УДК 636.1:619:616.33

ETIOPATHOGENESIS AND DIAGNOSIS OF GASTRIC ULCERS IN HORSES

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Gastric ulcers is a common problem in horses. In the past few years, it seen a worsening of the problem, both in adult horses, as well as in foals [1, 3]. Increased diagnosis frequency of peptic ulcers is associated with the introduction of endoscopy of the stomach (gastroscopy) [2, 5, 6, 7]. Underlying to the increased incidence of gastric ulcers, are wrong nutrition, stress factors and the type of work, as well as the overuse of nonsteroidal anti-inflammatory drugs (NSAIDs) [4, 8, 13, 14, 16]. Currently the horses, because of the specific keeping way, and increased participation of ground feed (pellets, ground feed), spends less time eating, and extended or irregular intervals between meals compounded by the adverse effect of food is poor in fiber. Taking into consideration this aspect of the nutrition and heavy use in sport, the horses are exposed to the negative processes in the stomach, leading to ulceration of the mucosa. According to the Polish and foreign literature, gastric ulcers in horses depending on use, are in the following frequency: racing horses (mostly thoroughbreds) — 60–90 %, trotting horses — 50 % (94 %), jumping and dressage horses — up to 58 %, recreational horses-about 15 % [17, 20, 21].

Anatomy and physiology of the horse stomach

Horse's stomach is a monogastric complex, with a low capacity (8–15 liters). Indivisible stomach cavity is covered with 2 types of mucosa-mucosa lining the saccus cecus, similar to the those, lining of the esophagus, and the glandular mucosa, the appearance of velvet, stocked in the gastric glands *(glandulae gastricae)*. Esophageal zone *(pars esophagica),* devoid of glands and covered with a thick multi-layered flat epithelium, creates numerous folds which close esophagus and ends by the undulating course, called *margo plicatus*, bordering the glandular zone. Esophagus enters the stomach obliquely and closes its mouth inlet sphincter *(sphincter cardiae)* in the shape of a loop, built with well-developed here gastric smooth muscle [19]. Physiologically in the stomach there is a balance between the «aggressive» factors (gastric acid, pepsin, bile acids, propionate and acetate) and protective factors (mucus covering the mucosa, bicarbonates, close connections epithelial cells, rapid and continuous regeneration of the epithelium, appropriate blood flow in the mucosa) [10, 20, 21, 23]. Secretion of hydrochloric acid takes place under the influence of stimulation of the vagus nerve *(nervus vagus)*, as well as the action of gastrin and histamine, which plays in horses potentially the more important role (Tab. 1). Gastrin also stimulates the secretion of water, sodium, chloride and bicarbonate from the pancreas into the duodenum. Some of these secrets physiologically shows reflux in the direction to the stomach [22, 23]. The horse's stomach secretion of hydrochloric acid takes place in a continuous manner, even if the animal does not receive food. Acidic stomach pH (1,0–7,0 in foals, adult horses in 1,5–7,5) is reduced while eating, as a result of saliva rich in bicarbonates [20, 22]. In a short time after the cessation of foraging by a horse, acidification of gastric contents, is increasing rapidly, reaching a pH value below 2,0 [20, 22, 23].

Table 1

Regulation of gastric acid secretion (23)

Gastric acid secretion mediators (paracrine, endocrine or central effect)			
Generators		*Inhibitors*	
Paracrine or endocrine effect	Central effect	Paracrine or endocrine effect	Central effect
Gastrin	Thyrotropin — releasing hormone	Somatostatin	GRP
Histamine	Cholecystokinin	PGE_2	Opiates
Acetylocholine	Somatostatin	Secretin	Corticotropin — releasing factor
GRP (gastrin — releasing peptide)	Oxytocin		

Glandular part of the stomach has created mechanisms to safeguard against the adverse effect of hydrochloric acid. Include them mucose-bicarbonate barrier, adequate blood supply needed to repair the damage and growth factors which support healing. Blood flow in the mucosa is conditioned mainly by synthesis of prostaglandin (PG) and nitric oxide (NO). The biggest role of prostaglandin E2 (PGE_2) which increases the secretion of rich in bicarbonates mucus, improves blood circulation and inhibits the secretion of hydrochloric acid. Capillary perfusion is an integral component of mucosa, and any disruption of blood flow is a factor conducive to the occurrence of ulcers [21, 22, 23].

Etiopathogenesis

The most common causes leading to the formation of gastric ulcers in horses are:

1. wrong feeding and maintenance,
2. stressful sports training,
3. overuse of medicaments.

