Equine Grass Sickness (Equine Dysautonomia)

Dr. Patrick Craig D.V.M., PhD.

Equine grass sickness (EGS) is a polyneuropathy affecting the central, peripheral and enteric nervous system of horses with particularly serious consequences for the alimentary function in the acute form of the disease, which is rapidly fatal. The disease affects young horses (2-7 yrs old), stressed horses and those which had recently been moved to a different pasture. Grass sickness is rarely seen in suckled foals below 6 months of age, despite ingesting significant volumes of grass. It is not certain whether this apparent protection is due to maternally derived colostral antibodies, age related differences in physiology, toxin metabolism or due to the way ingesta is processed in the young.

The aetiology of EGS today remains unknown. There have been several speculations about infectious and toxic agents, including plants, fungi, insects, viruses, bacteria and the toxic compounds that these organisms may produce. Despite the speculations, the current leading hypothesis is that EGS is the result of a toxicoinfection with *Clostridium botulinum* types C (BoNT/C) and D (BoNT/D), whereby toxin is produced locally within the horse's gastrointestinal tract by resident bacteria. Acute toxaemia of bacterial origin was suspected based on post-mortem examinations carried out when the aetiopathogenesis of EGS was first proposed (BoNT/C and BoNT/D are neurotoxins produced by C. botulinum types C and D, respectively). Bacterial isolates from cases of EGS revealed morphological and toxigenic properties characteristic of *C. botulinum*. The boulinum theory was discarded for some undetermined reasons, but soon there was renewed interest in the role of *C. botulinum* and this has led to a significant amount of research and results so far are highly suggestive of its association with EGS.

Clinical signs associated with EGS

The disease has been subdivided in acute, sub-acute and chronic forms. These forms reflect the severity of neuronal loss, mainly in the enteric nervous system. Animals with acute of sub-acute EGS usually present with dull demeanour than severe colic, usually profoundly anorexic, have marked tachycardia. Bilateral ptosis due to autonomous dysfunction leading to paralysis of the Muller's muscle is a characteristic sign along with salivation, which likely reflects dysphagia. Rhinitis sicca can be difficult to appreciate in acute and sub-acute cases. The chronic form of EGS has a more insidious onset with varying degrees of anorexia, dysphagia and bilateral ptosis.

Differential diagnoses frequently associated with equine grass sickness

Clinical similarities		Clinical and historical differences	
		Differential diagnosis	Equine grass sickness
Small intestinal strangulating lesion	Sweating Tachycardia Abdominal pain Ileus Gastric reflux Small intestinal distension	Often severe abdominal pain with progressive increase in severity. Progressive signs of endotoxaemia (congested mucous membranes, prolonged capillary refill time) Abdominocentesis may yield serosanguinous peritoneal fluid	Primarily acute grass sickness Abdominal pain rarely severe Depression Salivation Bilateral ptosis Dysphagia
Oesophageal choke	Dysphagia Dullness Sweating Salivation Tachycardia Reduced intestinal sounds	History of consumption of particulate feed Dysphagia associated with the presence of saliva and feed material at the external nares	Salivation rarely seen at the external nares No resistance to the passage of a nasogastric tube, which may result in reflux of gastric fluid
Botulism	Salivation Dysphagia Ptosis Muscle tremors	Profound myasthenia (increasing frequency of recumbency) Pupillary dilation	Patchy sweating Rhinitis sicca tachycardia
Haemoperitoneum	Sweating Mild colic Tachycardia Reduced intestinal sounds Muscle tremors	Pale mucous membranes Whole blood obtained during abdominocentesis may have low PCV, RBC and haemoglobin concentration Abdominal US reveals swirling intra-abdominal haemorrhage	Normal mucous membrane colour Possible reflux following nasogastric intubation Abdominal US reveals generalized distention and absent motility of the small intestine
Hypocalcaemia	Dysphagia Tachycardia Ileus Muscle tremors	May have a history of lactation hyperaesthesia May see synchronous diaphragmatic flutter	Absence of hyperaesthesia Normal serum ionized calcium concentration
Equine motor neuron disease	Weight loss Muscle tremors low head carriage	More profound myasthenia May have fundic lesions Good appetite (occasionally polyphagic)	Absence of fundic lesions Rhinitis sicca Dysphagia Varying degrees of anorexia

Table taken from: In Practice, Lyle and Pirie, 2009.

Treatment of EGS is supportive, however, the marked degeneration and loss of enteric intramural plexuses is irreversible and therefore euthanasia should be recommended once a diagnosis is reached.

Where possible, a postmortem examination of EGS should be attempted in cases diagnosed clinically. This is important because of the association of the disease and the premises on which

the horse was grazing. Absolute diagnostic confirmation is reliant on the identification of the characteristic chromatolytic neuronal changes histologically (loss of Nissl substance, neuronal swelling and vacuolation, intracytoplasmic eosinophilic spheroids, eccentric and pyknotic nuclei).