

Gastric ulceration in Persian Arab horses in Iran: frequency, haematology and biochemistry

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Summary

Gastric endoscopy was performed in the 24 Persian Arab horses from several race training in Tehran and Tabriz for detection of gastric ulcer. Gastric ulcer was evident in 14 Persian Arab horses (58.3%). Ten out of 14 ulcers were in nonglandular region of the stomach. The horses with the history of long term treatment with non-steroidal anti-inflammatory drugs (NSAIDs) had high prevalence of the gastric ulcer in the glandular mucosa. In this study the prevalence of gastric ulceration was higher in horses with active training program than others. Twelve out of 14 (71%) horses with gastric ulcers had a history of active training. The difference of two groups in respect to training was not statistically significant ($P>0.05$). The number of monocytes was significantly lower and concentrations of potassium were significantly higher in horses with gastric ulcer ($P<0.05$). The results of this study showed that the frequency of gastric ulceration in the training Persian Arab horses was relatively high. Further studies are required to evaluate the clinical importance of ulcer in these horses.

Key words: Gastric ulcer, Persian Arab horse, Frequency, Haematology, Biochemistry

Introduction

Equine gastric ulcer syndrome is a complicated and multifactorial problem and the common cause of poor health in mature horses. The clinical findings associated with gastric ulceration are inappetence, loss of body weight, melena, soft faeces, evidence of mild to moderate abdominal pain and decrease in performance in race horses (Murray, 1992; McClure *et al.*, 2005). The prevalence of gastric ulceration in mature horses is variable and reported to be 17% in a population of Swedish horses evaluated at post-mortem, 82-91% in race horse population and 58% in show horses (Murray *et al.*, 1996; McClure *et al.*, 1999; Vatisias *et al.*, 1999; Sandin *et al.*, 2000; Rabuffo *et al.*, 2002). The prevalence of gastric ulcers in Thoroughbred race horses is reported to be between 66 and 93% (Hammond *et al.*, 1986; Murray *et al.*, 1996). Several risk

factors have been identified such as parasites, tumors, gastric phytobezoars, stress, excessive work and more use of NSAIDs (Radostits *et al.*, 2000). Hammond *et al.* (1986) showed a significantly higher prevalence (80%) of gastric ulcers among Thoroughbreds in race training compared with horses that had retired (52%).

The purposes of this study were to estimate the frequency of gastric ulcers, and to determine the risk factors of gastric ulcers in adult Persian Arab horses and also to study haematological and biochemical changes. To the knowledge of the authors, this is the first report of frequency and clinical pathology changes of gastric ulceration in Persian Arab horses.

Materials and Methods

A cross-sectional study was performed on 24 Persian Arab horses aged 5-14 years

(Mean, 8.5; Median 8) in several race training in Tehran and Tabriz. Horse owners were asked to complete a questionnaire prior to endoscopic examination. The questionnaire contained questions on age, gender, and status of training. The horses were classified into two groups on the basis of type and duration of routine training program. Group A (mild) comprised horses having no speed training per week and were habituated to slow walking for 2 h daily. Group B (active) consisted of horses undergoing 2 speed training per week and were habituated to train several hours every day. The horse owners were asked two more questions about medical treatments during the last 4 months and parasitic control programs. Horses that were received anti-parasitic treatment every 45 days were considered as having regular anti-parasitic program and horses that were not administered the drugs within 4 months prior to endoscopy were considered as having no parasitic program.

The person who performed endoscopy was blind to the answers in the questionnaires. Before gastroscopy, horses were fasted for 8 h, and then were sedated with xylazine hydrochloride (Alfasan, Woerden-Netherlands) at a dose of 0.3-0.5 mg/kg. The examination was performed with a 12 mm diameter, 3 m flexible videoscope VFS 300 (Vet Vu, USA).

A complete examination of gastric epithelium was performed including greater curvature, lesser curvature, dorsal fundus with special emphasis on the region closest to *margo plicatus*. Ulcers were scored from 1 to 5, where:

- 1 = no lesion
- 2 = hyperaemia
- 3 = presence of small erosion
- 4 = extensive erosion in mucosa (mild ulceration)
- 5 = deep and extensive ulcer in gastric mucosa (Bayly *et al.*, 2004)

All examinations were performed by the same clinician. Prior to endoscopy, 10 ml of venous blood was taken into plain tube and the blood was allowed to coagulate. Serum was separated by centrifugation (3500 rpm for 10 min). Biochemical constituents including calcium (arsenazo method), inorganic phosphorus (phosphomolybdate

UV method) and magnesium (xylydyl blue-EGTA method) were measured by an autoanalyser RA-1000 (Technicon, USA), and sodium and potassium were determined by a flame photometer PFP7 clinical (Jenway, England). Three ml of obtained venous blood were collected into EDTA-contained tubes and analysed for haematological tests. The haemoglobin and haematocrit were determined by cyanmethemoglobin method and microcentrifugation, respectively. Total plasma protein and fibrinogen were measured by refractometry and refractometry-precipitation methods (Erma, Japan), respectively. Complete blood count (CBC) was also performed by an automated veterinary haematology cell counter (Nihin Kohden, Celltac α , Tokyo, Japan).

