Equine grass sickness in the Czech Republic: a case report

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ABSTRACT: Equine grass sickness (EGS) is a degenerative polyneuropathy affecting postganglionic parasympathetic and sympathetic neurons. The major clinical signs relate to dysfunction of the gastrointestinal tract and the condition is frequently fatal. EGS has been reported in different parts of the world including Europe. This paper describes the first case of equine dysautonomia in the Czech Republic. The ante mortem diagnosis was based on typical clinical signs and a positive phenylephrine eye-drop test and was confirmed at necropsy following observation of pathognomic histopathological lesions in the enteric neural system of the ileum.

Keywords: equine dysautonomia; dysphagia; Czech Republic; horse

Equine grass sickness (EGS), also known as equine dysautonomia, is a degenerative polyneuropathy affecting the autonomic and enteric nervous systems and some lower motor neurons in the central nervous system (Hahn et al. 2001). The condition affects grazing equids and is largely fatal. EGS occurs most frequently in Great Britain and has also been sporadically reported in other European countries (Wylie and Proudman 2009; Protopapas et al. 2012; Schwarz et al. 2012) and in the United States (Wright et al. 2010). A similar condition "mal seco" is regularly observed in South America and is generally considered as the same disease (Wylie and Proudman 2009). The major clinical signs, which relate to the dysfunction of the gastrointestinal tract, include colic, dysphagia, sweating and muscle tremors. EGS can be classified according to the duration and severity of the clinical signs as the acute, subacute or chronic form (Hudson and Pirie 2005).

The purpose of this report is to describe a clinical case of equine grass sickness in a horse born and kept in the Czech Republic, outside the typical region of occurrence of this devastating disorder.

Case description

A nine-year-old Standardbred mare was examined by a private practitioner because of an acute onset of fever, weakness, patchy sweating and lethargy. Muscle fasciculation and trembling developed four days later. The mare was used for pleasure riding and kept at pasture with other horses. None of the other horses showed clinical signs of illness. The affected mare had been in a suboptimal body condition for more than one year. The mare was treated with procaine penicillin, trimethoprim-sulfadiazine and flunixin meglumin for seven days. The general condition of the mare deteriorated despite treatment and the animal was referred to the clinic.

At the time of presentation the mare had a poor body condition (BCS 2/9) and was depressed and weak. She showed a tucked-up abdomen and a base-narrow stance. Muscle fasciculations were observed in the flank region and in the triceps and quadriceps muscles. Patchy flank sweating was also noted. Clinical examination revealed a mild tachycardia (46 beats/min), slightly elevated rectal temperature (38.3 °C) and a respiratory rate

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of 20 breaths per minute. The mare showed bilateral ptosis. Mucous membranes were pink with a capillary refill time of less than two seconds. A small amount of mucous discharge was present in both nostrils and the mucous membranes appeared swollen resulting in snuffling respiration. Auscultation of the heart and lungs was normal. Intestinal sounds were slightly decreased with markedly reduced passage of faeces, but no obvious signs of abdominal pain were observed. Rectal examination revealed the large and small colon to be filled with a soft doughy content and dry, hard faeces in the rectum. With abdominal ultrasound imaging an increased amount of free fluid in the abdominal cavity, a thickened small intestinal wall (7 mm) and decreased intestinal motility was seen. An abdominal tap was performed. Total nucleated cell count and total protein content were within normal limits, which was consistent with transudate. Venous blood gas analyses revealed mild metabolic acidosis. PCV was normal and routine haematology was unremarkable. Serum biochemistry revealed an increased blood urea nitrogen (12.4 mmol/l, ref. range 3.5–8 mmol/l), total bilirubin (91.2 μmol/l, ref. range 1.7–39 μmol/l) and mild elevation of muscle enzyme activity (aspartate aminotransferase 423 IU/l, ref. range 150–300 IU/l; creatine kinase 373.8 IU/l, ref. range 60–300 IU/l). The plasma fibrinogen level was increased (4.59 g/l, ref. range 1.0–3.3 g/l). Other parameters were within normal ranges.

On the basis of clinical findings and the obtained laboratory results equine grass sickness was suspected. This *ante mortem* diagnosis was supported by the positive result of a phenylephrine eye-drop test (Hahn and Mayhew 2000).

The mare was treated with flunixin meglumine 1.1 mg/kg *i.v.* SID, trimethoprim-sulfadiazine 25 mg/kg *i.v.* BID and intravenous fluids. Despite the treatment she remained anorectic, lethargic and severe dysphagia developed. Because of the poor prognosis the mare was subjected to euthanasia after two days of treatment.

The necropsy revealed extensive impaction of the right dorsal colon; the rest of the large bowel was filled with a doughy content with a black coating on the surface. Marked infestation of tapeworms was found in the ileum. A thick mucus discharge consistent with rhinitis sicca was evident in both nostrils. The other organ systems were unremarkable.

