors for ESGD are summarized in Table 1. Although stress is often cited as a risk factor for EGUS, there is little scientific evidence to support this. ESGD has also been found in wild horses that are not exposed to recognized risk factors and many horses show no clinical signs and mild lesions can heal spontaneously, raising the question of whether some degree of ESGD is "normal" [2,8].

The pathophysiology of EGGD is only partially understood. It is thought that a breakdown in the glandular mucosal barrier, which has protective factors (mucus, bicarbonate, prostaglandins, mucosal blood flow and epithelial restitution [9]), stress or inflammation may be responsible [3,10]. Prostaglandin E plays a crucial role in protecting the stomach by stimulating bicarbonate secretion, inhibiting hydrochloric acid secretion, maintaining microvascular flow and increasing mucus production. The severity of EGGD is associated with increased adrenal cortical sensitivity to ACTH, suggesting that stress inducing management factors should be investigated in horses with EGGD [11]. Risk factors for EGGD are shown in Table 2.

Prevalence rates for ESGD ranged from 11% to 100% and for EGGD from 6% to 70% in different studies, depending on the study population [12–15]. The prevalence of EGUS is highest in performance horses and reflects changes in management and intensity of exercise [2,10].

In recent years, there have been a number of notable publications, particularly on diagnostic markers and new therapeutic and preventive approaches. These developments underline the need for this review to provide a comprehensive overview of the current state of knowledge and to critically discuss the practical clinical applicability of these findings.

Table 1. Risk factors for ESGD.

Signalement	
Breed	The highest prevalence of gastric ulcers was found in Thoroughbreds and Standardbred trotters [16].
Sex (variable depending on study)	The mean non-glandular ulcer severity score was higher among mares when compared with geldings [12]. Female animals were more likely to have lower diagnostic scores than male castrated animals [17]. Gastric ulcers were significantly more prevalent in stallions than in geldings and mares [16].
Behavior	
Stress, which leads to reduced forage intake	A higher risk of developing ESGD or EGGD in relation to a high level of stress (i.e., intensity, duration or frequency of the exercise, travelling changes in the environment), and management practices that reduce the amount of time the horse spends eating [18].
Stereotypies	Horses with stereotypies were more likely to have ESGD [19].
No aggression towards human	Horses aggressive to humans were less likely to have ESGD [19].
Dietary	

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Fasting	Alternating periods of feed deprivation resulted in erosion and ulceration of the gastric squamous epithelial mucosa [20].
Low number of meals per day	The risk of nonglandular ulcers significantly increased when the interval between forage feeding was >6 h [21].
High feeding of starch (2 g/kg bwt of starch intake/day or > 1 g/kg bwt/meal)	Exceeding 2 g/kg bwt of starch intake per day was associated with an approximately 2-fold increase in the likelihood of EGUS ≥2 [21]. Alternatively, when included on a per meal basis, a starch intake between 1 g/kg bwt per meal and 2 g/kg bwt per meal, was associated with a 2.6 times increase in the likelihood of EGUS ≥2 and an intake greater than 2 g/kg bwt per meal increased the likelihood of EGUS ≥2 by 3.2 times [21].
Straw as the only available forage	The risk of nonglandular ulcers significantly increased when straw was the only forage available [21].
Barley feeding	Lesions of the pars nonglandularis (ESGD) occurred statistically more frequently when barley was fed [22].
Hypertonic electrolyte solutions administration	Oral hypertonic electrolyte administration (56.7g of a commercial electrolyte supplement every hour for 8 doses) to horses was associated with exacerbation of gastric ulcers [23].
Intermittent access to water	When water was not available in the paddock the likelihood of EGUS ≥2 increased by 2.5-2.7 times [21].
Husbandry	
Short term ownership	ESGD grade ≥ 1 is associated with years of ownership [24].
Usage	
Increased exercise intensity and duration of work	A higher risk of developing ESGD or EGGD in relation to a high level of stress (i.e., intensity, duration or frequency of the exercise) [18]. This findings appear to place the endurance horse at increased risk due to the duration of exercise in this sport [23].
Travelling/Transport	Horses that were transported and housed off-site (transported via trailer for 4 hours on day 0 and transported back on day 4) had a significantly higher incidence of hyperkeratosis and reddening in the nonglandular mucosa [25].

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Domesticated horses (intensive management)	Both squamous and glandular ulceration were more prevalent in domesticated horses when compared to the feral horses studied [8].
Medical history	
NSAIDs in combination with fasting	Feed-fast/NSAID model induce ESGD and EGGD in healthy horses [26].

Table 2. Risk factors for EGGD.

Signalement	
Breed	Warmblood breed was associated with an increased risk of EGGD [27].
Sex (variable depending on study)	Female animals were more likely to have lower diagnostic scores than male castrated animals [17]. Gastric ulcers were significantly more prevalent in stallions than in geldings and mares [16].
Behavior	
Stress	A higher risk of developing ESGD or EGGD in relation to a high level of stress (i.e., intensity, duration or frequency of the exercise, travelling changes in the environment), and management practices that reduce the amount of time the horse spends eating [18].
Dietary	
Feeding alfalfa chaff	Feeding alfalfa chaff induced glandular mucosal lesions at the antrum [28].
Straw as the only available forage	An increased likelihood of EGUS ≥ 2 was demonstrated when straw was the only forage provided [21].
High feeding of starch (2 g/kg bwt of starch intake/day or > 1 g/kg bwt/meal)	Exceeding 2 g/kg bwt of starch intake per day was associated with an approximately 2-fold increase in the likelihood of EGUS ≥2 [21]. Alternatively, when included on a per meal basis, a starch intake between 1 g/kg bwt per meal and 2 g/kg bwt per meal, was associated with a 2.6 times increase in the likelihood of EGUS ≥2 and an intake greater than 2 g/kg bwt per meal increased the likelihood of EGUS ≥2 by 3.2 times [21].
Intermittent access to water	When water was not available in the paddock the likelihood of EGUS ≥2 increased by 2.5-2.7 times [21].
Husbandry	

