Gastroscopic evaluation of gastric ulcer syndrome in sport horses with poor performance

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Abstract: Gastric ulceration is common in performance horses. More than 90% of thoroughbred horses in the training season and approximately 60% of racing horses from other disciplines have gastric ulcers. Excessive secretion of gastric acid, impairment of gastric mucosal blood flow, weakening of prostaglandin E or the gastric mucus/bicarbonate layer, and gastric emptying disorders are important factors in the onset of ulceration. In this study, 12 horses with poor performance were evaluated using gastroscopy following clinical examinations. Gastroscopy revealed ulceration in 5 horses and ulcerative lesions in 7. A daily dose of 4 mg/kg omeprazole was administered orally for 4 weeks in all horses with clinically suspected gastritis, with or without gastric lesions. At the end of this time, a protective dose of 2 mg/kg per day of omeprazole was used for an additional 2 weeks. No rest for the horses was advised during the medical treatment period. According to the histories obtained from riders and trainers, all horses showed improved performance during the second week of treatment. No gastric lesions were detected at endoscopic examination following medical treatment of horses with gastric ulcerative lesions. In conclusion, gastric ulcer syndrome with or without gastric lesions in sport horses is a significant problem causing poor performance. This is corroborated by the improvement in performances following medical treatment.

Key words: Horse, gastritis, gastric ulcer, gastroscopy

1. Introduction
Gastric ulcers are erosions that form on the wall of the stomach. Gastric ulceration is very common in performance horses and the most common stomach problem in horses generally. Over 90% of thoroughbreds during the training period and approximately 60% of horses competing and working in other disciplines have gastric ulcers. It is also a significant problem in foals (1,2).

Ulcers usually form close to the margo plicatus on the squamous part of the stomach with no protective mucus layer on its surface. However, they can also form on the glandular part of the stomach. Clinical signs in foals with gastric ulcers vary, depending on which part of the stomach the lesion is located in. Foals may not exhibit symptoms, or they may exhibit growth retardation, rough hair in their coats, teeth grinding, excessive salivation, intermittent colic, lying down more than normal, and sometimes diarrhea (3). Clinical signs in adult horses are usually anorexia and chronic intermittent pain of different intensities. Other clinical findings are poor or lower performance than expected, decreased body condition, poor hair appearance, and a reduction in the consumption of concentrated feed. Signs of pain in some horses with gastric ulcers occur after eating. Many horses may appear clinically normal at endoscopic examination (2–4).

Acid is continuously secreted in the horse stomach. In nature, a horse pastures for approximately 16 h a day. This forage, and also the bicarbonate in saliva, reduces acidity. If horses are housed in stables, however, and fed high-concentrate diets with only limited access to hay and grazing, the acidity in the stomach increases. The greater the time without forage, due to type of diet or to illness, the higher the stomach acid levels and the risk of gastric ulcers (1).

Intensive training and racing lead to an increased incidence of gastric ulcers. Intense exercise is known to have numerous negative effects on the physiology of the stomach. These include reduction of blood flow to the stomach, an increase in gastric acid secretion, and delayed emptying of stomach contents into the duodenum (1,5,6).

Administration of nonsteroidal antiinflammatory drugs, such as flunixin, meglumine, or phenylbutazone,
increases the risk of gastric ulceration, especially in the glandular stomach. These types of drugs reduce blood flow to the inner part of the stomach, increase acid secretion, and disrupt the protective mucus barrier with their bicarbonate characteristic (1).

The purpose of this study was to show, with clinical and gastroscopic examinations and positive results obtained after medical treatment, that gastric ulcer syndrome can cause poor performance during the training period.

2. Materials and methods

2.1. Case selection

Twelve horses (8 females, 4 males) of different breeds, ages, and sexes, which were in the training period and exhibiting poorer performance than expected, were included in the study (Table). Medical histories were taken from hostlers and riders before examination. In addition, complete medical histories of the horses and the medications they received were also evaluated.

2.2. Clinical examination

Resting pulse, respiratory rate, and body temperature (37.5 to 38.5 °C) were taken, and abdominal auscultation was performed. Blood samples were collected for complete blood and biochemical analysis (albumin, alkaline phosphatase, alanine transaminase, amylase, blood urea nitrogen, calcium, creatinine, globulin, glucose, potassium, sodium, phosphate, total bilirubin, and total protein) in order to exclude any other diseases. Condition during training was also evaluated. Clinical examinations were repeated in the 2nd and 4th weeks.

2.3. Gastroscopic examination

Before starting the process, all horses were fasted for at least 12 h. Sedation was achieved with the application of detomidine HCl (Domosedan®, Pfizer, 0.02 mg/kg i.v.). A 325-cm-long, 1.3-mm-diameter gastroscope (Storz model PV – G 34 – 325, flexible video endoscope, Karl Storz GmbH & Co. KG) was inserted into the nostril and pushed forward until reaching the stomach. In the meantime, the pharynx and larynx were examined for possible structural and functional abnormalities. The stomach was evaluated from the cardia to the pylorus (squamous mucosa, margo plicatus, glandular mucosa). The end of the endoscope was set in a retroflex position for examination of the cardia. For examination of the initial portion of the duodenum and stomach up to that point, the tip of the endoscope was pushed forward in the direction of the curvatura major. The ulcers detected in horses were scored from 0 to 3, according to the defined classification for gastric ulcers (4,7). Accordingly, 0: stomach with normal mucosal surface without ulceration, 1: minor ulceration in the form of a single erosion or ulcer or a few small lesions, 2: a single ulcer or more than one ulcer with a large surface area, 3: deeper and bleeding multiple ulcers covering a large surface area or deep and multifocal ulcers in a combined structure.

