



Original Full Paper

Gastroduodenal Lesions in Racehorses: Evaluation and Mapping According to the Updated Sidney System and Equine Gastric Ulcer Syndrome Council Classification

Juliana da Silva Leite*, Leopoldo José Cury, Ana Maria Reis Ferreira

Department of Pathology and Veterinary Clinics, Veterinary Faculty, Universidade Federal Fluminense (UFF),
Rua Vital Brasil Filho, 64, Vital Brasil, Niterói, RJ 24230-340, Brazil.

*Corresponding author: E-mail: jsleite@id.uff.br

Submitted March 23rd 2012, Accepted July 19th 2012

Abstract

Gastroduodenal ulceration is found in 90% of Thoroughbred race horses. The clinical and economical importance of gastric ulcers and the scarce information about the subject in Brazil make this investigation essential. The aim of this study was to evaluate and map grossly and microscopically the gastroduodenal lesions in race horses according to the Updated Sidney System and the Equine Gastric Ulcer Syndrome Council (EGUSC) Classification. Necropsy was performed in 14 horses and macroscopy was described. Samples of the gastric regions (14 animals - nonglandular fundus, glandular fundus, margo plicatus, antrum and pylorus) and duodenum (9 animals) were obtained and processed for routine histopathology. Macroscopic evaluation of the lesions based on the Updated Sidney System showed that 100% of the horses had gastric ulcers or erosions, mainly located at the margo plicatus. The Updated Sidney System also allowed a macroscopic gastritis classification and revealed that seven horses had enanthematous gastritis (50%), four had hemorrhagic gastritis (28, 6%), and three had erosive gastritis (21, 4%). Based on the EGUSC Classification, the most frequent score is 2 (50%). The score 3 was found in 35,7% of the horses and the scores 1 and 4 in 7,1% each. The score 0 which means intact epithelium, was not seen in any of the horses. Histopathological evaluation based on the Updated Sidney System revealed mild to severe lymphoplasmacytic infiltrate, sometimes with areas of neutrophilic infiltrate, demonstrating some activity mainly in the injured mucosa. Lymphoid follicles were found in 92.9% of the animals. This study confirms that gastric ulceration in Brazilian race horses is common, and the majority consists of chronic ulceration of the margo plicatus. The use of the histopathological division of the Updated Sidney System demonstrated that chronic gastritis was predominant and the majority of cells in the inflammatory infiltrate were lymphocytes and plasmocytes. This study also showed that the association of the Updated Sidney System and the EGUSC Classification is feasible and leads to a better understanding of equine gastroduodenal ulceration. Although usually neglected, these lesions may be involved in low performance rates and therefore other investigations are necessary for the elucidation of the etiology and pathogenesis of these ulcers.

Key Words: Gastroduodenal disease, horse, histopathology, gastric ulcer, Updated Sidney System, Equine Gastric Ulcer Syndrome Council Classification.

Introduction

Gastric ulcer is a prevalent and important disease in the Equine clinic (30, 37) affects the Equidae family (9) and also is one of the main problems that affects the welfare and performance of horses. Pathogenic

mechanisms are being elucidated by the scientific community (30, 37).

Gastric ulceration affects a great number of colts, young and adult horses (7, 10, 18, 26, 27, 29). It is present in up to 50% of the asymptomatic colts endoscopically examined (7, 15, 28) and in 50% of the Brazilian adult animals not submitted to race training (16). Additionally,

race training horses are at greatest risk with diagnosed gastric ulcers in 90% of the individuals (27).

The signs of the Equine Gastric Ulcer Syndrome (EGUS) in adults are: acute or recurrent colic, excessive decubitus, low body condition, partial anorexia or loss of appetite, low performance/training, changes of attitude and chronic diarrhea (1).

An ulceration or inflammation of the gastric and duodenal mucosa can occur every time when the glycoprotein mucosal barrier and the integrity of the epithelial cells are disrupted, allowing the mucosa self-digestion by pepsin and gastric acid (38). The use of non-steroidal anti-inflammatory drugs, aspirin, cigarettes, alcohol ingestion, gastric hyperacidity, gastroduodenal reflux, drop in the mucosa blood flow, shock, late gastric emptying, deficient epithelial repair and host factors are considered causes of peptic ulceration in humans. Psychological stress is also considered an important contributing factor to the development of ulcers in humans (21). In colts, stress is believed to contribute or even cause gastroduodenal ulcers (29).

It is accepted that stress lowers the defense mechanisms of the gastric mucosa against the ulceration induced by gastric acid and pepsin (20). Stress related to the environment such as excessive heat or a sudden drop of the barometric pressure are also factors implied in the formation of ulcers in colts (38).

Besides stress, other risk factors for the occurrence of EGUS are transportation, high-grain diet, stabulation, intermittent feeding, intense exercise, race training, disease and use of non-steroidal anti-inflammatory drugs (NSAIDs) (1).

The anatomic location of the lesion must be taken into consideration for the study of peptic disorders pathogenesis in horses. The lesions on the squamous gastric mucosa result mainly from the excessive acidity, while the glandular gastric lesions result from the defective mucosal protection. Erosions can occur on the non glandular mucosa within 24 hours of exposure to excessive acidity (30). Contributing factors to gastric lesions of the non glandular region include the ingestion of concentrate, periods of fasting and the intensive training programs which lead to an increase of the abdominal pressure taking gastric acid to the non glandular portion of the stomach (30). On the other hand, the pathophysiology of the glandular mucosa lesions is much less understood. The factors that are considered promoters of the glandular mucosa lesions include excessive doses of non-steroidal anti-inflammatory drugs (30, 17); alterations in the mucosa blood flow, disruption of the mucosa protection, periodical retrograde reflux of duodenal content; a role on these lesions for *Helicobacter* spp. must also be considered (30).