Statistical analysis

No or very mild lesions (ulcer scores 1 and 2) were compared with those with mild to severe lesions (ulcers score 3 and above). The former group was termed "without ulcer" and the latter "with ulcer".

The effect of each recorded risk factors was determined using logistic regression for outcome "with ulcer" vs. those "without ulcer". The SAS procedure LOGISTIC was used. Dummy variables created for risk factors; sex (0 for female and 1 for male), age (0 for ≤ 10 and 1 for > 10 -year-old), type of training (0 for mild and 1 for active training), anti-parasitic control program within 4 months before endoscopy (0 for no regular program and 1 for regular program during the last 4 months) and history of NSAID administration before gastroscopy (0 for no usage or administration for ≤ 3 days and 1 for administration for > 3 days within 4 months prior to endoscopy).

Haematological and biochemical data were analysed with non-parametric Mann-Whitney test using PROC NPARIWAY in SAS. P-value less than 0.05 was considered statistically significant.

Results

Gastric ulceration was evident in 14 Persian Arab horses (58.3%). Ulcer scores from examination of the gastric mucosa are shown in Fig. 1. Ten horses (71.4%) were

affected with non-glandular region ulcers and 4 ulcers (28.6%) were in glandular region ($P = 0.1$).

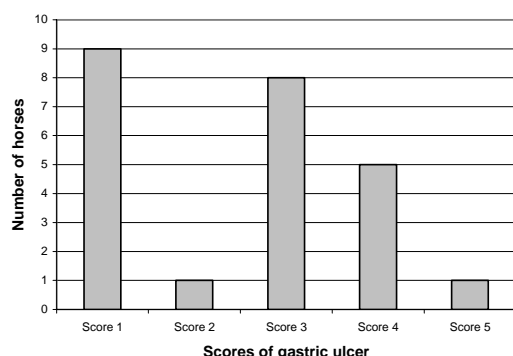


Fig. 1: Distribution of ulcer score in 24 Persian Arab horses in race training

The distribution of the registered variables in the 24 horses is presented in Table 1. Table 2 shows the results of logistic regression analysis. All recorded variables were non-significant. However, the OR for type of training was 6.82 ($P = 0.14$). The NSAID administration had not a significant

effect ($P = 0.87$) on ulceration. The horses with the history of long term treatment with NSAIDs, had high prevalence of the gastric ulcer in the glandular mucosa ($P = 0.23$). Three of four horses with gastric ulcer in the glandular mucosa had the history of long term use of NSAIDs, and only one of them had not been treated with NSAID. On the contrary, three out of 10 horses with gastric ulcer in the non-glandular mucosa were administered NSAIDs.

Comparison of haematological parameters and serum biochemical constituents between two groups revealed that the numbers of monocytes were significantly lower and concentrations of potassium were significantly higher in horses with gastric ulcer (Table 3).

Discussion

The frequency of gastric ulceration was 58.3% in Persian Arab horses in this study. No clinical signs were seen in the horses. The frequency of gastric ulcer in the non-

Table 1: Distribution of possible risk factors as well as number and percentage of 24 Persian Arab horses undergoing gastroscopy

Variable	Total No.	No. of ulcer	% of ulcer
Gender			
Female	12	7	58.3%
Male	12	7	58.3%
Age			
<10-year-old	16	10	62.5%
≥10-year-old	8	4	50%
Type of training			
Mild	7	2	28.5%
Active	17	12	70.6%
Anti-parasitic control (the last 4 months)			
Yes	12	5	41.7%
No	12	9	75%
NSAID administered history			
For ≤3 days	10	6	60%
For >3 days	14	8	57.1%

Table 2: Odds ratio (OR) and 95% confidence interval of risk factors for gastric ulceration in 24 Persian Arab horses

Variable	Estimate	SE	OR ¹	95% CI ²	P-value
Intercept	-0.8430	1.7018			0.62
Sex	0.3952	1.1780	1.48	0.15-14.94	0.74
Age	0.8257	1.6830	2.28	0.08-61.82	0.62
Type of training	1.9202	1.3155	6.82	0.52-89.88	0.14
Anti-parasitic drug	-1.1813	0.9680	0.31	0.05-2.05	0.22
NSAID administration history	0.1576	0.9537	1.17	0.18-7.60	0.87

¹Odds ratio and ²Confidence interval

Table 3: The comparison of haematological and biochemical parameters between horses with and without gastric ulcers, using Mann-Whitney test