After medical treatment, a second gastroscopic examination was performed in the sixth week in order to evaluate the appearance of the lesions.

2.4. Medical treatment

Omeprazole in paste form (E – gastro®, BAVET) at a dose of 4 mg/kg was administered orally once a day for 4 weeks to all horses 30–60 min before the first morning feed. At the end of this period, 2 mg/kg omeprazole continued to be used as a protective dose for 2 weeks. During medical treatment, horses continued to train and the amount of roughage in their rations was doubled.

3. Results

Records kept for the horses revealed no gastritis-related complaint or medical treatment administered for such. Nonsteroidal antiinflammatory drugs had been administered (for 3 to 7 days) to all horses in the previous 8 to 12 months due to simple lameness. In addition, infection-related hepatitis was treated in 1 horse, this having been concluded 2 months before. Except for vitamin mineral supplements, the horses had received no medication in the previous 2 months.

Evaluation of the nutritional condition of the horses revealed that they consumed insufficient amounts (0.5 kg/100 kg) of roughage, but a normal amount (1 kg/100 kg) of concentrated feed. Horses were kept in their boxes when not training and were not able to graze.

Histories of the horses taken from riders revealed that the animals exhibited much poorer training performances than previously, but that there was no abnormality in their respiratory sounds. Anamnesis taken from the hostlers revealed that the amount of concentrated feed consumed at every meal was low, food was becoming wet because of excessive saliva secretion, and horses exhibited symptoms such as loss of appetite, constant sleepiness, and teeth grinding (Table). Some horses also showed signs of mild colic, but the symptoms disappeared by themselves without any treatment.

No abnormal condition was determined during clinical examination and complete blood and blood biochemical tests. No abnormal respiratory sounds were identified in the assessments made during training. On the basis of clinical examination and anamnesis, it was concluded that the horses were having gastric problems, and gastroscopic evaluation was performed.

No abnormal condition was determined at endoscopic examinations of the pharynx and larynx before gastroscopy. Gastroscopic evaluations revealed no ulcerative lesions (degree 0) in the stomach in 5 horses (Figure 1a). Two to 3 small, superficial lesions (Figure 1b), localized away
from the margo plicatus in the squamous part of the stomach (degree 1), were seen in 1 horse and 1 or 2 small, superficial lesions (degree 1) localized close to the margo plicatus in the squamous part of the stomach were detected in 6 (Figure 2). There were no lesions in other parts of the stomach.

No abnormal condition was determined at clinical examinations performed in the second week during medical treatment. Moreover, the conditions identified at anamnesis, such as poor appetite, excessive salivation, excessive drowsiness, and teeth grinding, disappeared within 2–3 days after starting medical treatment. All horses

### Table. Breed, age and sex, and gastroscopic and clinical findings of horses with poor performance.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Breed</th>
<th>Age and sex</th>
<th>Gastroscopic findings</th>
<th>Clinical findings*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Thoroughbred</td>
<td>5-year-old gelding</td>
<td>1st degree</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>Westphalian</td>
<td>9-year-old mare</td>
<td>0</td>
<td>+</td>
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<tr>
<td>3</td>
<td>Half-blood Thoroughbred</td>
<td>6-year-old mare</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>Half-blood Thoroughbred</td>
<td>12-year-old mare</td>
<td>1st degree</td>
<td>+</td>
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<tr>
<td>5</td>
<td>Half-blood Thoroughbred</td>
<td>10-year-old mare</td>
<td>1st degree</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>Hanoverian</td>
<td>13-year-old gelding</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>Half-blood Holsteiner</td>
<td>5-year-old mare</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
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<td>5-year-old mare</td>
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<td>+</td>
</tr>
<tr>
<td>9</td>
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<td>9-year-old mare</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td>10</td>
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<td>+</td>
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<tr>
<td>11</td>
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<td>1st degree</td>
<td>+</td>
</tr>
<tr>
<td>12</td>
<td>Half-blood Thoroughbred</td>
<td>9-year-old mare</td>
<td>1st degree</td>
<td>+</td>
</tr>
</tbody>
</table>

±: Lesions localized away from the margo plicatus. *: Clinical findings based on anamnesis.

*Figure 1. Normal gastric mucosa (a) and small superficial ulcerative lesions in the squamous part of gastric mucosa away from the margo plicatus (b); first-degree gastric ulceration.*
were reported to have reached the desired performance in the second week. Preexisting lesions were observed to have healed and no ulceration was observed in gastroscopic examinations performed at the sixth week after medical treatment of horses with ulcerative lesions.