Inadequate maintenance and feeding

Inadequate maintenance and feeding systems lead to abnormal physiological processes occurring in the gastrointestinal tract. The natural feeding habits of horses, ie, frequent takeing of small quantities of feed causes the stomach is almost always filled with content, and the production of saliva, which has a buffering capacity, there is practically constant. The rate of gastric emptying is highly dependent on the type of feed — emptying of the stomach after eating concentrate takes about 30 min., while the administration of hay takes up to 24 hours. Situation in which the stomach is empty, and reduced production of saliva, leads to damage effects on the gastric mucosa of the stomach. On those grounds, the main dietary cause of the formation of gastric ulcers in horses are:

— lack of access to pasture;

— long intervals between feedings;

— hunger strike for many hours (effect continuously secreted gastric juice in the absence of buffers, such as saliva and food content);

— the use of concentrated pelleted feed (high content of digestible carbohydrates leads to an increase in the concentration of volatile fatty acids in the stomach);

— limit the amount of roughage with a high fiber content (rapid emptying of the stomach, small production of saliva) [12, 20, 21].

Stressful sports training

Stressful sports training leads to increased risk of stomach ulcers. During a stressful situation can occur:

— increase intraabdominal pressure and release of gastric motility, which has a consequence in reduced gastric emptying and increased influence on gastric mucosa, mainly in parts nonglandular (when horses are moving faster than the walk, pressure that arises in the abdomen

causes the «bites» of the stomach, with the consequence of the «shift» the gastric content in to the nonglandular mucosa);

— reducing blood supply to the stomach wall, which decreases the production of gastric mucus and bicarbonate;

— inhibition in the production of prostaglandins and decrease in mucus production due to increase of the concentration cortisone in the blood;

— obstruction the blood vessels in gastric mucosa, thus reducing its protective ability [9, 20, 21, 23].

The use of anti-inflammatory drugs with low selectivity for cyclooxygenase.

Inhibition of prostaglandin production by administration of anti-inflammatory drugs can lead to ulceration of the gastric mucosa. Particularly the main risk is associated with administration of the old generation of NSAIDs (phenylbutazone, piroxicam). These preparations inhibit both cyclooxygenase-1 (COX-1), and cyclooxygenase-2 (COX-2). Modern anti-inflammatory drugs show significant selectivity and inhibit only COX-2 (responsible for inflammation proces), in the absence of inhibition of COX-1 (responsible for the production of protective prostaglandins acting in the stomach). In contrast to gastric ulcer in humans, in horses so far failed to conclude bacterial factor such as *Helicobacter pylori*, which is potentially important etiologic reason causing gastric ulcers. This has an impact on the therapeutic procedure in which the use of antibiotics is not justified [20–23].

Diagnostics

Clinical symptoms of gastric ulcers in horses are nonspecific. In foals we usually observed: poor hair condition, diarrhea, fever, excessive salivation, teeth grinding, loss of appetite, cessation of suckling and increased lying down on back. The most frequently reported symptoms occurring in adult horses include: poor condition, worse performance, weight loss, apathy, decreased or finicky appetite, diarrhea or constipation, frequent colic (especially after feeding), teeth grinding, chewing without food and *fetor ex ore* [11, 15, 20–23]. Differential diagnosis should include disease of the teeth, guttural pouches, oesophagus as well as gastric and intestinal diseases with different etiologies.

A final diagnosis can be placed basis only by the gastroscopy, performed in horses using videoendoscope with working length about 2,5–3 m, which allows for accurate assessment of the gastric mucosa. Introduced 5-step scale for assessing the gastric mucosa:

— grade 0 — epithelium intact, normal mucosa;

— grade 1 — intact mucous membrane, but the visible areas of congestion and/or hyperkeratosis;

— grade 2 — small single erosions or ulcers;

— grade 3 — extensive erosion and / or multiple ulcers;

— grade 4 — large ulcers reaching submucosal membrane [2, 18, 21].

Laboratory tests are not relevant due to the lack of specific morphological changes in the parameters of the blood or in the biochemical profiles [20]. It is possible to carry out a study aimed at the presence of fecal blood, which could indicate bleeding from the gastrointestinal tract, but this is only possible in foals. Later, in adult horses, the colon microflora, absorbs hemoglobin and exclude the possibility of blood detection [23].

Aim of the study. The aim of this study was to describe the current knowledge concerning the etiology, pathogenesis and diagnosis of gastric ulcers in horses based on our own experience.