Variable	Unit	Without gastric ulcer (No. 10)			With gastric ulcer (No. 14)		
		25	75	75	25	75	75
		Percentile	Median	Percentile	Percentile	Median	Percentile
T. Protein	g/dl	7.1	7.2	7.4	7.20	7.25	7.40
Fibrinogen	mg/dl	200	200	400	200	200	400
PCV	%	35	36.5	39	32	36.5	39
Hb	g/dl	11.7	12.15	13.0	10.7	12.15	13.0
WBC	Cells/ μ l	7500	7750	8200	7500	7850	8300
RBC	10 ⁶ Cells/ μ l	7.4	7.95	8.6	7.6	7.7	8.1
Neutrophil	Cells/ μ l	4662	5014	6308	3684	4658	5760
Lymphocyte	Cells/ μ l	1577	2202	2296	1428	1740	2088
Eosinophil	Cells/ μ l	81	151	225	159	284	415
Monocyte*	Cells/ μ l	222	263	328	102	158	228
Potassium*	mEq/l	3.8	3.95	4.2	4.2	4.3	4.5
Sodium	mEq/l	133	136	141	133	134	138
Magnesium	mg/dl	1.4	2.7	2.8	1.4	1.65	1.90
Phosphorus	mg/dl	2.5	2.7	2.8	2.4	2.8	2.9
Calcium	mg/dl	10.4	10.75	10.90	10.4	11.05	11.50

*Shows significant difference in each row (P<0.05)

glandular region was higher than glandular region (71.4 and 28.6%, respectively). An imbalance between mucosal aggressive factors and mucosal protective factors induce gastric ulceration in horses (Colahan *et al.*, 1999). The glandular region of the stomach has been elaborate mechanisms including the mucus-bicarbonate barrier, prostaglandins, mucosal blood flow and cellular restriction to protect itself from peptic injury. But, the non-glandular region does not have these protective factors; thus, the incidence of gastric ulceration is high in the non-glandular mucosa (Colahan *et al.*, 1999; Radostits *et al.*, 2000). In spite of the higher percentage of gastric ulcers in non-glandular portion in comparison with glandular portion, the difference was non-significant (P = 0.1). The small number of horses was supposed to be the major limiting factor of the study.

It has been reported that the use of some NSAIDs may predispose horses to gastric ulceration (Traub-Dargatz *et al.*, 1988; Goodrich *et al.*, 1998; Bayly *et al.*, 2004). Non-steroidal anti-inflammatory drugs may damage gastric mucosa by inhibition of prostaglandin production. Prostaglandins increase gastric mucosal blood flow and mucus production, and decrease gastric acid production (Roth and Bennett, 1987). Prostaglandins also increase migration of basal cells towards the lumen to repair

mucosal injury (Tarnawski *et al.*, 1985). Seventy-five percent of horses with ulcers in glandular portion had a history of administration of NSAID, but only 30% of horses with ulcers in non-glandular portion had the same history. However this remarkable difference was not statistically significant.

The prevalence of the gastric ulcer in horses depends on the degree of activity and exercise. The training horses were affected higher than the horses in the rest (Colahan *et al.*, 1999; Radostits *et al.*, 2000). In this study, the prevalence of gastric ulceration was higher in horses with active training program than others. Twelve and 2 out of 14 horses with gastric ulcers had a history of active and mild training, respectively. The difference of two groups in respect to training was not statistically significant. However, the power of the study was limited, because of the small number of horses in each group. The increased incidence of gastric ulcers with increasing training level was shown in several studies (Hammond *et al.*, 1986; Murray *et al.*, 1989; McClure *et al.*, 2005). In California, 81% of race horses in active training had gastric ulcers, and poor performance was correlated with increasing ulcer severity (Bayly *et al.*, 2004). Exercise could cause gastric ulceration by delaying gastric emptying and/or increasing gastric acid secretion

(Pagan, 1997; Nadeau and Andrews, 2003). Secretion of gastrin hormone increases with work and exercise, and this hormone induces the production of hydrochloric acid in the stomach and injured the mucosa (Colahan *et al.*, 1999; Radostits *et al.*, 2000; Bayly *et al.*, 2004). In addition, performance horses often go without feed several hours during training days, permitting acid build-up within the stomach, causing more damage (Pagan, 1997).

The prevalence of the gastric lesions in those horses that were administered antihelmintic drugs was low, but not statistically significant. Parasites are one of the risk factors for gastric ulceration in the horses (Nieto *et al.*, 2004; Bayly *et al.*, 2004).

Comparison of haematological parameters and serum biochemical constitutions in horses with and without gastric ulcers revealed that horses with gastric ulcers had significantly ($P < 0.05$) lower number of monocytes and higher concentrations of serum potassium. Cellular breakdown associated with the ulceration could be the source of increased concentrations of potassium in the affected horses. Altered serum levels could lead to metabolic problems that could be potentially significant in racing horses.

It should be noted that the number of monocytes in two groups of horses were at normal range (0-700 cells/ μ l) (Latimer *et al.*, 2003) and the significant difference of monocyte between two groups may be a random significant effect.

We concluded that the frequency of gastric ulceration in the training Persian Arab horses was relatively high. The small number of studied animals is a limiting factor of this study to determine the exact prevalence and significant risk factors. However, the clinical relevance of gastric ulceration in the Persian Arab horses of this study is unclear and further studies are required to evaluate the clinical importance of ulcer in these horses.

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