

Equine grass sickness in Cyprus: a case report

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Abstract: Grass sickness (EGS) is a fatal disease of the family Equidae, which was first reported in 1909, in Scotland. Since then, the disease has been reported in many places around the world; however, it mostly occurs in Great Britain. This case report describes EGS in a 2-year-old colt thoroughbred racehorse in Cyprus. To the author's knowledge, this is the first histopathologically confirmed case of EGS in southern Europe.

Key words: Grass sickness, equine dysautonomia, Cyprus

Introduction

Equine grass sickness (EGS), or equine dysautonomia, is a largely fatal disease of all equidae of unknown etiology. According to Tocher et al. (1), the first outbreak of grass sickness in Scotland occurred in 1909, in army horses at Barry Camp near Broughty Ferry, Angus. A review article from Wylie and Proudman (2) reveals that since then the disease has been reported in Britain, with a predilection in Scotland. It has also sporadically been reported in central Europe, in the Falkland Islands, Australia, and recently in the United States by Wright et al. (3). A condition indistinguishable from grass sickness known as "mal seco" has been recognized in Argentina, the Falklands, and Chile. Both the clinical signs and the postmortem findings of mal seco are similar to those of grass sickness, and the histological lesions in horses with mal seco are also very similar to those described in horses with grass sickness. This is the first time EGS has been recognized in southern Europe.

Case history

The aim of this report was to describe one case of EGS in Cyprus with clinical signs and a histological diagnosis consistent with EGS. This is the first report of EGS in southern Europe, previously noted as "personal communication" in the review article of Wylie and Proudman (2)

A 2-year-old colt thoroughbred racehorse was examined for the first time in March 2006, with signs of acute colic. The colt had been bred in Cyprus and was stabled at Nicosia racetrack for the last month, before the onset of the clinical signs.

Clinical findings

Day 1: Clinical examination revealed tachycardia (90 beats/min), slightly increased rectal temperature (38.6 °C), and decreased gut sounds. On nasogastric intubation, no reflux was obtained.

Day 2: There was a lack of defecation and on rectal examination a large colon impaction was identified.

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The rectal mucosa was dry and small mucus-coated pellets were present. Other clinical signs observed the following days were excessive salivation and spontaneous nasogastric reflux of brown, foul smelling fluid via the nostrils. The colt was seen to regularly play with its water and was eating very slowly and had difficulty swallowing (dysphagia). Muscle tremors and patchy sweating were seen.

Days 3-7: The horse remained dysphagic and signs of rhinitis sicca were apparent with bilateral nasal discharge.

Day 8-14: The peritoneal fluid had a normal gross appearance but the protein content was increased (36 g/L). Hematology showed a packed cell volume (PCV) of 46% and a hemoglobin concentration of 17 g/dL. The white blood count (WBC) was increased ($14.4 \times 10^9/L$) with neutrophils 89% and lymphocytes 8%. Biochemistry included a concentration of plasma bilirubin 6.7 mg/dL, urea 150 mg/dL, alkaline phosphatase (ALP) 1338 U/L, serum glutamic oxaloacetic transaminase (SGOT) 1190 IU/mL, and gamma glutamyl transpeptidase (GGT) 434 IU/L. Plasma concentrations of sodium, potassium, and chloride were 127, 3, and 92 meq/L, respectively.

Day 15-19: At 2 weeks following the onset of the clinical signs, the horse had severe weight loss and

was 'tucked-up' in appearance (see Figure 1). It had a base-narrow stance and a sleepy expression caused by bilateral ptosis. When it walked, it had a weak, short-stride gait.

Day 20-25: The horse's general condition deteriorated gradually leading to its death, 25 days following the onset of the clinical signs.

The postmortem examination showed distention of the stomach, jejunum, and cecum, with air and no ingesta.

