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Poor Performance Associated with Equine Gastric Ulcer in an Arabian Racehorse

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Summary: An eight year old, Arabian stallion was presented at the Jockey Club (TJK) racecourse hospital with poor performance reported on its last two races. The horse underwent a through clinical examination, which included lameness, endoscopy, gastroscopy, haematology and biochemistry. The only clinically significant was that the horse had grade 4 EGUS (Equine gastric ulcer syndrome) of the squamous mucosa (SM) and glandular mucosa (GM). The horse was consuming 3.08g/kgbw (body weight)/meal of starch and only 40% of the diet was forage when diagnosed with EGUS. The horse was treated medically and by a change of diet. Gastroscopy was repeated monthly until the ulcer lesions were healed. After four months,the horse returned to racing successfully. In conclusion, this case report describes a potential relationship between EGUS and decreased performance in the Arabian racehorse and indicates that EGUS may be an important cause of poor athletic performance in some racehorses. The case report highlights that dietary treatment is just as vital as medical treatment for EGUS. The use of smaller and more frequent meals, lower starch, corn oil, increased forage and extra alfalfa are major recommendations for feeding horses with EGUS.

Key words: Gastric ulcer, horse, poor performance, starch-rich diet

Bir Arap Yarış Atında Equine Gastrik Ülser ile İlişkili Zayıf Performans

Özet: Son iki yarışında zayıf performans gösteren 8 yaşlı bir arap aygırı Jokey Kulubü at hastanesine getirildi. Atın topallık ve endoskopik muayeneyi içeren klinik muayenesi yapıldı ve hematoloji, biyokimya ve gastroskopi için kan örnekleri toplandı. Atta tek önemli klinik bulgu olarak SM(skuamözmukoza) ve GM (glandülermukoza)'da 4. derece EGUS tespit edildi. EGUS tanılı at, 3.08 g/kgVA (vücutağırlığı) nişasta tüketiyordu ve diyetinin sadece %40'ı kaba yem idi. At medikal olarak ve diyeti değiştirilerek tedavi edildi. Ülser iyileşinceye kadar gastroskopi aylık olarak tekrarlandı. Dört ay sonra, at yarışlara başarılı bir şekilde geri döndü. Sonuç olarak, bu olgu sunumu EGUS ve arap yarış atlarındaki performans azalması arasındaki bağlantıyı ayrıntılı olarak gösterdi ve EGUS'un bazı yarış atlarında zayıf atletik performansı için önemli nedeni de olabileceğini gösterdi. Olgu sunumu, EGUS için diyet tedavisinin medikal tedavi kadar önemli olduğunu vurgulamaktadır. Daha küçük ve daha sık öğünler, düşük nişasta, mısır yağı, artan yem ve ekstra yonca kullanımı EGUS'lu atların beslemesi için başlıca önerilerdir.

Anahtar kelimeler: At, gastrik ülser, nişastalı diyet, zayıf performans

Introduction

Equine gastric ulcer syndrome (EGUS) is a common condition affecting racehorses characterized by erosions and ulcers in the terminal esophagus, squamous and glandular

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gastric mucosas and proximal duodenum. Clinical signs of gastric ulceration in adult horses are vague, but may include reduced appetite, ill thrift or weight loss, behavioral changes (depression, aggression or anxiety), and poor performance (10,12). Some horses show no obvious signs except poor athletic performance or aversion to training. However, there is little scientific evidence to confirm this. To date, only three studies have reported a significant association between the presence of gastric ulceration and decreased performance (4,5,15). In some cases this is severe enough to result in retirement from racing. The cause of gastric ulcers is still unknown. The high incidence of EGUS reported in performance horses is likely iatrogenic, related to feeding management, confinement in stables and exercise. A lower prevalence and severity of gastric ulcers occur in horses which are kept on pasture with continuous feed availability than in those which are stabled and fed with high-grain diets. Grain based meals, often interspersed with extended periods of fasting lead to excess gastric acid output without adequate buffering from saliva production. Additionally, production of volatile fatty acids (VFAs) as result of fermentation of grain in the stomach makes the squamous epithelium particularly susceptible to acid damage. This case report highlights to use of combined medical and nutritional management in the treatment of gastric ulceration as a proposed cause of poor performance in Arabian racehorse.