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Horses kept in a single barn with an open paddock or in a single barn with a pasture in groups	Lesions of the pars glandularis (EGGD) occurred significantly more frequently with housing in the individual stall and a freely accessible paddock or in the individual stall with grass pasture in groups [22].
Usage	
Training more than 4 days a week (not exercise intensity or duration)	A 10.4-fold increased risk of EGGD was associated with exercising ≥ 5 days per week in the present study, although no effect of exercise intensity or duration was observed [19].
Racing below expectations	Horses racing below expectation were 3.7 times more likely to have EGGD [19].
Trainer	Trainer was also identified as a risk factor for EGGD [19].
Multiple caretakers/riders	Horses with three riders and/or four caretakers had increased risk of EGGD [27].
Competition season (not international competition)	Currently showing increased the risk of EGGD grade ≥ 2/4, while competing at the international level decreased the odds of EGGD grade ≥ 2/4 [14].
Domesticated horses (intensive management)	Both squamous and glandular ulceration were more prevalent in domesticated horses when compared to the feral horses studied [8].
Medical history	
Administration of inappropriate doses of NSAIDs or NSAIDs in combination with fasting	The glandular portion of the stomach was most severely affected by phenylbutazone, flunixin meglumine, and ketoprofen [29]. Feed-fast/NSAID model induce ESGD and EGGD in healthy horses [26].
No history of colic or sand in the colon	Horses that had sand in their colon had a decreased risk of EGGD [27]. Horses that have experienced previous colic had decreased risk of EGGD [27].

2. Diagnosis

ESGD can be associated with a variety of very non-specific clinical signs, such as inappetence, poor body condition or weight loss, behavioral changes, acute or recurrent colic, bruxism, poor performance and stereotypic behavior [3]. In addition, the signs do not necessarily correlate with the severity of the lesions [30]. It is difficult to distinguish which signs are associated with ESGD or EGGD [31].

To date, gastroscopy remains the gold standard for antemortem diagnosis of equine gastric ulcers [2]. Various scoring systems are available, such as the commonly used 0-4 scoring system of the Equine Gastric Ulcer Council (EGUC) of 1999 [2] (Table 3). The EGUC system has demonstrated

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acceptable intraobserver and interobserver reliability in a study and was found to be reliable regardless of clinician experience [32].

Table 3. The Equine Gastric Ulcer Council grading system for squamous and glandular gastric disease [32].

Grade	Squamous mucosa	Glandular mucosa
0	The epithelium is intact and there is no appearance of hyperkeratosis	The epithelium is intact and there is no appearance of hyperemia
1	The mucosa is intact, but there are areas of hyperkeratosis	The epithelium is intact, but there are areas of hyperemia
2	Small, single or multifocal lesions	Small, single or multifocal lesions
3	Large single or extensive superficial lesions	Large single or extensive superficial lesions
4	Extensive lesions with areas of apparent deep ulceration	Extensive lesions with areas of apparent deep ulceration

The ECEIM Consensus Statement [3] recommends a descriptive rather than a grading system for EGGD, as the clinical relevance of the various manifestations of glandular disease has not yet been adequately investigated. However, EGGD scores have been used in many studies [28,33,34] to better assess treatment success.

In addition to a purely visual diagnosis of the gastric mucosa, it is also possible to perform a biopsy in combination with gastroscopy. This can be useful in the area of the glandular mucosa, as the appearance of the lesion is a poor indicator of the underlying severity [35]. Mucosal biopsies can determine the type and severity of the underlying disease. Biopsy can be particularly helpful in cases of EGGD that do not respond to management adjustments and medical therapies [36]. The biopsies can be examined histopathologically and/or culturally.

The clinical relevance of gastroscopically identified lesions should be assessed in the context of the severity of the clinical signs observed. It is also necessary to consider alternative differential diagnoses as previously described [37].

Other diagnostic methods for EGUS such as biomarkers or owner questionnaires have been investigated with variable results [2]. In the following section, biomarkers that have been recently identified as a result of research into alternative diagnostic methods are examined in more detail, with the corresponding abbreviations explained in Table 4 and in the abbreviations section.

Serum protein markers for a normal horse stomach were B4GALNT2 and XDH, for mild/moderate ESGD it was KRT10, while the marker for severe ESGD was KLK13 [38]. Other markers for both types of ESGD were SLC4A7, PPARG, FCGBP, PKP1, ASPRV1 and KRT5-like proteins. A total of 10 serum proteins were found to be potential markers for ESGD.

Using gel electrophoresis and mass spectrometry, 14 proteins were identified as potential markers of NSAID-induced EGGD. Many of them are mitochondrial proteins (MIA40, ACSM3, HSCB, DNAJA3, ECI1, AGXT2, AK2, AK4 and MRPL2) released from apoptotic gastric cells, while some proteins interact directly with phenylbutazone (PTGR2, UGT1A1 and PTBP1). These proteins may enter the blood circulation during NSAID-induced inflammation.

Horses with moderate or severe EGGD had a larger and quicker increase in cortisol concentration after ACTH stimulation with the best diagnostic accuracy 60 minutes after injection (sport horse population: 100% sensitivity and 75% specificity; general population: 75% sensitivity and 52% specificity)[39]. There were no significant associations with ESGD.

Hair cortisol concentration (HCC) was lower in horses with ESGD and was a negatively correlated with lesion severity [40]. HCC was inversely related to the severity of ESGD, as high plasma cortisol concentrations associated with stress lead to lower levels in the hair via a negative feedback mechanism [31]. Mares had lower HCC than geldings and a higher prevalence of ESGD. Age did not show a significant effect [40].