The histopathological examination of the jejunum revealed large numbers of lymphocytes and plasma cells in the *lamina propria* mucosae. In the ileum a hyperaemic *lamina propria* was infiltrated by lymphocytes, plasma cells, eosinophilic granulocytes and macrophages. There was a marked neuronal degeneration and diffuse loss of neuronal cells in the ileum and the remaining intramyenteric neurons showed cytoplasmic vacuolation. No normal neurons were noted. Also, in the colon sections a few submucosal autonomic neurons exhibited conspicuous cytoplasmic vacuolation. The sympathetic ganglia and brain stem neurons were not examined. Nevertheless, the histological changes of the neurons of the myeneteric plexus were compatible with a clinical diagnosis of equine grass sickness.

DISCUSSION AND CONCLUSIONS

EGS was first recognized in Scotland in 1909. Great Britain is also the country with the highest incidence of EGS. Other frequently affected countries are Belgium, France, Netherlands, Germany, Sweden and Switzerland. Isolated cases have also been reported from Italy, Cyprus, Austria, Finland, Norway, Denmark, Poland and Hungary (Wylie and Proudman 2009; Wright et al. 2010; Protopapas et al. 2012; Schwarz et al. 2012). Although the Czech Republic is surrounded by countries with reported EGS cases, this is the first case of equine grass sickness diagnosed here. Because the definitive aetiology of EGS remains unclear, it is not known why some countries report remarkable higher prevalences than others. Considering the strong association between the development of EGS and access to grazing, an aetiological agent present in the soil or the grass is thought to be involved. It is currently hypothesised that EGS is a toxico-infectious form of botulism, with local neurotoxin production by *Clostridium botulinum* type C within the gastrointestinal tract (Hunter et al. 1999; Wylie and Proudman 2009). Different risk factors have been identified. The disease is diagnosed in all ages of horses, but predominantly affects young animals (most commonly two to seven years old) (Wood et al. 1998). In Great Britain, a strong seasonal variation has been described. The greatest number of cases occurs in spring, especially in May, and a second smaller peak is reported in the autumn (Wylie and Proudman 2009). This corresponds to our case, which also occurred in the autumn. There is evidence that dietary trigger factors are involved in the aetiology of EGS. Toxic *Ranunculus* spp. (butter-

cups) were found in abundance at the EGS site and significantly higher levels of iron and heavy metals in herbage growing in EGS sites was reported by Edwards et al. (2010). In our particular case, the mare had been kept at the same pasture for more than one year and no dietary changes were made. Other horses in the herd were not affected.

The clinical signs in this mare were consistent with chronic EGS and included weight loss, tucked-up abdomen, dysphagia, rhinitis sicca, patchy sweating and muscle fasciculation. The severe dysphagia, rhinitis and marked depression also presented in this mare are thought to indicate a poor prognosis and may preclude successful treatment (Milne et al. 1994).

It has been noted that horses with EGS may show clinical and clinical pathologic signs of systemic inflammation. Copas et al. (2013) reported that serum amyloid A and fibrinogen are markedly elevated in EGS cases compared to healthy controls and non-inflammatory colic cases. In our patient, haematology and plasma biochemistry revealed an increased fibrinogen concentration, while serum amyloid A concentration was not determined.

The main differential diagnoses for thickened small intestinal walls found at ultrasound examination are inflammatory bowel disease, proliferative enteropathy, intestinal lymphoma and intestinal tuberculosis. These were all excluded *post mortem*.

Ante mortem diagnosis of EGS is problematic and often presumptive. The most reliable method currently available is histopathologic examination of the enteric plexuses in ileal biopsy samples obtained by laparotomy (Scholes et al. 1993; Milne et al. 2010). Because this procedure is highly invasive, various clinical diagnostic tests have been evaluated. The ancillary method, which may provide evidence to support a diagnosis of EGS, is testing the reversal of ptosis by phenylephrine (Hahn and Mayhew 2000). Bilateral ptosis is a common clinical sign caused by paralysis of one of the smooth muscles of the upper eyelid. Using 0.5% phenylephrine eye drops in one of the eyes, reversal of ptosis can be observed 30 min later if the eye lashes of the treated eye lift about 15–30° compared to the hanging lashes of the untreated eye.

In our patient the diagnostic laparotomy was declined by the owner and intestinal samples were taken during necropsy. The definitive diagnosis of EGS was confirmed by histopathologic examination of ileal tissue. Equine dysautonomia is characterised by neuronal degeneration, chromatolysis and a reduction in the number of neurons in the autonomic ganglia, enteral plexuses and central nervous system (Doxey et al. 1995; Hahn et al. 2001; Milne et al. 2010). The rarity of enteric neurons in our case was deemed to be highly suggestive of equine dysautonomia. The remaining neurons in the ileal and colonic samples showed marked cytoplasmic vacuolation. Another histopathological feature of EGS is diffuse intestinal inflammation (Whitwell 1997), which was present in ileal and jejunal samples. Unfortunately, autonomic ganglia and central neurons were not examined in this horse.

This is the first published report from the Czech Republic describing a case with clinical signs and histological diagnosis consistent with EGS. Equine dysautonomia should be considered as a differential diagnosis in horses with supporting clinical signs in this country.

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