Case Report

An eight year old, Arabian stallion was presented at the TJK racecourse hospital in Istanbul with poor performance reported on its last two races. BW was 415 kg, and body condition score was five out of nine. The clinical history reported that the horse had a good appetite and shiny coat, with slightly higher incidence of yawning than normal. He was also reluctant to stretch out at the gallop during training.

The horse underwent a through clinical examination, which included lameness assessment, resting and dynamic endoscopy of the upper airways (during and post-exercise), gastroscopy, haematology and biochemistry. Prior to gastroscopy the horse was fasted for 24 horses, and water was restricted for four hours. The horse was sedated with detomidine hydrochloride (0.01 mg/kg i.v., Detome Vet®, Ceva Pty Ltd, Australia), and gastroscopy was performed using a 3.2m gastroscope (Teknofocus LG200). The ulcers were graded on a scale zero to four, as defined by the EGUS Council (2):

Grade 0: The epithelium is intact and there is no appearance of hyperaemia or squamous hyperkeratosis.

Grade 1: The mucosa is intact, there are areas of reddening or squamous hyperkeratosis.

Grade 2: Small, single or multifocal lesions.

Grade 3: Large, single or multifocal lesions or extensive superficial lesions.

Grade 4: Extensive lesions with areas of apparent deep ulceration.

The analysis of blood was essentially normal, except for a decreased level of alkaline phosphatase (90 U/l; ref. range 138-251 U/l) and albumin (2.4 g/dl; ref. range 2.7-3.7 g/dl). The only finding of the clinical significance was that the horse had grade 4 EGUS with multiple extensive ulcers of the SM and GM with many appearing deep and coalescing. Ulceration of the cardiac sphincter of the oesaphagus was also observed.

The horse was treated with omeprazole (4 mg/kg once a day per osGastrogard®, Merial Animal Health Ltd., Harlow, Essex, UK.) for

four weeks. The level of exercise undertaken was decreased during the treatment period. A revised dietary plan was introduced to reduce the energy intake, increase forage intake and reduce starch intake per meal (Table 1).

At follow up gastroscopy after a month, no SM ulceration was observed but large multiple lesions were observed within the GM (EGUS 3). Thus, both the medical treatment and dietary regimen was followed for a further four weeks. The third gastroscopy examination was repeated after another month, and showed significant healing of the ulcers in the GM, although the number of ulcers and score remained as a grade 1. The horse's attitude during training improved, it returned to racing successfully after four months and earned prize money. As the horse was performing well it was not investigated again.

Discussion and Conclusion

The effect of gastric ulceration on athletic performance is not well investigated. However, it is frequently suggested that gastric ulceration may be linked to poor performance and there is some evidence to support this (1, 4, 5, 15). The first case report of a link between EGUS and decreased performance in Thoroughbred racehorses was published by Franklin et al. (4). This is the first case report detailing the link between EGUS and decreased performance in the Arabian racehorse. Although the mechanisms are not clear, gastric ulceration appear to impair VO_2 max (12).

The horse is a grazing animal, having evolved over millions of years to digest highly fibrous diets. However to maximize performance, racehorses are often fed low fiber high grain diets. This case report confirms the results of Luthersson et al. (6), which showed a link between higher starch intake and infrequent forage feeding and risk of EGUS. A Danish study of over 200 horses (7) found a significantly greater odds ratio for SM ulcer development in those animals fed high starch intakes of >1g/kg BW per meal or >2g/kg BW per day, straw being the only forage available or infrequent forage intake. Possibly reasons for this observation could be (a) more starch in the diet resulted in increased intra-gastric VFA production, or (b) increasing percentage of starch slows gastric emptying (9) prolonging exposure time of VFA and HCL to the SM. Ingestion of grains and concentrates increases gastrin production stimulating gastric acid production (14), whereas ingestion of hay causes little stimulation of gastrin production. Therefore, horses fasted for significant periods or fed high grain rations are likely to produce more gastric acid with less saliva than horses offered ad lib fibrous forage. Thus, this case confirms the risk factors of high starch intake and low forage intake for EGUS, by the fact that the horse was consuming 3.08 g/kg bw/ meal of starch and only 40% of the diet was forage when diagnosed with grade 4 EGUS.