The Updated Sidney System was developed to classify gastritis in human beings by gastroscopy and histologically. This classification emphasizes the importance of the topographic, morphological and etiological information for reaching the correct diagnosis

(13) and has already been used to classify gastritis in dogs (14), but no reports have been found about its use in equines.

In equines, a gastric ulceration graduation system was developed by the Equine Gastric Ulcer Syndrome Council (3). Its use is easy and the correlation among independent observers is satisfactory (6).

The association of the two classification systems would be ideal for a better understanding of the infirmity because it would guarantee uniformity regarding the interpretation of the endoscopic (gross lesions) and histopathological exams, and a better interconnection of these complementary exams with clinical evaluations. This interaction is essential for a more precise and reliable final diagnosis and to supply a more accurate prognosis.

The clinical and economic importance of gastroduodenitis and gastroduodenal ulceration and the scarce information about them in Brazil makes this investigation essential. The aim of this study was to evaluate and map grossly and microscopically gastroduodenal lesions in racehorses according to the Updated Sidney System and the Equine Gastric Ulcer Syndrome Council Classification (EGUSC).

Material and methods

Necropsy was performed in 14 Thoroughbred race horses. Three of these animals died and 11 underwent euthanasia due to other diseases that did not involve the focus of this study. This study was submitted to and approved by the Committee of Bioethics on Research (protocol – UFF 032/06). The horses clinical data were obtained from their medical records.

The stomach was opened along the greater curvature and grossly divided into regions: non glandular fundus, *margo plicatus*, glandular fundus, antrum and pylorus. Gross evaluation was descriptive. The ulcers and erosions were macroscopically classified according to the grading system proposed by the EGUS Council (3) in the following grades: 0- intact epithelium; 1- mucosa with areas of hyperemia and/or hyperkeratosis (thickening of the non-glandular epithelium); 2- small, isolated or multifocal erosions or ulcers; 3- large, isolated or multifocal ulcers, or extensive erosion and bleeding; and 4- extensive ulcers, with deep submucosa penetration areas.

All regions were sampled and for this, the non glandular and the *margo plicatus* regions were split into three areas and the non glandular and antrum regions into two areas (Fig. 1). A duodenum fragment was also sampled (9 animals). Besides the standard sampling described above, areas with macroscopic morphological changes of the mucosa were obtained for analysis. The gastric and duodenal samples that underwent histopathological analysis were fixed in 10% buffered formalin, cleaved, paraffin embedded and 4µm sections were stained with hematoxylin and eosin.

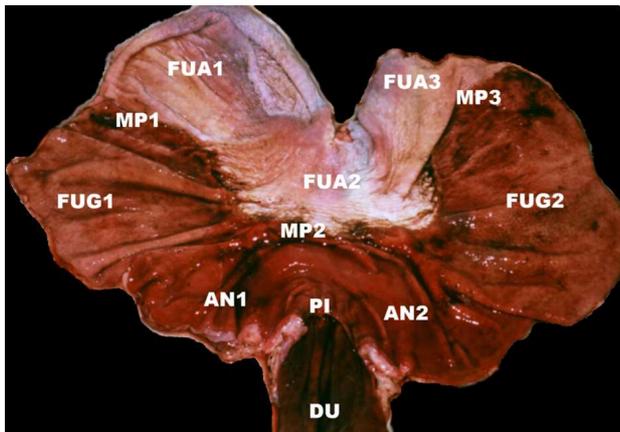


Figure 1. Equine stomach opened along the main curvature in which sampling is indicated: three samples from the non glandular fundus (FUA), three samples from the *margo plicatus* rugae (MP), two samples from the glandular fundus (FUG), two samples from the antrum (AN), one from the pylorus (PI) and one from the duodenum (DU).

The study of gastritis was carried out based on the gross and microscopic morphological concepts and classifications of the Updated Sydney System histopathological and endoscopic divisions (12, 13). Regarding the histopathological analysis, the normal gastric mucosa morphology was described as well as any observed alterations. For the gastric mucosa morphological evaluation, the inflammatory cells were graded under 400X magnification, obtaining the average from five randomly chosen microscopic fields. The following grades were established: absent = no cell/field, mild = 1 to 50; moderate = 51 to 150; severe > 151 cells / field, approximately.

Results

The study was performed in six Thoroughbred male and eight female horses at ages between 22 months and 10 years with an average age of about 4 years. The training period of the studied animals varied from 5 months to 2 years (average time of 14 – 75 months). Lumen evaluation, made according to the Updated Sydney System, detected no parasites, stenosis, masses or strange bodies in any of the analyzed animals, but mucus was found in 50% (7/14), fluid in 92.9% (13/14), fibrinous exudates in 28.6% (4/14) (Fig. 2A and 2B) and blood in 14.3% (2/14) of the horses.

All animals had, by the macroscopic analysis based on the Updated Sydney System, some morphological alteration in the gastroduodenal mucosa at different grades, such as: edema in 92.9% of the animals (13/14), erythema in 92.9% (13/14), hemorrhage in 78.6% (11/14), friability in 35.7% (5/14), granularity in 50% (7/14) and thickening of the non glandular epithelium in 71.4% (10/14) (Table 1).

Gastric rugae evaluation, item that composes the Updated Sydney System, revealed that 7.1% (1/14) of the equines had atrophy, 21.4% (3/14), hypertrophy (Fig.2A) and 71.4% (10/14) normal gastric rugae. The submucosal veins were visualized in 35.7% (5/14) of the individuals. The macroscopic analysis according to the Updated Sydney System showed that 100% of the animals had gastric lesions (Fig. 2A, B and C). There was a variation in the location, activity and evolution time of these ulcers and erosions. Regarding the location, the *margo plicatus* was the most affected (92.9% or 13/14), followed by the non glandular fundus (28.6% or 4/14). The glandular fundus, the antrum and the pylorus showed one lesion each (7.1% or 1/14).