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Blood sucrose was neither a sensitive (51-79%) nor specific (43-72%) test for the detection of EGUS in adult horses with naturally occurring gastric ulcers [41].

Serum levels of gastrin and pepsinogen showed no significant changes in horses with EGUS compared to healthy horses [42]. TNF-α and IL-6 showed a significant increase in serum levels in horses with EGUS. Due to oxidative stress, higher levels of malondialdehyde (MDA; product of lipid peroxidation) and decreased serum levels of total antioxidant capacity (TAC), superoxide dismutase (SOD), glutathione (GSH) and nitric oxide (NO) are present in EGUS patients compared to healthy horses. MDA and TNF-α showed better sensitivity and specificity than IL-6 in differentiating horses with EGUS from control horses. In conclusion, higher levels of TNF-α and IL-6 indicate the development of EGUS. For preliminary screening of EGUS, MDA, TNF-α and IL-6 could be used as non-specific biological markers.

Twenty-three salivary biomarkers were tested for non-invasive EGUS diagnosis in 147 horses [43]. Samples were collected just prior to gastroscopy. The biomarkers tested included enzymes (ADA1, ADA2, ALP, AST, BChE, CK, gGT, LIP, LDH and sAA), metabolites and proteins (Creat, Ferr, TChol, TP, Trig and urea), redox biomarkers (AOPP, FRAS and UA) and minerals (Ca and P). Analytes that showed significantly higher concentrations in the animals with EGUS included ADA (both isoenzymes), ALP, AST, BChE, CK, gGT, LDH, sAA, Ferr, TP, Trig, urea, FRAS, UA, Ca and P (AOPP showed a trend with $P = 0.055$). The analytes that showed differences between the group of animals with EGUS and the group of animals with similar clinical signs but with other diseases were Ca, Trig and UA, which were higher in the EGUS group, and Ferr, which was higher in the group with other diseases. In general, no differences were found between animals with ESGD, EGGD or ESGD and EGGD, with the exception of ADA2, which was higher in the ESGD and EGGD group compared to ESGD alone, and Trig and Ca, which were higher in the ESGD and EGGD group compared to the EGGD alone.

Measurement of salivary calprotectin (CALP) and aldolase was evaluated as potential biomarkers for EGUS [44]. CALP is a calcium-binding S100 leukocyte protein associated with the innate immune response and inflammation. Aldolase belongs to a family of proteins involved in gluconeogenesis and glycolysis. Commercial assays are available for the measurement of CALP and aldolase. CALP was significantly higher in horses with EGUS than in healthy horses. Salivary CALP was able to detect EGUS patients from healthy horses with >84% sensitivity and 100% specificity. However, no significant differences were found between horses with EGGD and ESGD and between horses with EGUS and horses with other diseases. Aldolase activity was significantly higher in patients with EGUS than in healthy horses (67% sensitivity and 86.7% specificity). No significant difference was found between horses with EGGD and ESGD and between horses with EGUS and horses with other diseases. The increase in CALP in horses with ESGD may be influenced by the hyperkeratosis of the squamous epithelial cells. Aldolase is involved in the impairment of cell growth and proliferation of gastric epithelial cells. In conclusion, CALP and aldolase can be considered as potential biomarkers to differentiate horses with EGUS from healthy horses.

Horses with EGUS showed an increase in salivary proteins such as adenosine deaminase (ADA), triosephosphate isomerase, keratins and immunoglobulin heavy constant mu and a decrease in carbonate anhydrase (CA), albumin and prolactin induced protein [45]. These changes indicate that several pathophysiological mechanisms are involved in this disease, such as activation of the immune system, reduction of gastric defences mechanisms and inflammation. The omeprazole-treated horses showed lower levels of thioredoxin (TRX) expression after successful treatment, as measured by proteomic analysis and commercially available ELISA kit. These proteins could be potential biomarkers for the detection and monitoring of treatment success in EGUS.

Another study [46] showed that horses with EGUS had changes in the trace elements of saliva. In particular, zinc and magnesium concentrations were significantly lower in horses diagnosed with EGGD, while horses with both ESGD and EGGD had reduced salivary iron concentrations. These results are promising in two aspects: on the one hand, the altered concentrations could serve as

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potential biomarkers; on the other hand, they open up interesting perspectives for therapy, especially with regard to the possible effectiveness of systematic supplementation.

Table 4 provides an overview of the potential diagnostic markers for EGUS and an outlook on the clinical potential.

Table 4. Potential diagnostic markers for Equine Squamous and Glandular Gastric Disease and Equine Gastric Ulcer Syndrome in general according to current research.

Gastric Disease	Diagnostic Markers	Future Directions and Clinical Potential
Normal horse stomach	Serum protein markers: B4GALNT2 and XDH [38]	The absence or reduction of proteins specific for normal gastric mucosa may also be used as ESGD markers [38].
ESGD	Serum protein markers: KRT10 (mild/moderate ESGD), KLK13 (severe ESGD), SLC4A7, PPARG, FCGBP, PKP1, ASPRV1, KRT5-like proteins [38]	These proteins, which may be used as putative ESGD markers, are not well characterised in horses and future investigation is required to fully understand the function and association of these proteins to ESGD [38].
	Hair cortisol concentration is lower (inversely related to the severity) [40]	HCC would be a more logical parameter than plasma or saliva cortisol, which reflects more short-term stress. Further studies are required to determine the relevance of this association, and if such an association also exists for EGGD [40].
EGGD	NSAID-induced EGGD blood markers: MIA40, ACSM3, HSCB, DNAJA3, ECI1, AGXT2, AK2, AK4, MRPL2, PTGR2, UGT1A1 and PTBP1 [47]	However, confirmation of these candidate marker proteins is required with specific antibodies in the larger horse population before they can be considered for application in the field [47].
	Larger and quicker increased cortisol concentration after ACTH stimulation test (moderate/severe EGGD) [39]	The wide confidence intervals and thus the lack of diagnostic accuracy do not presently support clinical use. Horses with other pathologies may also show altered responses to ACTH administration. Excessive cortisol secretion appears to be associated with various medical problems such as stereotypies and EGGD, which are thought to be related to chronic stress [39].
	Lower Zinc and Magnesium concentrations in saliva [46]	Measure Zn, Cu, Mg and Fe in the saliva in a simple, fast and precise manner to detect changes [46]. Further studies should be performed to clarify the possible practical applications and their