Materials and methods

The study included 15 horses of different races and sexes, aged from 1 to 8 years. Gastroscopy was performed after appropriate dietary preparation, which included a 24-hour hunger strike and 6–8 hour break in the administration of water before the test. Examination was conducted

in horses after detomidine sedation (at a dose of 40 mg/kg). The study was performed with Storz videoendoscop with working lenght of 3,25 m and a diameter of 1,5 cm

Results

Basis on the gastroscopy, in the 13 examinated horses gastric ulcers were diagnosed, which were located in the glandular or nonglandular mucosa (Fig. 1, 2). Using the 5-step scale for assessing damage of the gasric mucosa, we observed congestion and hyperkeratosis in 2 horses (grade 1), 5 horses were found in a single erosions (grade 2), in 6 horses we diagnosed the extensive ulceration (grade 3), while none of the identified lesions was classified as grade 4. In animals subjected to clinical examination, clinical symptoms of gastric ulcers were nonspecific. After performance an anamnesis, as the main cause of the ulcers were feeding mistake and stressful training. In 5 horses, the development of the disease occurred as a result of excessive use of NSAIDs with low selectivity for cyclooxygenase (phenylbutazone).

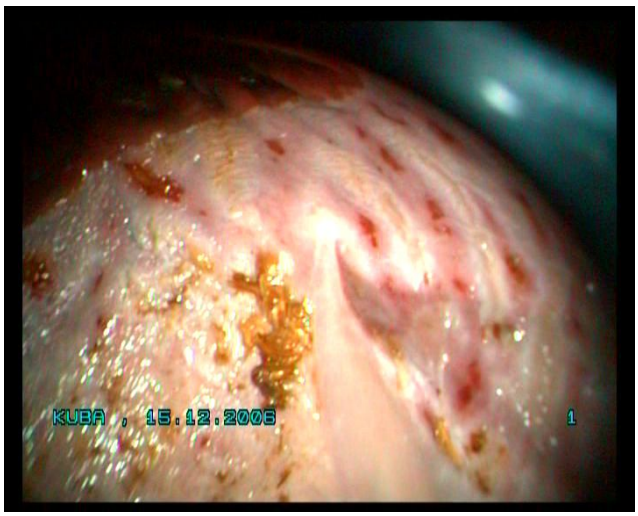

Fig. 1. Gasric ulcers in nonglandular part

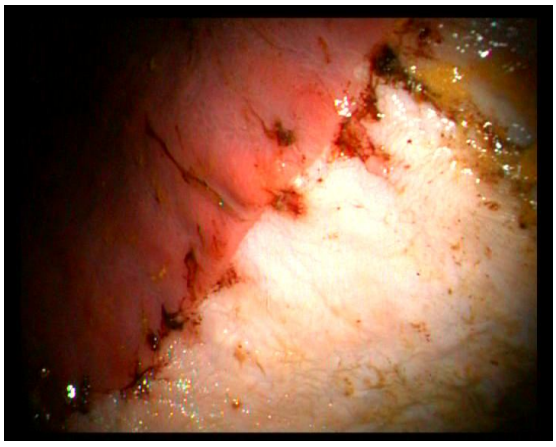

Fig. 2. Gasric ulcers in *margo plicatus*

Conclusions

Gastric ulcers are more common problem in horses subjected to an intense workout for sport, and wrong nutrition, leading to disruption of physiological processes occurring in the gastrointestinal tract. This condition can significantly reduce the utility value of the horse.

Currently, gastroscopy is the only opportunity to fully recognize gastric ulcers. With early diagnose and after the appropriate treatment, the prognosis is favorable.

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ЕТІОПАТОГЕНЕЗ ТА ДІАГНОСТИКА ВИРАЗКОВОЇ ХВОРОБИ ШЛУНКА У КОНЕЙ

Р е з ю м е

Виразки шлунка поширені у коней при інтенсивних фізичних навантаженнях і порушеннях годівлі. Хвороба спричиняє порушення функціональних процесів у травному тракті. Розвиток виразок шлунка веде до зменшення продуктивності коней. Використання гастроскопії дає можливість діагностувати виразкову хворобу шлунка. За умов ранньої діагностики та при застосуванні ефективних терапевтичних заходів виразка шлунка у коней виліковується.

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ЭТИОПАТОГЕНЕЗ И ДИАГНОСТИКА ЯЗВЕННОЙ БОЛЕЗНИ ЖЕЛУДКА У ЛОШАДЕЙ

А н н о т а ц и я

Язвы желудка распространены в лошадей при интенсивных физических нагрузках и нарушение кормления. Болезнь приводит к нарушению функциональных процессов в пищеварительном тракте. Развитие язв ведет к уменьшению производительности лошадей. Использование гастроскопии дает возможность диагностировать язвенную болезнь желудка. При условиях ранней диагностики и использовании эфективных терапефтических мероприятий язвы желудка у лошадей вылечиваются.

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