According to the macroscopic morphological analysis based on the Updated Sydney System endoscopic version, seven animals (50%) had enanthematous gastritis; four (28.6%) had hemorrhagic gastritis (28.6%); and, three had erosive gastritis (21.4%) (Fig.2A) with grading varying from mild to severe (Fig.3).

Table 2 indicates the classification of ulcers and erosions of each animal and demonstrates the evolution time of the lesions considering their morphological aspect and based on the macroscopic alterations grading system proposed by the EGUS Council.

The most frequent EGUS grade was 2 (50% or 7/14) followed by 3 (35.7% or 5/14). Grades 1 and 4 were found in one animal each (7.1% or 1/14). And grade 0, which indicates intact epithelium, was not found in any animal (0/14). Thus, the EGUS grade average in the evaluated horses was 2.43.

Histopathology revealed that the squamous epithelium of the stomach of eleven animals (78.5%) showed hyperplasia of the basal layer. Equines number 5 and 10 (14.3%) presented normal basal layer in the non glandular gastric region samples, and Equine 12 (7.1%) had an irregular basal layer. Samples of all the horses had some degree of *parakeratosis*, acanthosis and hyperkeratosis in the non glandular region (Fig.2D).

Some degree of congestion was observed in at least one of the gastroduodenal mucosa samples of all animals (Fig.2D). Only gastroduodenal mucosa fragments of horses 2 and 12 lacked hemorrhages. Deposit of fibrin in the gastroduodenal mucosa surface was found in, at least, one sample of the gastroduodenal mucosa of 71.4% (10/14) of the evaluated animals.

Histopathological analysis also revealed mild atrophy of gastric glands (28.6%) in animals 5, 10, 12 and 14. Horse number 6 (7.1%) had degenerated gastric glands. A gastric gland hyperplasia was noticed in 50% of the animals.

Evaluation of the inflammatory infiltrate in the gastroduodenal mucosa samples of the 14 horses showed predominance of a diffuse lymphoplasmacytic inflammatory infiltrate in 85.7% (12/14) of the horses (Fig.2E). The inflammatory infiltrate in the gastroduodenal samples graded from mild to moderate (1-150 cells/field) in five (35.7%) of the analyzed animals; and from

moderate to severe (51 to over 151 cells /field) in seven (50%) of the studied animals. Among these, Equines 3, 6, 8 and 10 (28.6%) also showed mixed, multifocal or focal inflammatory infiltrate in, at least, one mucosal sample of the studied regions. All regions of the individual 13 presented predominance of mononuclear inflammatory infiltrate – from mild to moderate; however, in *margo plicatus* and duodenal samples, there was severe mixed inflammatory infiltrate with the presence of eosinophils in the duodenum. In Equine 7, severe mixed inflammatory

infiltrate was observed in the fragments of the non glandular regions and mild or moderate lymphoplasmacytic infiltrate in those of the glandular regions. Eosinophils were found in the duodenum samples of Equines 13 and 14 (22.2% - 2/9), in the pylorus samples of Equine 10 (7.1% - 1/14) and in the non glandular fundus samples of Equine 5 (7.1% - 1/14). Intraepithelial infiltration of inflammatory cells, was observed in 78.6% of the evaluated animals (11/14), in at least one of the gastroduodenal mucosa samples.

Table 1 – Lesion grading of the gastroduodenal mucosa lesions of Thoroughbred race horses according to the Sydney System (n=14). Lesions were graded as: 0 – absent; 1 – mild; 2 – moderate; e, 3 - severe.

Horse	Lesions of the Gastroduodenal Mucosa					
	Edema	Erythema	Hemorrhage	Friability	Granularity	Thickening
1	2	3	0	0	0	1
2	1	1	1	0	0	2
3	2	2	3	0	1	1
4	2	2	2	0	0	2
5	0	1	0	0	0	1
6	2	3	3	0	0	0
7	1	0	1	2	1	0
8	2	2	1	2	2	0
9	2	3	2	0	3	2
10	2	2	0	2	3	2
11	2	2	1	0	0	1
12	1	2	2	0	0	3
13	1	1	2	2	2	0
14	2	2	2	1	1	2

Table 2 – Equine Gastric Ulcer Syndrome Council Grading and evolution time of Thoroughbred race horses gastric lesions (n=14).

Animal	EGUS grading	Lesions (ulcer/erosion)
1	2	Chronic
2	2	Chronic
3	2	Chronic
4	3	Acute
5	1	Chronic
6	3	Acute
7	4	Acute
8	3	Acute
9	2	Chronic
10	2	Chronic
11	2	Chronic
12	3	Chronic
13	2	Chronic
14	3	Chronic and acute

Lymphoid follicles were present in at least one sample of all the studied animals except one. All animals had lymphoid clusters in the gastric and duodenal samples.