Results

Based on the clinical and necroscopic findings, a tentative diagnosis of EGS was made. Tissue specimens from all of the major organs were collected and fixed in 10% buffered formalin, processed, and embedded in paraffin. The histopathological examination of the ileum revealed that the majority of ganglia in the Auerbach's (myenteric) and Meissner's (submucosal) plexuses were characterized by neuronal loss of moderate degree, while the remaining neurons showed central to total chromatolysis, shrinkage, and nuclear pyknosis. In certain neurons, vacuoles adjacent to the cell membrane were observed (see Figure 2).



Figure 1. Appearance of the colt with chronic grass sickness. Note the sleepy expression, severe weight loss, and the tucked-up appearance.

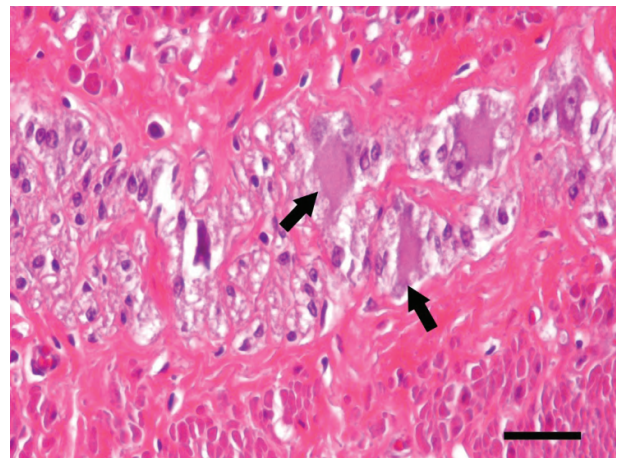


Figure 2. Myenteric (Auerbach's) plexus of a horse with EGS. Neuronal depletion, shrinkage, chromatolysis, and absent or peripherally located nuclei are obvious. Note the vacuoles situated in the periphery of the neuronal perikaryon (arrows). Haematoxylin and eosin stain, Bar = 25 μ m.

Discussion

The 3 main clinical forms of EGS have been described: a) the acute form, which is characterized by dullness, colic of less than 2 days' duration, b) the subacute form, which has a case history of 2-7 days, with similar but less severe signs than the acute stage, and c) the chronic form, which has a case history of more than 7 days and can last up to 2 months. Even though EGS has never been reported in Cyprus, the presence of these typical symptoms in the patient played a vital role in allowing us to include EGS in the differential diagnosis. Owen and Kelly (4) suggested that diagnosis can be correct on the basis of clinical signs alone; however, the only definitive way of diagnosing grass sickness according to Scholes et al. (5) is histopathologically, either from an ileal biopsy or from sympathetic ganglia, and/or ileal tissue taken postmortem. The findings of the histopathological examination in this case confirmed the tentative diagnosis of EGS.

The causal agent of grass sickness is unknown. According to the risk factors identified for EGS, it is thought to be an etiologic agent, which is ingested during grazing and is capable of producing a potent neurotoxin under certain environmental conditions and following dietary changes, as Wylie and Proudman (2) suggest. In this particular case, though, the horse had been stabled for at least a month prior to the onset of clinical signs and no dietary changes

were reported. Lannek et al. (6) have reported rare exceptions where entirely stable-kept horses have suffered the disease. Unfortunately, no effort was made in this study to identify the causative factor.

Age was one of the first risk factors to be identified from McCarthy et al. (7). Younger animals are more likely to be affected by grass sickness than older animals, as reported from Gilmour and Jolly (8). More specifically, horses between the ages of 2 and 7 years are more susceptible to the disease than younger or older horses, according to Wood et al. (9). This was the case in the patient presented in this study.

In Great Britain, where the EGS occurs most frequently, a seasonal variation in the incidence of grass sickness exists. It is noticeable that a spell of warm weather following a wet period brings a large number of cases. May is reported to have the largest number of cases. In the present study, this case did follow the epidemiological pattern of the disease and was noted in March.

A century after its first outbreak, EGS is being reported in many areas of the world. Equine practitioners globally should consider EGS as a differential diagnosis in horses with supported clinical signs. Diagnosis can be aided by histopathological examination after an ileal biopsy or postmortem examination, which is required for a definite diagnosis in order not to miss cases of EGS.

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