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		supplementation's effect during the treatment.
EGUS	Increase in serum levels: TNF-α, IL-6 and MDA [42]	TNF-α, IL-6 and MDA are not specific markers for EGUS, further specific markers are required for diagnosis [42].
	Decrease in serum levels: TAC, SOD, GSH and NO [42]	TAC, SOD, GSH and NO are markers for oxidative stress and not specific for EGUS [42].
	Higher concentrations in salivary biomarkers: ADA1, ADA2, ALP, AST, BChE, CK, gGT, LDH, sAA, Ferr, TP, Trig, urea, FRAS, UA, Ca and P [43]	From those, UA, Trig and Ca could have a significant discriminant power between horses with EGUS compared to horses with other diseases with similar clinical signs. Higher values of UA, Trig and Ca in horses with clinical signs of EGUS would indicate a high probability of having EGUS in gastroscopy. These assays have the advantages of being non-invasive and also easy to measure because most of them are commercially available [43].
	Higher concentrations in salivary biomarkers: CALP and aldolase [44]	CALP and aldolase could be considered as potential biomarkers to differentiate horses with EGUS from healthy horses, but they did not show significant differences between horses with EGUS and horses with other diseases [44].
	Increase in salivary proteins: ADA, triosephosphate isomerase, keratins, and immunoglobulin heavy constant mu [45]	Horses with EGUS have changes in saliva proteins compared to healthy control horses. These changes would indicate the involvement of various physiopathological mechanisms (such as the activation of the immune system, decrease in the stomach defence mechanisms and inflammation) [45].
	Decrease in salivary proteins: carbonate anhydrase, albumin, and prolactin induced protein [45]	
	Lower Iron concentrations in saliva [46]	Measure Zn, Cu, Mg and Fe in the saliva in a simple, fast and precise manner to detect changes [46]. Further studies should be performed to clarify the possible practical applications and their supplementation's effect during the treatment.
	Omeprazole treatment: Lower levels of TRX expression after successful treatment [45]	This protein could be a potential biomarker for monitoring treatment response in EGUS and it could analysed

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	using a commercially available ELISA kit [45].

3. Treatment and Prevention

The successful treatment of gastric ulcers in horses requires not only the use of medications, but also a change in management to minimize the risk factors mentioned above, such as stress [48]. In the older literature, general recommendations were often given for EGUS, which now leads to misunderstandings in practice regarding the specific recommendations for the subtypes ESGD and EGGD. For example, it is now recognized that stress management plays a greater role in the management of EGGD, whereas dietary management is crucial for ESGD. For this reason, the recommendations for ESGD and EGGD are presented separately below.

3.1. Equine Squamous Gastric Disease

3.1.1. Oral Omeprazole

The drug of choice is omeprazole, a prodrug, which is absorbed in an alkaline environment, such as the small intestine, due to its enteric coating, which protects it from degradation in the stomach, and is converted into its active form, a sulfonamide, in the acidic secretory parietal cells. This in turn irreversibly binds to the gastric H+/K+-ATPase, and thus inhibits the acid production of the stomach [49]. The half-life of the oral formulations was longer (approximately 100 minutes) than that of the intravenous formulation (approximately 35 minutes) [50]. The oral formulations are approved for use in horses in many countries and are available in buffered and enteric-coated forms. Enteric-coated formulations have been shown to have higher bioavailability and that lower doses can be as effective as higher doses [50]. The bioavailability was with a mean of 21.5% higher in the orally enteric-coated omeprazole (4 mg/kg once daily) and fast (no feed for 16 h) group than with orally unprotected plain omeprazole under the same conditions with a mean of 10.1%. Similar differences were also observed in the area under the concentration-time curve (AUC), while there was no difference in the maximum plasma concentration (Cmax) between the both formulations. In addition, the data show that bioavailability is comparable in fed and non-fed horses receiving enteric-coated omeprazole. This indicates that the influence of feeding is less than previously assumed. In a further contrast to the recommendation to give omeprazole before feeding, it has been shown that for the most effective bioavailability, buffered omeprazole (Gastrogard™) should be administered during feeding to achieve a rapid increase in intragastric pH above 4 and to reduce the degradation of omeprazole by excessively acidic gastric contents [51]. On the other hand, the effects of diet (16 hours fasting or free choice alfalfa hay) or formulation (enteric-coated (Gastrozol™) or buffered (Omoguard™) omeprazole) on bioavailability was not statistically significant [52]. But examination of the raw data indicated a feeding effect that could reduce the bioavailability of omeprazole in the fed animal.

In Thoroughbreds in race training clinical signs often improved within 48 hours of starting omeprazole treatment [53]. Cure rates with oral omeprazole (ORLO) range from 67-94% and improvement rates from 89-100% depending on the study [54,55]. For the buffered formulation, a standard dosage of 4 mg/kg once daily for 28 days was used, while for the enteric-coated formulation, the dosage ranged from 1 to 4 mg/kg once daily for the same period. Both oral omeprazole formulations, powder paste and gastro-enteric resistant granules, in a dosage of 4 mg/kg bwt for 28 days showed resolution of lesions in 78.6% of ESGD [56]. In the gastro-resistant omeprazole granules group, 100% of ESGD lesions were improved or healed, compared with 71% in the omeprazole powder-paste group.