According to the histopathological evaluation, the most common gastritis was chronic with predominance of

lymphocytes and plasmocytes. Horses number 3, 6, 7, 8, 10 and 13 (42.9%) had active chronic gastritis - inflammatory infiltrate with predominance of mononuclear cells and substantial foci of polymorphonuclear cells. Equine 7 showed extensive necrotic areas in the non

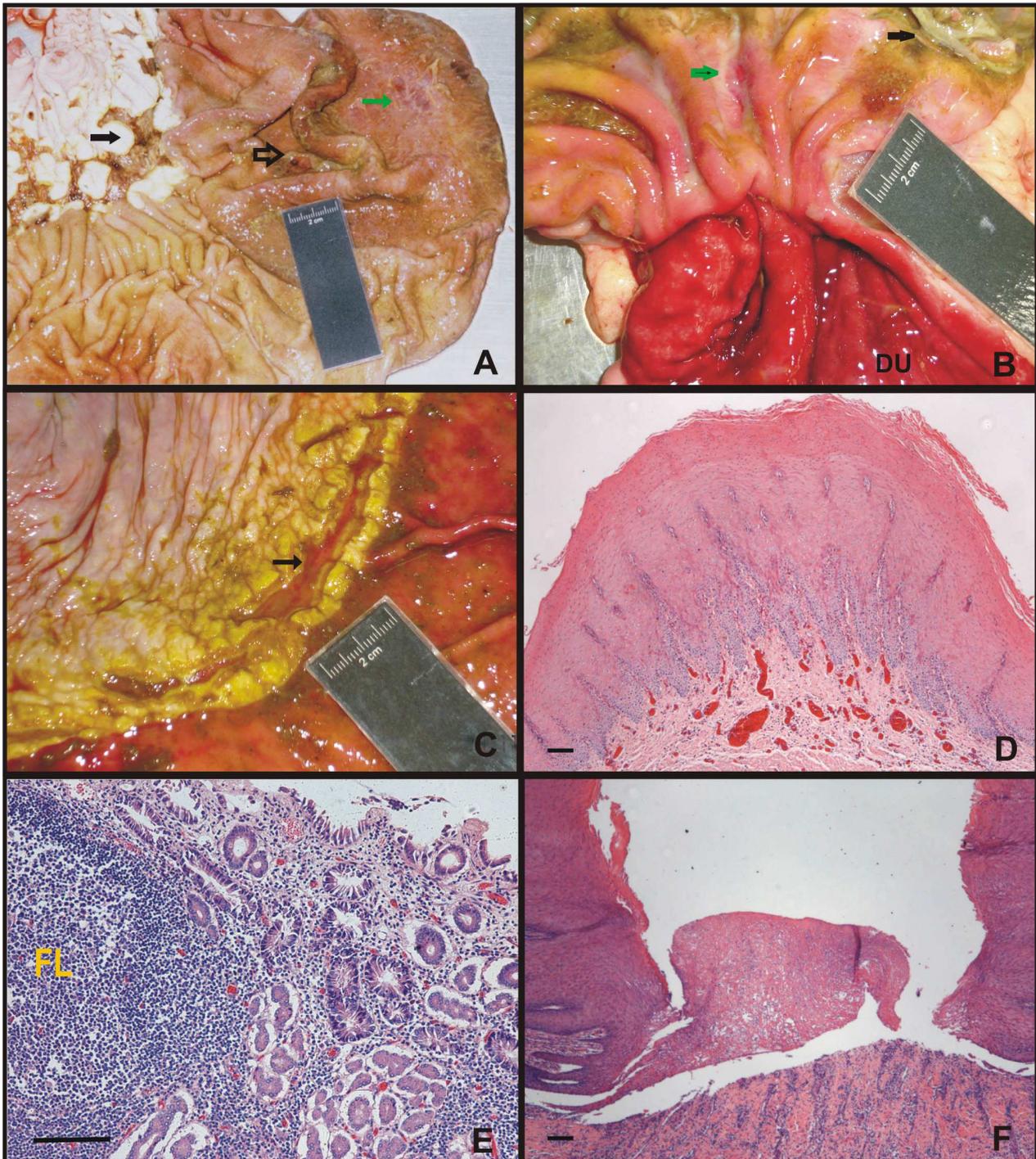


Figure 2. Macroscopic aspects of the stomach and anterior duodenum of Thoroughbred race horses: (A) Equine number 8: non glandular fundus with severe ulceration (full black arrow) and glandular region with hypertrophy of the gastric rugae (green arrow) and fibrinous exudate (black empty arrow). (B) Equine number 14: antrum region with erosions partially covered by fibrinous exudate (green arrow) and fibrinous exudate covering the mucosa (black arrow); duodenum –DU– Hyperemic with irregular mucosa. (C) Equine number 12: ulcerated *margo plicatus* (arrow) and squamous epithelium thickening around the ulceration. (D) Equine number 5 - non glandular fundus with hyperparakeratosis, acanthosis and vascular congestion. (E) Equine number 14 - duodenum: severe mononuclear duodenitis, notice a lymphoid follicle (FL) and abundant inflammatory infiltrate. (F) Equine number 10 - *margo plicatus*: ulceration of the squamous epithelium. D-F: HE. Bar: 100µm.

glandular region characterizing severe acute necrotizing gastritis in the non glandular region with mild chronic gastritis in the glandular region.

Microscopic analysis of duodenal samples (n= 9) revealed a severe chronic duodenitis in 33.3% of the horses (Equines 9, 13 and 14) (Fig.2E); a severe

The *margo plicatus* showed ulceration in 78.6% of the samples (Fig.2F). The duodenum presented ulceration in 33.3% of the animals (3/9), the glandular fundus in 28.5% and the pylorus in 7.1%. Erosions were seen in the samples of the glandular fundus of 85.7% of the animals, in the antrum samples of 78.2% of the animals and in the *margo plicatus* of 71.4% of the animals. Erosions were observed in the pylorus fragments of 71.4% of the horses; in the non glandular fundus of 42.9%; and in the duodenum of 55.5% respectively.

Discussion

The use of a grading system facilitates comparisons between different research and clinical groups (6). Thus, in this study it was carried out an association of one of the most used methods for gastric ulcer evaluation in equines, the EGUS Council classification, and the gastritis classification, the Updated Sydney System, applied by human pathologists.

