Article of the month – Equine Atypical Myopathy

What causes equine atypical myopathy?

Since atypical myopathy was first described, factors linked to **environmental toxins** (including plant, bacterial and fungal toxins) were considered as potential causal agents. A recent breakthrough in the search for the cause of atypical myopathy came from looking to human medicine for similar diseases.

Microscopic examination of the muscle tissues from horses affected by atypical myopathy showed severe degeneration, resulting from the **accumulation of lipids** (fat) within the **muscles cells**. This was most evident in Type 1 muscle fibres, which are the main fibre type in major skeletal muscles and those muscles involved in the respiratory system. In humans, a condition known as "Jamaican vomiting sickness" is triggered by the ingestion of ackee fruit, which contains the toxin hypoglycin A. The actions of hypoglycin A on muscle cell metabolism lead to the characteristic degeneration and fat accumulation seen in the muscle tissues of atypical myopathy cases, and high blood concentrations of a toxic metabolite of **hypoglycin A** have been identified in horses affected by the disease.

The presence of trees in or around the pastures where atypical myopathy cases are reported is a common feature both in Europe and the USA, although the tree species found vary considerably between countries and even between outbreaks in the same country. In the USA, a tree from the **Maple family** (*Acer negundo*, commonly known as the Box Elder) has been linked to cases of atypical myopathy and its seeds were found to contain different concentrations of hypoglycin A (Fig. 1). This particular tree is not usually present in Europe and it has not been encountered in pastures where atypical myopathy cases were reported. However, another member of the same family – the **sycamore** tree (*Acer Pseudoplatanus*) is commonly found in European countries and has been reported in pastures affected by atypical myopathy (Fig. 2). The level of hypoglycin A in the seeds of different trees from the Maple family is variable and can be affected by a number of environmental factors. For example, factors including dry conditions, compacted soil, and wind strength may affect the number of seeds produced, seed dispersal and concentration of different substances within fruits and seeds.

Fig. 1 The leaves and seeds of a Box Elder plant (*Acer negundo*)

Fig. 2 The leaves and seeds of the more common sycamore or sycamore maple plant (*Acer Pseudoplatanus*)

Epidemiology and risk factors

In contrast to the more commonly encountered muscle disease azoturia (also known as "tying up" or exertional rhabdomyolysis), atypical myopathy occurs independently of exercise; however some cases have been reported to have exercised immediately prior to the occurrence of clinical signs. Although early studies suggested that the disease was more common in young animals, it has been reported in adult horses of all ages and it **affects all breeds**, with cases also **reported recently in donkeys**.

Atypical myopathy is a disease of horses kept mostly at **pasture**, either affecting individual animals or several horses within a group, and outbreaks can **occur repeatedly** on a property. The disease occurs predominantly in **autumn** (93% of cases); however, some cases are reported during spring (4%), winter (3%) and summer (0.5%). The higher incidence of cases during autumn may be associated with particular unfavourable weather conditions, such as storms and high winds, which have important effects on the spread of sycamore seeds across grazing pastures.

Clinical Signs

The onset of atypical myopathy is rapid, with the major clinical signs relating to **acute, severe damage** to the **postural and respiratory muscle** groups. In some cases, the **early clinical signs** may be confused with **colic or laminitis**. Occasionally atypical myopathy can cause sudden death, but more frequently affected horses will show various **clinical signs** including reluctance to move, muscular weakness, stiffness, apparent sedation or depression, sweating, fine muscle tremors, dark discoloured urine, high heart rate, high respiratory rate and difficulty breathing. The substances produced following the rapid breakdown of muscle cells can reach high levels in the bloods stream, and are then processed via the kidneys, which can lead to acute kidney failure. Unfortunately, once signs of the syndrome are present, the **prognosis is very poor** whatever the treatment and case mortality has been reported from 74% to almost 90%.

Fig. 3 A horse with atypical myopathy receiving intensive supportive care, including fluid therapy via an intravenous drip. Due to the damage of postural muscles during the course of the disease, affected horses are often not able to hold their heads up on their own, and a supportive straw bale has been used in this instance to assist the horse to keep its head up and encourage eating.

Diagnosis and treatment

A presumptive diagnosis of atypical myopathy is based on **history, clinical signs, and laboratory findings.** Blood test reveal marked increases in the concentrations of serum muscle enzymes, confirming the presence of severe acute muscle damage.

Horses affected by atypical myopathy require **intensive supportive and nursing care**, and whenever possible, horses should be carefully transported to a deeply bedded stable (Fig. 3). There is no specific treatment for atypical myopathy and symptomatic treatment is aimed at reducing the risk of kidney failure. This includes intensive fluid therapy, multivitamin injections, antioxidants and pain relief.

Prevention

It is best to avoid grazing on **high-risk pastures** during **high-risk periods** (autumn to spring). Where complete avoidance of affected pastures is not possible, efforts should be made to **reduce** the risk of horses **accessing sycamore seeds**, either by fencing off areas where seeds and leaves have fallen from trees, or by removing the seeds. Providing horses with **supplementary forage** during high-risk periods is also recommended. Following autumn outbreaks, new cases or outbreaks can be expected to occur in the next spring, and the role of sycamore seedlings in causing these spring outbreaks has yet to be determined.

Where atypical myopathy is suspected, all co-grazing animals should be removed from the pasture and their muscle enzymes should be assessed via a blood test, which could help to detect any early cases before the onset of clinical signs. Co-grazing horses should be monitored for several days and receive medical care where required.

Surveillance

In 2000, the University of Liege established a surveillance network: the **atypical myopathy alert group** (AMAG) (http://www.myopathieatypique.be). This group was created to provide information about this disease, gather information about cases all over Europe and disseminate outbreak alerts. Thanks to this surveillance network, more than 600 cases have been recorded since autumn 2006, occurring in several countries in Europe.

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