



## **Focus Article: Update on Atypical Myopathy**

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### **Introduction**

Equine Atypical Myopathy (EAM) is an acute, severe rhabdomyolysis occurring in grazing horses. A frequently fatal myopathy or myoglobinuria of grazing horses has been reported sporadically in various parts of the world throughout the 20th century and was first recognized as a specific disease syndrome, atypical myopathy, following an outbreak in horses in Scotland. Over the previous decade, several outbreaks of the condition have been reported in Europe, and most recently an outbreak this autumn affecting several cases has been reported in the south-west part of England.

### **Epidemiology**

In contrast to exertional rhabdomyolysis, EAM occurs independently of exercise; however some cases have been reported to have exercised immediately prior to the occurrence of clinical signs. EAM 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. Cases occur predominantly in spring and autumn and the higher incidence of cases during these seasons may be associated with particular unfavorable climatic conditions, which appear to play a role in the pathogenesis of EAM. Although it is difficult to identify specific meteorological conditions, cold, damp weather has frequently been associated with outbreaks.

While affected pastures have been variable, overgrazed pastures or poor quality grazing, particularly with poor natural drainage, appear to increase the risk of disease. Many affected fields contain and/or are bordered by trees or shrubs and the presence of fallen leaves or branches has been identified as a risk factor. Additionally, manure spreading on pastures has been shown to significantly increase risk of EAM.

The condition predominantly affects young animals; however, it has also been reported in adult horses. No sex or breed predisposition has been described, although to date, no cases of disease have been reported in donkeys. Normal or underweight body condition is associated with an increased risk of disease, with the majority of affected horses reported to be in good condition, and overweight animals appear at reduced risk. Preventive health care measures such as regular vaccination and anthelmintic administration appear to be protective.

Plant, bacterial and fungal toxins have all been considered as possible causal agents but the aetiology remains unclear. Other possible causes including drugs, environmental toxins, viruses and thyroid dysfunction have not been associated with EAM. Recent research has demonstrated the presence of *Clostridium sordellii*-specific lethal toxin (TcsL) in the skeletal muscle of affected horses, suggests that this toxin may play a role as trigger or even as the lethal factor in this disease. Possibly additional factors such as antioxidant deficient or toxic states may be involved; however the role of nutritional deficiencies, such as selenium or vitamin E deficiency, in the pathogenesis of this disease remains to be investigated.

### **Clinical Signs**

The onset of EAM is acute, with the major clinical signs relating to acute postural and respiratory muscle rhabdomyolysis, although early presentations may be confused with colic or laminitis. Horses may be found dead or more frequently showing various clinical signs including reluctance to move, muscular weakness, stiffness, apparent sedation or depression, sweating, fine muscle tremors, myoglobinuria, congested mucous membranes, tachycardia, and tachypnoea with expiratory dyspnoea. Progression to lateral recumbency is often rapid, and is a poor prognostic indicator. Once signs of the syndrome are present, the prognosis is very poor whatever the treatment and case mortality has been reported from 75% to almost 90%.



### **Diagnosis**

A presumptive diagnosis of EAM is based on history, clinical signs, and laboratory findings. Marked elevations of serum muscle enzymes activities (creatinine kinase [CK], aspartate aminotransferase [AST] and lactate dehydrogenase [LDH]) confirm the presence of severe acute muscle damage. Confirmation of myoglobinuria is also useful in clinical diagnosis, but may be complicated by the increase in urine opacity which can interfere with several methods of urine analysis and many dipstick tests are not able to differentiate myoglobin from haemoglobin.

Confirmation of diagnosis is based on specific histological findings in the affected muscles (postural and respiratory muscles, and occasionally, cardiac muscle). As prolonged recumbency may induce histological muscle degeneration indistinguishable from the lesions of EAM, post mortem muscle samples should be obtained from sites that have not been compressed during recumbency.

### **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 with the aim to disseminate information about this disease, gather information about cases all over Europe and disseminate outbreak alerts. Thanks to this surveillance network 600 cases have been recorded since autumn 2006 in several countries in Europe. Veterinary practitioners are encouraged to report cases to Dominique Votion ([dominique.votion@ulg.ac.be](mailto:dominique.votion@ulg.ac.be)) by filling in and sending both a **clinical** and an **epidemiology** questionnaire.

According to the AMAG, as of 26th November 2010, 39 clinical cases compatible with the diagnosis of atypical myopathy have been reported. These cases were recorded in Belgium (7 cases), France (22 cases), Germany (1 case), United Kingdom (3 cases in the south-west of England), The Netherlands (3 cases), Switzerland (1 case), Spain (1 case) and Czech Republic (1 case). In autumn 2009 the AMAG reported the largest clinical series ever encountered in Europe, with 371 cases identified and a survival percentage of 22%. Back then, 35 cases were reported from United Kingdom.

### **Prevention**

Veterinary surgeons should be aware that new cases or outbreaks can be expected to occur in the spring, following autumnal outbreaks and should institute preventive measures to minimize the risk of disease. Young horses should not be grazed on high risk pastures during autumn and spring, particularly on previously affected premises. Ensuring appropriate preventive health care, management and pasture management practices is also advisable.

Where EAM is suspected, all co-grazing animals should be removed from the pasture and their serum muscle enzymes should be assessed. This may facilitate detection of subclinical cases, which should be monitored for several days and receive medical care where required.