The Updated Sydney System is a classification of gastritis used in humans that has the merit of integrating the description of the lesions observed by endoscopy with histopathological interpretation through systematic biopsies (24). It was chosen for having already been established and used in the routine human gastric pathology, and for being used in scientific studies in Veterinary Medicine of dogs, also bringing an important detailing of the macroscopic as well as the microscopic aspects of the disease. EGUS Council grading was chosen for being broadly used and for having its easy use, reproducibility and correlation among different observers demonstrated (6).

The association of the two classification methods enabled a better understanding of gastroduodenitis and gastroduodenal ulcerations, once it describes minutely the gross and microscopic alterations of the gastric and duodenal mucosae, allowing their grading. With the utilization of classification systems, the dialog among physicians, endoscopists and pathologists becomes facilitated by the uniformity of terms and correlation among the obtained data. This allows a better interaction among similar professionals and promotes scientific research reproducibility, therefore contributing to a better understanding of the infirmity and to the animal welfare once the diagnosis is more precise and reliable, the treatment will be correct and the prognosis accurately defined.

lymphoplasmacytic chronic duodenitis in 33.3% (Equines 4, 8 and 11), and a moderate lymphoplasmacytic chronic duodenitis in 33.3% (Equines 5, 6 and 10). The inflammatory infiltrate was predominantly located close to the surface of the mucosa.

During macroscopic evaluation by the Updated Sydney System, the study of the lumen discarded the possibility of partial or total stomach and duodenum obstruction, and provided a good parasitic management of the animals. Lumen alterations found in most of the horses (mucus in 7 animals and fluid in 13) are important to determine the secretory mucosal state and can be correlated with specific diseases, as for instance, the *Ménétrier* disease in humans characterized by excessive mucus or gastrinoma that generates abundant gastric secretion (12).

This study revealed that edema, erythema, hemorrhage, friability, granularity, fibrinous exudate, and thickening of the non glandular epithelium were common macroscopic findings in the stomach. According to Castro et al. (1993), these listed descriptive terms are considered inflammatory endoscopic characteristics just as gastric rugae hyperplasia and submucosa vein visualization because the normal mucosa shows uniform and brilliant pink gastric coating, without adherent exudate, with rugae of maximum 5mm in the body and antrum almost smooth.

Macroscopic analysis according to the Updated Sydney System yet showed that 100% of the animals of the present study had gastric ulcer or erosion, 92.9% in the non glandular region and 21.4% in the glandular region; which were mainly adjacent to the *margo plicatus*. This area seems to be the most dynamic of the glandular portion and it is also the most exposed to the gastric acid (23), thus being described as the most targeted by gastric ulcers (8, 16, 35). In the present study, the *margo plicatus* showed a high percentage of lesions. That is in accordance with the location and frequency of lesions in racehorses already described (80 to 90%) (5, 27, 34, 36). In Brazilian rodeo horses, Buonora et al (2004) found up to two ulcers in 14.29% of the Brazilian rodeo animals and none of the animals had over three ulcers. In endurance equines the ulceration hits 67% of the individuals, 57% of them with lesions on the squamous mucosa and 27% with hemorrhage in the glandular mucosa (33).

The percentage of lesions on the glandular mucosa of the studied racing horses (21.4%) was smaller than in the endurance equines (27%) (33) and in the horses evaluated endoscopically by Murray et al. (2001). These authors observed 58% of erosions or ulcers in the antrum and pylorus.

In the present study, morphology compatible with duodenum ulceration has not been observed. In spite of that, the organ showed hyperemia and mucosa irregular appearance in six equines (6/9 – 66.6%), characteristics

also seen in 16 horses of several breeds in an endoscopic study carried out by Murray et al. (2001).

The enanthematous endoscopic gastritis is the most frequent type found in humans and was observed in 50% of the horses in the present study. Its features are reddish plaques, finely nodulous mucosa surface, loss of the normal mucosa brightness, punctiform exudate and occasionally, mild friability (12). Based on the Sidney System, erosive gastritis was diagnosed in 21.4% of the horses. Erosions are the main change and they can be coated by an exudate layer and associate to focal enanthema (12). Finally, hemorrhagic gastritis was identified in 2.4% of the animals. It was characterized by hemorrhage in the lumen of the stomach, being graded based on the number of hemorrhagic spots (12). In this study, predominance of gastritis with moderate to severe grading over mild gastritis was demonstrated. In an endoscopic study performed on Brazilian rodeo horses, normal mucosa was observed in 37.14%, non erosive gastritis in 32.86%, erosive gastritis in 15.71%, and ulceration (up to two ulcers) in 14.29% (11). Although both sports, the Brazilian rodeo and horseracing, involve high speeds and extreme physical demand, a higher percentage of gastritis was observed in this present study, probably because 100% of the studied equines are stalled, with a very long confinement.

Based on the macroscopic characteristics of the gastroduodenal mucosa, 2 was the most frequent EGUS grade (50%) in the equines of this study. Grade 3 was found in 35.7% of the animals. And grades 1 and 4 were found in one animal each or 7.1% of the animals. Grades 2 and 3 were the most frequent. Nicol et al. (2002) used a very similar grading system (variation from 0 to 4 based on the number of lesions and on their severity) on colts with vices (as the habit of chewing the stall box) and normal colts, and noticed that the colts with vices had maximum score 4, and score average of 1.3. The normal colts had maximum score 3 and score average of 0.4 (32). In comparison racehorses present in this study had a higher grading average: 2.43. In a Swedish study, horses undergoing race training had squamous mucosa lesions evaluated macroscopically from grades 0 (no lesions) to 4 (ulcers with hemorrhage or extense ulcerated area), similar to the EGUS grade used in this study. In the Swedish study, 30% of the horses had grade 0.27, 5% grade 1.26, 2% grade 2.11, 2% grade 3, and 5% grade 4 (19). Unlike the Swedish study, in this present study, individuals with grade 0 were not observed. It was possible to observe a tendency of the animals analyzed in this study to score grades 2 and 3 while the equines in the Swedish study showed a tendency to grades 0-2. A higher percentage of ulceration from moderate to severe was also observed in another study with racehorses (34), similar to the results of the present study. However, some authors used a different score to compose with the Updated Sidney System (25).