Without initial omeprazole treatment, the dietary change had no additional effect [57]. Horses with grade ≥ 3/4 ESGD showed a significant effect of omeprazole. In the group initially treated with omeprazole and maintained on their original diet, there was a significant improvement in ESGD, but

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this did not reflect any apparent long-term benefit of the medication. It was also shown that an appropriate feed conversion may be a beneficial management strategy for ESGD.

At a dosage of 1 mg/kg, omeprazole paste significantly prevented the occurrence of ESGD in training horses, with similar efficacy observed at a dosage of 2 mg/kg. Omeprazole prophylaxis was shown to be superior to placebo treatment, with only 23.4% of horses in the omeprazole group developing gastric ulcers compared to 77.2% in the placebo group. In addition, the severity of ulcers was significantly lower in the omeprazole groups [58]. It was shown that the 'ultra-low' dosage of 0.5 mg/kg omeprazole is equivalent to the 1 mg/kg dosage for the prevention of EGUS [59].

It was investigated whether the rebound gastric hyperacidity (RGH) known in human medicine, which leads to increased gastrin production and hypergastrinemia due to the loss of negative feedback to gastrin by the use of proton pump inhibitors, also exists in horses [60]. An additional effect of gastrin, which has a positive trophic effect on enterochromaffin-like cell (ECL) density, was used by measuring this increased cell density by serum chromogranin A. The aim was to investigate, whether this effect also occurs in horses, leading to a rapid recurrence of ESGD after omeprazole withdrawal. The mean serum gastrin concentrations increased 2.5-fold from the initial value up to the 7th day, but did not increase with further treatment. Mean serum gastrin concentrations returned to baseline within 2 to 4 days after administration of the last dose of omeprazole. No clear increases in serum chromogranin A concentrations were observed as an effect of treatment or discontinuation. In conclusion, these results do not support the use of tapering protocols in horses.

3.1.2. Long-Acting/Extended Release Injectable Omeprazole

The long-acting injectable omeprazole treatment (LAIO), administered intramuscularly at 4 mg/kg once weekly for two or four weeks, showed success rates of 86% and 97% in ESGD and had an improvement rate of 100% [54]. A minor complication rate of 5.1% was observed with the injectable treatment, which did not require further medical intervention. This study [54] showed that in contrast to a previous study in thoroughbred racehorses [61] which reported 100% healing after 2 doses of LAIO, it took 4 weeks for almost 100% of lesions to heal with LAIO therapy. In horses receiving four intramuscular injections of 2 g of a 100 mg/ml extended-release injectable omeprazole (ERIO) the cure rates (grade 0) for ESGD were 97% when administered every 5 days and 82% when administered every 7 days, with no significant difference. [62]. Injection-site reactions occurred in only 1% of cases. A measurement with the same dosage showed that the pH value was above 4 in 66% of all horses on days 1 to 4 and in 4 out of 6 horses on days 1 to 7 [61].

3.1.3. Esomeprazole

A comparison between oral esomeprazole and oral omeprazole concluded that treatment with esomeprazole, an S-enantiomer, was more effective when given once daily for 28 days at a dosage of 4 mg/kg for ESGD [63]. Esomeprazole achieved a cure rate (grade 0/4) of 85% compared with 59% for omeprazole. Currently, there are no registered esomeprazole formulations for horses.

3.1.4. H2-Receptor Antagonists

H2-receptor antagonists (such as ranitidine, famotidine and cimetidine) reduce acid secretion, but have been shown to be inferior to omeprazole for the treatment of ESGD [64]. H2-receptor antagonists bind to histamine receptors and thus reduce proton pump stimulation (without affecting gastrin and acetylcholine, which stimulate acid secretion) [10]. The duration of acid supression is short, which is why oral administration of 6.6 mg/kg ranitidine, for example, should ideally take place every 8 hours [64], which represents a significant additional expense compared to omeprazole.

3.1.5. Non-Pharmacological Treatment Options

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Alternative, non-pharmacological treatments have been studied, as omeprazole is very expensive, horses should not be unnecessarily treated with medication and there are restrictions on the use of omeprazole in some equestrian competitions [31].

It could be shown that severe ESGD could be improved or even cured within 4 weeks without drug therapy by providing horses with roughage ad libitum and a small amount of a low-starch supplement [65]. Also a predictable daily routine with a limited number of good caretakers may help to reduce stress levels and therefore improve gastric health. However, spontaneous healing of ulcers is rare and refusal of treatment may be considered unethical [66].

The protective management factors are set against the risk factors and include, for example, open stabling, the use of hay nets and recommend omeprazole in stressful situations [22].

Fibre is important in the horse's diet, and failure to meet the 1.5% of bodyweight fibre requirement, as well as inadequate access to forage for at least eight hours a day, can have consequences [67]. Providing hay can reduce behavioral abnormalities such as crib biting and wood chewing, as well as stress. To provide horses with a source of energy, rations often contain starch rather than fibre, which can lead to gastrointestinal health problems. Choosing a high fibre alternative to starch significantly reduces the risk of EGUS and acidosis and improves digestion, gastrointestinal pH, body condition, behavior and performance. The increased energy provided by starch-rich diets can also be provided in fibrous form, such as sugar beet pulp. Fibre can form a barrier between gastric acid and the cutaneous gastric mucosa when fed 30 minutes before exercise, which increases intra-abdominal pressure during exercise and pushes gastric acid to the cutaneous mucosa. Starch increases the production of short chain fatty acids and reduces the buffering capacity in the stomach by reducing saliva production due to low chewing activity, which in turn increases the risk of ESGD. Fibre type is crucial, which is why lucerne hay is said to be a better solution for gastric ulcers than grass hay. A high-fibre, low-starch diet has been shown to have a positive effect on the microbiota in the stomach and faeces and to promote the healing of gastric ulcers [68]. In horses with gastric ulcers, a lower diversity of the microbiota was observed (ulcer improvement with an increase in specific carbohydrate-utilizing and a decrease in lactate-fermenting species).