4. Discussion

Gastric ulcers in horses generally form on the squamous part of the stomach, which has no protective mucous layer on its surface, and especially close to the margo plicatus. However, they can also be seen in the glandular part of the stomach (1). Ulcerations on the glandular part of the stomach are more often seen in foals under 1 year old than in adult horses. Glandular mucosal ulcerations in adult horses generally emerge as a result of nonsteroidal antiinflammatory drug use (8). All the patients in our study were adult horses aged 5 or above. All lesions determined by gastroscopy were in the squamous part of the stomach and, with one exception, all were close to the margo plicatus. No lesions were detected on the glandular mucosa.

Studies performed with a large number of horses show that males, both stallions and geldings, are affected more than females (9). One study reported the opposite, however (10). In that study, the majority (n = 8) of horses were mares.

Although acid is continuously secreted in stomach, gastric acidity is reduced by roughage and bicarbonate in saliva. Under natural conditions, a horse pastures for approximately 16 h a day, thus obtaining the necessary forage. Roughage is therefore of much greater importance for horses who spend a significant part of their lives in boxes without grazing. Under these conditions, acidity in the stomach increases, especially if the horses are given concentrated feed. In the event of forage malnutrition, the level of acid in the stomach and the risk of gastric ulcer increase (1,3,8). It is probable, therefore, that lack of grazing and insufficient amounts of hay have negative effects on the stomach.

Stress caused by intense training and racing is known to have many adverse effects on the physiology of the stomach, such as reduction of blood flow to the stomach, increased acid secretion, and delayed emptying of the stomach contents to the duodenum. The incidence of gastric ulcers is therefore high in horses during periods of intensive training (1,4). In addition, some studies have shown an increased incidence and severity of gastric ulcers depending on the duration of the training period or racing (5,6). In this study, the horses were in an intensive training season and exhibited a lower performance than that expected. In accordance with the literature, this suggested the possibility of gastritis. The findings of the clinical evaluation and the positive response to the medical treatment confirmed this diagnosis.

Gastric ulcer syndrome is diagnosed on the basis of clinical symptoms and results obtained after treatment if endoscopy is not possible. Clinical symptoms in adult horses include inability to consume a meal completely (reduced concentrated feed intake), depression, behavioral changes, poor performance at training and competitions, usually mild but also other severities of chronic intermittent colic, decreased body weight, and a poor coat of hair (3,4). In this study, as well as a decrease in performance, all horses had one or more of the other symptoms of gastritis. That gave rise to a suspicion of gastric ulcers, while the clinical findings related to other systems were normal.

Gastroduodenal ulcer syndrome in horses can be diagnosed on the basis of clinical symptoms, endoscopic findings, and observation of response to treatment. However, no lesions are observed at endoscopic
examination in most horses exhibiting clinical symptoms of it (3). One study reported that without any clinical symptoms, ulcerative lesions were encountered randomly at gastroscopy (11). Although gastroscopy is an effective method for determining lesions in gastric ulcer syndrome, clinical symptoms and results of gastritis treatment should be taken into consideration in order to establish a definitive diagnosis, since not all patients with gastritis need have ulcerative lesions. That lesions were seen in only 7 of the horses with poor performance in the study, despite all horses showing various symptoms of gastric ulcer syndrome, supports this conclusion. Examination revealed no finding of other possible diseases. Horses with or without ulcerative lesions responded positively to the medical treatment administered for gastritis and gastric ulcer syndrome, and they quickly achieved the desired performance level.

Many drugs, with different characteristics, are available for the treatment of gastric ulceration. The main principle of treatment is reducing the secretion of stomach acid to allow healing of the damaged mucosa. For this purpose, proton pump antagonists (omeprazole at 4 mg/kg daily) or H2-receptor antagonists (cimetidine at 20 mg/kg per os 3 times a day or 4.4 mg/kg i.v. 4 times a day, or ranitidine at 6.6 mg/kg per os or i.v. 2 times a day) can be used. Omeprazole in paste form is the most effective drug (12–15). Omeprazole acts by preventing stomach acid secretion and maintains its effect up to 27 h after administration. One dose a day is therefore sufficient. No matter which drug is chosen for treatment, appetite, signs of colic, diarrhea, and other clinical findings should begin to improve within 48 h, and treatment should be continued for at least 28 days (1–3). Abnormal findings in our study based on anamnesis and clinical findings obtained while treatment with omeprazole was continuing disappeared within 2–3 days. The horses reached their desired performance levels in the second week. Ongoing check-ups revealed no further abnormalities. In addition, no lesions were identified in the final endoscopic examination of horses with previous lesions. These facts all demonstrate the effectiveness of treatment with omeprazole.

In conclusion, gastric ulcer syndrome is a significant problem that gives rise to performance impairment in sport horses, whether or not it causes ulcerative lesions on the gastric mucosa. The improvement in horses' performance confirms the validity of the medical treatment. Gastroscopy is the most practical and convenient examination technique for establishing final diagnosis, grading gastritis, and monitoring the recovery period of gastric ulcerative lesions.

References