Based on the histopathological evaluation according to the Updated Sidney System, hyperplasia and

parakeratosis were frequent findings in the non glandular stomach regions, because both are present in normal equines. When they are born, equines have a thin and a little keratinized gastric squamous epithelium, but within a few days, the mucosa becomes hyperplastic and parakeratotic (27). However, hyperkeratosis, dysplasia, excessive basal layer hyperplasia and acanthosis found in the analyzed equines also reveal some kind of alteration of the stratified epithelium. In adult horses, spontaneous gastric lesions occur more frequently in the squamous epithelium than in the glandular mucosa (27, 28). Racehorses, as they increase speed, get their abdominal muscles under tension, thus raising the abdominal pressure and pushing the acid gastric content to the portion of the stomach coated by the squamous epithelium. This long period of exposition to the HCl during the intense exercise, that can be daily, is the reason why the squamous mucosa lesions tend to develop or worsen when the horse is undergoing an intensive training program (22, 30).

Microscopically, following the Sidney System criteria, a fibrin deposit was found on the gastroduodenal mucosa surface of 71.4% of the evaluated animals. This alteration can be associated with the erosions (24). Yet, mild atrophy of the gastric mucosa glands was found in four animals and glandular degeneration was observed in another animal. Atrophy is a common denominator in all pathological processes and it causes serious damage to the mucosa. The loss of glands can originate from the mucosa erosion or ulceration with destruction of the glandular layer or result from a prolonged inflammatory process in which the glands are destroyed (13).

In the histopathological analysis, 50% of the animals had hyperplasia of the gastric mucosa glands. In human beings, this lesion is associated with hypertrophic gastropathy (4).

When the microscopic study of the gastroduodenal mucosa samples was carried out, based on the Sidney System criteria, lymphoid follicles were observed in 92.9% of the animals. That reveals a high percentage of this alteration which consists of lymphoid aggregates with germinal centers (13).

It is important to point out that an infiltrate within the *lamina propria* of a maximum of 2 to 5 lymphocytes, plasmocytes and macrophages under great magnification (400X) is considered normal. The presence of plasmocytes is an especially important indicator of the chronic inflammatory response (13). According to the Updated Sidney System classification, a prevalence of the diffuse lymphoplasmacytic inflammatory infiltrate was observed in 12 animals, characterizing lymphoplasmacytic gastritis. One animal had an inflammatory infiltrate composed of lymphocytes, plasmocytes and macrophages. Therefore, 13 animals had chronic gastritis, but five of them presented mixed inflammatory infiltrate associated with erosions and ulcers, being diagnosed with active chronic gastritis. Equine 7 was studied separately for showing extensive necrotic areas in the non glandular region, confirmed by

histopathology; a severe acute necrotizing gastritis in the non glandular region and a mild chronic gastritis of the glandular region were diagnosed. This different diagnosis between the glandular and non glandular regions comes, according to Murray (2003), from differing pathogenesis within both anatomical sites. It is worth emphasizing that all the samples with multifocal or focal mixed inflammatory infiltrate, also had ulcers or erosions.

Following the evaluation criteria of the Updated Sydney System histopathological division, the region most affected by ulcers was the *margo plicatus* with 78.5% of the animals affected, followed by the duodenum (33.3%), the non glandular fundus (21.4%), and the pylorus (7.1%). The erosions were more frequent in the non glandular fundus (85.7%), followed by the antrum (71.4%), the *margo plicatus* (71.4%), pylorus (57.1%), duodenum (55.5%) and glandular fundus (42.9%). The glandular region was the most affected by erosions while the non glandular region was the most affected by ulcers.

Two factors that apparently contribute to the squamous gastric mucosa ulceration are the intensity of training and the alimentary management (29). It is well documented that a hard training program is associated with high percentage of gastric lesions in horses; however, it has not been possible yet to differentiate the interrelated effects of the alimentary constituents, the alimentary management and behavior, and the effects of exercise in gastric physiology involving the peptic infirmity (30). However, it was recently observed that the percentage and severity of gastric ulcers are not determined only by the intensity of training and competitions, but also by the confinement period (11).

In the present study, only one animal had histopathologically proved ulcer in the glandular mucosa, confirming a smaller frequency than in the Andrews et al. (2002) study, in which 6 in 23 equines had the lesion. Yet, it is worth mentioning the large number of animals with glandular mucosa erosions, alteration that can be unnoticed in the macroscopic analysis as seen in previous studies (2). According to Murray (2003), the greatest part of the equine glandular mucosa lesions occurs in the antrum and pylorus mucosa, because of a mucosal barrier lesion or by bile action. The periodic retrograde reflux of the duodenal content into the stomach is a common finding in humans and equines, with biliary concentration generally higher in the stomach antrum (30).