Water is also important because water deprivation causes stress, which increases serum cortisol concentrations, which is reflected in serum gastrin concentrations, which stimulates HCl secretion [67,69].

There are many supplements, such as antacids that work by buffering stomach acid. A combination of aluminium (30g) and magnesium (15g) hydroxide can increase gastric pH ≥ 4 for up to 2 hours [70]. A 30-day supplementation with a mixture of magnesium oxide (MgO) (20 g ionized Mg^{2+} per horse per day) given to 2-year-old French trotters in training significantly reduced ESGD severity, while the mean/median grade in the control group was unchanged [71]. This may be related to the buffering capacity of the Mg supplementation. There was no significant correlation between the plasma concentration of lipid peroxides (markers of oxidative stress) and the ESGD value.

Feeding the nutraceutical supplement Trophogast Pellet for 30 days (200 g once daily) significantly decreased the ESGD score in the treatment group (supplements + management changes), while in the control group (management changes only) the score remained the same [72]. The management changes carried out in both groups included an increase in pasture time, constant access to good quality hay and a reduction in the intake of non-structural carbohydrates. There was a significant reduction in the ESGD score in the treatment group, but no change in the control group. In conclusion, Trophogast pellet may be effective in promoting the healing of mild ESGD lesions in endurance horses. Trophogast pellet is designed to protect the gastric mucosa and contains pectin, soy lecithin, zinc oxide and sweet chestnut extract. Pectins turn into gel in an acidic environment and can therefore protect the mucous membrane from acid. They can also stimulate an increase in the pH value in the stomach. Lecithin is an exogenous phospholipid that can form a highly hydrophobic protective layer, thereby enhancing the acid-repellent properties of the phospholipids of the squamous mucosa. Zinc oxide and sweet chestnut extract may provide additional benefits due to their antioxidant properties by protecting the mucosa from the formation of harmful free oxygen

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radicals. Sugar beet pulp also contains pectin and lecithin [73]. Beet pulp feeding decreased the odds of ESGD grade ≥ 1/4 in a study of show jumping Warmbloods [14]. The beneficial effect of pectin-lecithin complex was also demonstrated in another study [74], but in still another study, lesions caused by intermittent feed deprivation could not be prevented [75].

Feeding oil has shown variable results in studies, but may be useful as a caloric replacement when switching to a low starch diet [2]. Feeding 118 ml of soybean oil, wild-caught fish oil and d-alpha-tocoperol before morning feed for 28 days did not prevent gastric ulcers [76], although omega-3 fatty acids found in fish oil have been used with some success in humans with gastric ulcers associated with Helicobacter pylori [77]. Omega-6 fatty acids found in soybean oil reduce gastric acid secretion in ponies [78]. Alpha-tocopherol supplementation in rats reduces the frequency and increases the healing rate of gastric ulcers [79].

Furthermore aloe vera (17.6 mg/kg bwt BID) appeared to have a positive effect on ESGD improvement [66].

An overview of ESGD prevention is summarised in Table 5. For the effective implementation of preventive measures, it is essential to consider the table of risk factors (Table 1) in combination with this table (Table 5), as the avoidance of risk factors is also a preventive approach.

Table 5. Prevention of ESGD.

Dietary	
Provide horses with roughage ad libitum and a small amount of a low-starch supplement	Severe ESGD can improve, and even heal, in 4 weeks with the provision of a diet consisting of ad libitum roughage and a small amount of a low-starch compound complementary feed [65].
High fibre, low starch diet	Choosing a fibrous alternative for starch in a high-energy diet will greatly reduce the risk of EGUS [67].
Provision of sufficient roughage and roughage meals per day (at least eight hours of feeding time per day)	Failure to meet the fibre requirement of 1.5% of the horse's bodyweight and the opportunity for foraging for a minimum of eight hours a day (not going without this opportunity longer than five hours) can have physiological and behavioural consequences [67].
Fibre fed 30 minutes before exercise	Giving a small forage meal within 30 minutes of starting exercise should reinforce stratification and limit splash lesion development [69].
Using a hay net	"Hay feeding from a net" mostly showed no or only minor EGUS [22].
Feeding oil (variable results, but useful as caloric replacement in a low starch diet)	Supplementation with Equine Omega Complete (omega-3 fatty acids and alpha-tocopherol) did not prevent gastric ulcer formation [76]. The addition of oil may be useful in replacing calories when transitioning away from a high starch diet [2].
Supplementation with magnesium oxide (20 g ionized Mg2+ per horse per day)	The supplementation of MgO (20 g ionized Mg2+ per horse per day) significantly decreased the ESGD scoring [71].

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Trophogast pellet (200 g once daily; contains pectin, soy lecithin, zinc oxide and sweet chestnut extract) combined with management changes	Trophogast pellet was effective at promoting healing of mild ESGD in endurance horses [72].
Sugar beet pulp feeding	Feeding beet pulp decreased the odds of grade ≥1/4 ESGD [14]. The increased energy supplied by high-starch diets can also provided in a fibrous form, such as sugar-beet pulp [67].
Aloe vera	Four weeks of treatment with aloe vera inner leaf gel, at 17.6 mg/kg bwt b.i.d., was inferior to treatment with omeprazole buffered paste, at 4 mg/kg bwt s.i.d. [66]. Nevertheless, 56% of cases showed some improvement in squamous lesion severity following 28 days of treatment with aloe vera [66].
No water deprivation	The importance of water is stressed as water deprivation increases the risk of EGUS [67].
Husbandry	
Open stabling	Horses with "open stables in one group", "open stables all year round" and "open stables plus grass pasture seasonally" mostly showed no or only minor EGUS [22].
Usage	
Predictable daily routine with limited number of good caretakers	A predictable daily routine, with a limited number of dedicated caretakers, may have contributed to lower stress levels and the improvement of gastric health [65].
Medication	
Prophylactic omeprazole treatment in times of expected reduced feed intake (1-2 mg/kg bwt oral omeprazole daily)	"Omeprazole as prophylaxis during stress" mostly showed no or only minor EGUS [22].