One way of reducing the starch intake, yet still supplying the energy needs of the horse, is to increase that fat content of the diet. The form of the fat fed may also have a direct influence on the incidence of EGUS. Corn oil contains high levels of the omega 6 fatty acid, linoleic acid, which is an arachadonic acid precursor and research in man and other species has shown similar increases in endogenous prostaglandin levels and reduced acid secretion in the stomach according to the results of Cargile et al. (3) in ponies. These changes indicate up-regulation of mucosal PGE1, an important component of the gastric mucosal protective mechanism (13) and suggest that oils high in omega 6 fatty acids might be useful in prevention and treatment of EGUS. In this case we added corn oil to the diet to supply extra energy from fat and as for the beneficial effects outlined above, but sunflower oil or seeds, and rice bran oil or

Table1. Feeding plan of the horse for 3 different stages

Feed ratio	**Before treatment***	**Treatment phase****	**After treatment***
Forage intake	4 kg (2 kg hay, 2 kgalfalfa)	6 kg (3 kg hay, 3 kg alfalfa)	6 kg (3 kg hay, 3 kg alfalfa)
Concentrate intake	6 kg (1.5 kg racemix A, 1.5 kg mueslimix B and 3 kg barley)	3 kg (2.5 kg racing FEED C and 0.5 kg balancer D)	4 kg (3.5 kg racing FEED C and 0.5 kg balancer D)
Feed additives	50 g vitamin&minerals	100 ml cornoil	100 ml cornoil
Forage:Concentrate ratio	40:60	65:35	58:42
Digestible energy (MJ)	109	91.7	105
Crude protein	1120 g	1100 g	1200 g
Crude fat	450 g (1.1g/kg bw)	380 g (0.9 g/kg bw)	430 g (1g/kg bw)
Crude fiber	1390 g (2.5g/kg bw)	1770 g(4.3g/kg bw)	1850 g (4.5g/kg bw)
Starch intake	2560 g (6.2 g/kg bw)	850 g (2 g/kg bw)	1140 g (2.7g/kg bw)
Starch intake per meal	1280 g (3.08 g/kg bw)	212 g (0.5 g/kg bw)	285 g (0.6 g/kg bw)
Meal sperday	2 equalmeals	4 equal meals	4 equalmeals

*The horse was on intense exercise **The horse was on moderate exercise

Nutrient intakes calculation was based on published values for commercial feeds and book values for other basic feedstuffs. The sugar content of the ration was not calculated as the information could not be obtained from feed company concerned.

A contains extruded corn and barley, cracked, whole sunflower seed, vegetable oils (sunflower seed, soy, linseed, corn and canola), molasses, extruded, stabilized rice bran, extruded vegetable proteins (cotton seed meal, sun flower seed meal, linseed meal and full fat soy), calcium, DCP, salt, minerals and vitamins

B contains extruded corn and barley, cleaned and graded oats, cracked corn, linseed meal, heat processed cotton seed meal, cracked, whole sunflower seed, Vegetable Oils – (sunflower seed, soy, linseed, corn and canola, Molasses, extruded, stabilized rice bran, extruded vegetable proteins (cotton seed meal, sunflower seed meal, linseed meal and full fat soy), calcium, DCP, salt, minerals and vitamins

C contains oats (bruised), micronised wheat, soya (bean) meal, molasses, micronised maize, micronised soya beans, micronised peas, soya oil, distillers' grains, DCP (Dicalcium Phosphate), cooked linseed, soya (bean) hulls, vitamins and minerals, calcium carbonate, grass meal, calcined magnesite, sodium chloride

D contains soya bean meal, micronised soya beans, distillers' grains, DCP, micronised wheat, cooked linseed, vitamins and minerals, calcium carbonate, whey, molasses, grass meal, sodium chloride

stabilized rice bran could also be used. Alfalfa hay has been shown to reduce the incidence and severity of EGUS in several studies (8,11). The mechanism has not been determined but may relate to alfalfas high calcium and protein content which act as buffers of gastric acid. Without a change in the dietary plan, ulcers can be cured by omeprazole but recur when back in full training. In this instance, feeding management was used to help keep the ulcers at bay.

In conclusion, this case report indicates that EGUS may be an important cause of poor athletic performance in some racehorses and dietary treatment is a vital adjunct to medical treatment. The use of smaller and frequent meals, lower starch, corn oil (or other omega 6 fat sources), increased forage and extra alfalfa are major recommendations for feeding horses with EGUS. However, much is still unknown about the clinical signs and causes of EGUS in individual horses and continued research is needed to clarify the role of nutritional factors and dietary management.

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