Microscopic lesions in the duodenal mucosa were observed in 88.8% indicating a high percentage of duodenal affections. The duodenal ulcer was seen in 33.3% of the animals aged 120 days, 48 and 36 months. However, Murray (1998) stated that the duodenal lesions affect more colts aged from 2 days to 12 months. All animals in which the duodenum was evaluated, duodenitis was observed with predominance of lymphoplasmacytic infiltrate. It can be concluded that many horses undergoing race training are affected by gastroduodenal lesions mainly ulcers and that the utilization of the Sidney System in

association with the EGUS Council classification proved to be feasible and enabled a more detailed study of the equine gastroduodenal lesions. Both classifications, when used together, complement each other, and can make the reproducibility of studies in the subject possible, once they try to diminish at the most the subjectivity of the evaluation, and standardize terms. These characteristics of the association of both classifications encourage their future use in scientific studies leading to a better understanding of these lesions and also enable appropriate diagnosis and treatment to promote a better quality of life of the affected horses. It is important to point out that these affections, although usually neglected, can cause abdominal pain lowering the performance of athlete horses, so further investigations of these lesions and their causes are necessary.

Acknowledgments

This research was supported by Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq), República Federativa do Brasil.

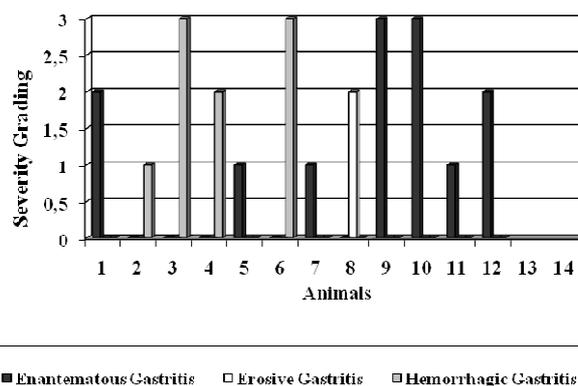


Figure 3. Gastritis classification based on the endoscopic division of the Updated Sidney System of Thoroughbred race horses (n=14). Identification of the gastritis type (enantematous, erosive and hemorrhagic) and its grading. Consider: 0 = absent, 1 = mild, 2 = moderate, 3 = severe.

References

- ANDREWS F.M.. Ulcers in the stomach and colon, diagnosis and treatment: A pain in the gut! Focus Meeting, American Association of Equine Practitioners, 2005 (<http://www.ivis.org>)
- ANDREWS F.M., REINEMEYER C.R., MCCracken M.D., BLACKFORD J.T., NADEAU J.A., SAABYE L., SÖTELL M., SAXTON A.. Comparison of endoscopic, necropsy and histology scoring of equine gastric ulcers. **Equine Vet. J.**, 2002, 34 (5), 475-8.
- ANDREWS F., BERNARD W., BYARS D., COHEN N., DIVERS T. & PIPERS F. Recommendations for the diagnosis and treatment of Equine Gastric Ulcer Syndrome (EGUS). **Equine Vet. Educ.**, 1999, 1, 2, 122-134.b
- BAYERDÖRFFER E., RITTER M.M., HATZ R., BROOKS W., RUCKDESCHEL G. & STOLTE M. Healing of protein losing