3.2. Equine Glandular Gastric Disease

3.2.1. Oral Omeprazole

Horses given an oral omeprazole paste (4 mg/kg po q 24 h for around 28 days), 50% of EGGD were cured, 75% improved and 25% were unchanged or worse [80]. In this study the cure rate with ORLO was greater than previously reported in other studies [81,82]. The comparison of both oral omeprazole formulations, powder paste and gastro-enteric resistant granules, showed resolution of lesions in 35.7% of EGGD [56]. The equal efficacy of enteric-coated (2 mg/kg) and buffered (4 mg/kg) omeprazole was also confirmed in another study [33].

When phenylbutazone (4.4 mg/kg po q 12 h) is given, the gastric pH increased more over time with omeprazole (4 mg/kg po q 24 h) than with phenylbutazone alone and the EGGD score increased

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with phenylbutazone alone compared to the combination with omeprazole [83]. Omeprazole reduced the formation of EGGD, but had no effect on the development of ESGD. In conclusion, the use of omeprazole improved phenylbutazone-induced EGGD, but was associated with an increase in intestinal complications (such as colic, impaction, diarrhoea, enterocolitis or typhlocolitis), whereas the use of omeprazole alone was shown not to alter the faecal microbiome [84].

3.2.2. Long-Acting/Extended Release Injectable Omeprazole

After 14 days of LAIO therapy (4 mg/kg once weekly), 52% of EGGD lesions were healed, 88% were improved, 6% were unchanged and a further 6% had worsened [80]. At the end of treatment (14 or 28 days), 82% were healed, 91% improved and 9% were unchanged or worse. The number of horses cured was greater with LAIO therapy than with ORLO therapy. There is no correlation between the cure or improvement rate and the resolution of clinical signs. 6.7% of the injection sites had self-limiting reactions.

Horses given four intramuscular injections of 2 g of a 100 mg/ml extended release injectable omeprazole (ERIO) with an 1% injection reacting, had a cure rate of EGGD (grade 0 or 1) of 93% with a 5-day treatment interval and 69% with a 7-day interval [62].

It is important to note that in many studies [54,62,80], owners were given dietary and management recommendations, which allowed several factors to influence the success of the therapy.

3.2.3. Esomeprazole

In horses with EGGD, esomeprazole (4 mg/kg once daily for 28 days) resulted in 55% success (grade 0 or 1) compared to 25% for omeprazole and for complete healing (grade 0), esomeprazole had a success rate of 47% compared to 17% for omeprazole [63].

3.2.4. Sucralfate

Omeprazole is less effective in treating EGGD, so a minimum of 8 weeks of therapy is recommended before considering complementary therapies [3]. Omeprazole is often combined with sucralfate, a hydroxyl aluminium salt of sucrose octasulfate, which binds to the negatively charged ulcer bed and forms a protective barrier [31]. The combination resulted in better healing of EGGD than omeprazole alone [85]. Sucralfate acts as a physical barrier that protects the mucosa from acid and prevents mucus breakdown, as well as increasing the viscosity of the mucus layer and enhancing hydrophobicity, resulting in reduced back diffusion of hydrogen ions [86]. It also stimulates mucus and bicarbonate secretion and may stimulate prostaglandin E (PGE) synthesis.

Omeprazole was more effective than sucralfate in reducing the development of EGUS [26]. In addition, horses treated with sucralfate developed a thickening of the colon.

When sucralfate (Sucrabest™, 12 mg/kg twice daily per os) was added to the omeprazole preparations GastroGard™ (buffered, 4 mg/kg once daily) or Equizol™ (enteric coated, 2 mg/kg once daily), 6/23 horses showed complete healing of EGGD (grade 0) and 13/23 horses showed only grade 1 findings [33]. No significant difference was shown in the effect of the combination of GastroGard™ and Sucrabest™ or the administration of high dose Equizol™ (3.5-4 mg/kg omeprazole) [87].

3.2.5. Misoprostol

Misoprostol, a synthetic prostaglandin E analogue, can enhance the protective mechanisms of the glandular mucosa in humans by improving blood flow, increasing mucus and bicarbonate secretion, and reducing acid production [88]. There are no veterinary products available for horses, instead products from human medicine (e.g., Cytotec™) are used. Monotherapy with misoprostol (approximately 28 d, Cytotec™, 5 µg/kg twice daily per os), with particular caution for pregnant owners and during the lactation period, was superior to both omeprazole/sucralfate therapy and combined omeprazole/misoprostol therapy in horses with EGGD [89]. Misoprostol (standard dosage

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mentioned before) showed a better cure rate of 72% on average than the combination of omeprazole and sucralfate with 20% and an improvement rate of 98% to 65% on average [81].

3.2.6. Glucocorticoids and Antimicrobials

As already mentioned in the diagnosis section, in addition to the preferred treatment options, the use of glucocorticoids and antimicrobial therapy are available as alternative treatment options in the event of a non-response [36]. Although the evidence base for antimicrobial treatments is still evolving and a previous study [90] did not support its inclusion in treatment regimens for EGUS. There is evidence that the targeted use of glucocorticoids may be beneficial, as there are similarities in the histological appearance of lesions in horses with IBD and EGGD [36]. Prednisolone at 1-2 mg/kg po SID or dexamethasone at 0.05-0.1 mg/kg po SID has been described, but is not licensed for this treatment [91].