- hypertrophic gastropathy by eradication of *Helicobacter pylori* is a pathogenic factor in Ménétrier disease? *Gut*, 1994, 35, 701-704.
5. BEGG L.M. & O'SULLIVAN C.B. The Prevalence and distribution of gastric ulceration in 345 racehorses. *Aust. Vet. J.*, 2003, 81 (4), 199-201.
 6. BELL R.J., KINGSTON J.K., MOGG T.D. A comparison of two scoring systems for endoscopic grading of gastric ulceration in horses. *N. Z. Vet. J.*, 2007, 55 (1), 19-22.
 7. BERTONE J.B.. Medical diseases of the lower alimentary tract. KOBLUK C.N., AMES T.R. & GEOR R.J. Eds. **The Horse: Diseases and clinical management.** Philadelphia: Saunders. 1995: 315-8.
 8. BRASHIER M.K., GEOR J.R. Gastroduodenal ulcer disease. KOBLUK C.N., AMES T.R. & GEOR R.J. Eds. **The Horse: Diseases and clinical management.** Philadelphia: Saunders. 1995: 315-8.
 9. BRICEÑO A.M, GONZÁLES F.G., LEÓN, D.V., PULGAR E., VELÁSQUES S., CÉLIX J., GONZÁLES G., BLANCO P. Colic and Gastric Rupture in a Captive Zebra (*Equus quagga*). *Braz. J. Vet. Pathol.*, 2011, 4(3), 183.
 10. BULLIMORE S.R., CORFIELD A.P., HICKS S.J., GOODAL C. & CARRINGTON S.D. Surface mucus in the non-glandular region of the equine stomach. *Res. Vet. Sci.*, 2001, 70, 149-155.
 11. BUONORA G.S., BASTOS AFONSO, J.A., ALMEIDA H.B., SILVEIRA ALVES, G.E.. Estudo da ocorrência de lesões gástricas em cavalos de vaquejada: Resultados preliminares. *Braz. J. Vet. Res. Anim. Sci.*, 2004, 14 (supl.), 263-264.
 12. CASTRO P.C., OLIVEIRA C.A., ANDRADE J.M.. Gastritis. DANI R., CASTRO, P.C. Eds. **Gastroenterologia Clínica.** Rio de Janeiro: Guanabara Koogan, 1993: 562-581.
 13. DIXON M.F., GENTA R.M., YARDLEY J.H., CORREA P. Classification and grading of gastritis: The updated Sydney System. *Am. J. Surg. Pathol.*, 1996, 20 (10), 1161-1181.
 14. DORE M.P., BILOTTA M., VAIRA D., MANCA A., MASSARELLI G., LEANDRO G., ATZEI A., PISANU G., GRAHAM D. Y., REALDI G. High Prevalence of *Helicobacter pylori* Infection in Shepherds. *Dig. Dis. Sci.*, 1999, 44 (6), 1161-4.
 15. FENGER C.K. Gastroduodenal ulceration. REED S.M., BAYLY W.M. Eds. **Equine Internal Medicine.** Philadelphia: Saunders. 1998: 960-961.
 16. FERNANDES W.R., BELLI C.B., SILVA L.C.L.C. Achados gastroscópicos em eqüinos adultos assintomáticos. *Arq. Bras. Med. Vet. Zootec.*, 2003, 55 (4), 405-410.
 17. FIALHO S.S., NOGUEIRA G. M., DUARTE C. A., NETO A.O.P., MACORIS D. G. Casearia sylvestris na permeabilidade gástrica à sacarose em eqüinos submetidos a protocolo de indução de úlcera gástrica. *Ciência Rural*, 2010, 40 (2), 348-355.
 18. JEUNE S.S., NIETO J.E., DECHANT J.E., SNYDER J.R.. Prevalence of gastric ulcers in Thoroughbred broodmares in pasture: A preliminary report. *Vet. J.*, 2009, 181 (3), 251-255.
 19. JONSSON H., EGENVALL A.. Prevalence of gastric ulceration in Swedish standardbreds in race training. *Equine Vet. J.*, 2006, 38 (3), 209-213.
 20. LEWIS L.D.. **Nutrição Clínica Eqüina: Alimentação e cuidados.** São Paulo: Roca, 2000.
 21. LIU C., CRAWFORD J.M. The Gastrointestinal tract: Stomach. KUMAR, V., ABBAS, A.K. & FAUSTO, N. Eds. **Robbins and Cotran, Pathologic Basis of Disease.** Philadelphia: Elsevier Saunders., 2004: 810-827.
 22. LORENZO-FIGUERAS M., MERRITT A. M. Effects of exercise on gastric volume and pH in the proximal portion of the stomach of horses. *Am. J. Vet. Res.*, 2002, 63 (11), 1481-1487.
 23. MACALLISTER C.G., MORGAN S.J., BORNE A.T., POLLET R.A. Comparison of Adverse Effects of Phenylbutazone, Flunixin Meglumine, and Ketoprofen in horses. *J. Am. Vet. Med. Assoc.*, 1993, 202 (1), 71-7.
 24. MAINGUET P., JOURET A., HAOT J. Le Sydney System, nouvelle classification des gastrites: Application pratique. **Gastroenterol. Clin. Biol.**, 1993, 17 (tires à part), T13-T17.
 25. MARTINEAU H., THOMPSON H., TAYLOR D. Pathology of gastritis and gastric ulceration in the horse. Part 2: A scoring system. *Equine Vet. J.*, 2009, 41 (7), 646-651.
 26. MURRAY M.J. Ulceração gástrica. SMITH B.P. Eds. **Tratado de Medicina Interna de Grandes Animais.** São Paulo: Manole. 1993: 655-9.
 27. MURRAY M.J. Gastroduodenal ulceration. Robinson N.E. Eds. **Current Therapy in Equine Medicine.** Philadelphia: Saunders. 1997a: 191-7.
 28. MURRAY M.J. Foal stomach and duodenum. Traub-Dargatz J.L. & Brown C.M. Eds. **Equine Endoscopy.** St Louis: Mosby. 1997b:159-171.
 29. MURRAY M.J. Gastroduodenal ulceration. In: REED, S.M & BAYLY, W.M. (ed.), **Equine Internal Medicine.** Philadelphia: Saunders. 1998:615-23.
 30. MURRAY M.J. Equine gastric ulcer syndrome: Pathophysiology and risk factors. *Anais I SIMEQ* (Simpósio Merial de Equinocultura), Campinas, SP, 2003.
 31. MURRAY M.J., NOUT Y.S., WARD D.L. Endoscopic findings of the gastric antrum and pylorus in horses: 162 cases (1996-2000). *J. Vet. Intern. Med.*, 2001, 15 (4), 401-406.
 32. NICOL C.J., WATERS A.J., WILSON A.D., DAVIDSON H.P.D., HARRIS P.A. Study of crib-biting and gastric inflammation and ulceration in young horses. *Vet. Rec.*, 2002, 151, 658-662.
 33. NIETO J.E., SNYDER J.R., BELDOMENICO P., ALEMÁN M., KERRC J.W., SPIERB S.J. Prevalence of gastric ulcers in endurance horses: A preliminary report. *Vet. J.*, 2004, 167(1), 33-37
 34. RABUFFO T.S., ORSINI J. A., SULLIVAN E., ENIGLES J., NORMAN T., BOSTON R. Associations between age or sex and prevalence of gastric ulceration in standardbred racehorses in training. *J. Am. Vet. Med. Assoc.*, 2002, 221(8), 1156-1159
 35. ROY M.A., VRINS A., BEAUCHAMP G., DOUCET M.Y. Prevalence of ulcers of the squamous gastric mucosa in standardbred horses. *J. Vet. Intern. Med.*, 2005, 19 (5), 744-750.
 36. VATISTAS N.J., SNYDER J.R., CARLSON G., JOHNSON B., ARTHUR R.M., THURMOND M., ZHOU H., LLOYD K.L. Cross-sectional study of gastric ulcers of the squamous mucosa in thoroughbred racehorses. *Equine Vet. J.*, 1999, 29 (suppl.), 34-39
 37. VIDELA R., ANDREWS F.M. New perspectives in equine gastric ulcer syndrome. *Vet. Clin. North Am. Equine Pract.*, 2009, 25, 283-301.
 38. WILSON J.H. Gastrointestinal problems in foals-gastrointestinal ulcers. In: Robinson N.E. Ed. **Current Therapy in Equine Medicine.** Philadelphia: Saunders, 1987: 239-241.