3.2.7. Non-Pharmacological

As already mentioned, the protective management factors are set against the risk factors and include, for example, open stabling, the use of hay nets and recommend omeprazole in stressful situations [22].

Sea buckthorn berry supplementation (35.6 g berries and pulp twice daily) prevented the formation of glandular lesions in horses, which may be a useful complementary prevention strategy for EGGD. The exact mechanism is unknown, but it is thought that high levels of antioxidants reduce oxidative stress in the mucosa [92].

Supplementation with essential fatty acids increases the endegenous PGE2 production of the gastric mucosa and reduces acid production [78]. Thus, corn oil supplementation (45 ml daily) can be considered or the therapeutic and prophylactic treatment of EGGD.

An overview of EGGD prevention is summarised in Table 6. For the effective implementation of preventive measures, it is essential to consider the table of risk factors (Table 2) in combination with this table (Table 6), as the avoidance of risk factors is also a preventive approach.

Table 6. Prevention of EGGD.

Behavior	
Reducing stress	Stress might play a role in the pathogenesis of EGGD, and stress minimization could be beneficial in reducing the risk of EGGD [19].
Dietary	
High fibre, low starch diet	Choosing a fibrous alternative for starch in a high-energy diet will greatly reduce the risk of EGUS [67].
Using a hay net	"Hay feeding from a net" mostly showed no or only minor EGUS [22]. The importance of water is stressed as water deprivation increases the risk of EGUS [67].
Feeding sea buckthorn berry (SBT, 35.6 g berries and pulp twice daily)	Glandular ulcer scores were significantly lower in SBT-treated horses after feed deprivation [92].
Corn oil supplementation (45 ml daily)	Corn oil supplementation could be considered an economical approach to the therapeutic and

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	prophylactic management of ulceration of the equine glandular mucosa [78].
Husbandry	
Open stabling	Horses with "open stables in one group", "open stables all year round" and "open stables plus grass pasture seasonally" mostly showed no or only minor EGUS [22].
Medication	
Prophylactic omeprazole treatment in stressful situations (1-2 mg/kg bwt once daily per os)	"Omeprazole as prophylaxis during stress" mostly showed no or only minor EGUS [22].
Combining phenylbutazone with omeprazole	Administration of omeprazole ameliorated phenylbutazone-induced EGGD [83].

4. Conclusions

The gold standard for the diagnosis of EGUS is still gastroscopy. Although studies with biomarkers for diagnosis have already shown promising results, a practical implementation is still missing. ESGD is usually treated with oral omeprazole, while the long-acting intramuscular formulation of omeprazole is not approved for use and carries risks associated with the injectable route, it may be reserved as a second-line therapy in refractory cases. Treatment of EGGD is currently based on a combination of omeprazole and sucralfate or alternative approaches such as misoprostol. Another important aspect is the prevention of gastric ulcers, such as stress reduction in case of EGGD, a high-fibre, low-starch diet and limiting the intensity of exercise in case of ESGD. However, it is necessary to distinguish between ESGD and EGGD, as the pathophysiology of EGGD is not completely understood. Future research should focus on the development of practical, non-invasive and accurate diagnostic techniques and a better understanding of the pathogenesis of EGGD to further optimise treatment and prevention.

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Abbreviations

The following abbreviations are used in this manuscript:

NSAID	Non-Steroidal Anti-Inflammatory Drug
bwt	body weight
po	per os
q	quaque, every
BID	bis in die, twice a day
SID	semel in die, once a day
B4GALNT2	beta-1, 4 N-acetylgalactosaminyltransferase 2
XDH	xanthine dehydrogenase/oxidase isoform X6
KRT10	keratin, type I cytoskeletal 10
KLK13	kallikrein-13 isoform X2
SLC4A7	sodium bicarbonate cotransporter 3 isoform X3

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PPARG	peroxisome proliferator-activated receptor gamma isoform X1
FCGBP	IgGFc-binding protein
PKP1	plakophilin-1
ASPRV1	retroviral-like aspartic protease 1
KRT5-like proteins	keratin type II cytoskeletal 5-like
MIA40	mitochondrial intermembrane space import and assembly protein 40
ACSM3	acyl-coenzyme A synthetase mitochondrial isoform X1
HSCB	iron-sulfur cluster co-chaperone protein HscB, mitochondrial isoform X4
DNAJA3	DNAJ homolog subfamily A member 3, mitochondrial isoform X3
ECI1	enoyl-CoA delta isomerase 1, mitochondrial
AGXT2	alanine–glyoxylate aminotransferase 2, mitochondrial isoform X2
AK2	adenylate kinase 2, mitochondrial isoform X2
AK4	adenylate kinase 4, mitochondrial isoform X1
MRPL2	39S ribosomal protein L2, mitochondrial
PTGR2	prostaglandin reductase 2
UGT1A1	UDP-N-acetylhexosamine pyrophosphorylase-like protein 1
PTBP1	polypyrimidine tract-binding protein 1
TNF-α	tumor necrosis factor alpha
IL-6	interleukin 6
ADA1	adenosine deaminase 1
ADA2	adenosine deaminase 2
ALP	alkaline phosphatase
AST	aspartate aminotransferase
BChE	butyrylcholinesterase
CK	creatine kinase
gGT	γ-glutamyl transferase
LIP	lipase
LDH	lactate dehydrogenase
sAA	α-amylase
Creat	creatinine
Ferr	ferritin
TChol	total cholesterol
TP	total proteins
Trig	triglycerides
AOPP	advanced oxidation protein products
FRAS	ferric reducing activity of saliva
UA	uric acid
Ca	calcium
P	phosphorus

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