Atypical myopathy (AM) in pastured horses is a severe nonexertional rhabdomyolysis that appears to emerge in Europe. The number of reports to the Atypical Myopathy Alert Group (AMAG) has increased in recent years and since 2000, about 1000 cases compatible with a diagnosis of AM have been recorded.

Recently, the lethal toxin of Clostridium sordelli has been observed in the skeletal muscles of horses with AM. Its presence in the myofibres of horses suffering from AM suggests that it might play a role as a trigger or even as the lethal factor in the aetiology of the disease. However, recent serological data suggest that naturally AM-affected horses neither mount a protective immune response nor show a substantial increase in anti-lethal toxin antibodies, respectively. So, the development of a protective vaccine remains hypothetical. Data collected during field outbreaks throughout Europe may nonetheless suggest a rational approach for disease prevention. This review summarises the latest knowledge about AM.

The disease is characterised by an acute degeneration of postural and respiratory muscles with possible involvement of the myocardium. The clinical signs of AM result from the severe rhabdomyolysis of these muscles, which leads to rapid death (within 72 h) in more than 75% of cases. Affected horses are commonly presented with congestive mucosa, tachycardia, tachypnoea, dyspnoea, moderate pain, myoglobinuria, depression, weakness, stiffness, recumbence and trembling. At rectal examination, a distended bladder is frequently palpated (68% of cases).

A tentative diagnosis of AM is strengthened in horses with marked increase in creatinine kinase activities levels in serum (>10,000 iu/l). Also, hypoglycaemia, hyperglycaemia and hyperlipidaemia are usual laboratory findings in AM.

The metabolic defect occurring in AM mimics a multiple acyl-CoA dehydrogenase deficiency, which blocks mitochondrial fatty acid energy metabolism. The rhabdomyolysis process affects more selectively the slow oxidative type 1 fibres than the glycolytic type 2 fibres with weak activity of the oxidative enzymes and lipid storage in the targeted fibres. At necropsy, pale areas may be seen in the affected muscles (i.e. postural and respiratory muscles which are rich in type 1 fibres).

Outbreaks occur seasonally, mostly in autumn with some cases in spring and sporadic cases in late winter or in late summer. Outbreaks appear to be triggered by inclement climatic conditions but autumnal series cease after several days of heavy frost, suggesting that extreme cold may destroy the causative agent.

Atypical myopathy appears to be a disease solely of members of the family Equidae that includes donkeys and zebras. From epidemiological studies, predisposing factors for AM at the horse’s level were found to be young age, inactivity (i.e. not used for work) and poor to normal body condition. Providing complementary alimentary ails all year round was found to be a protective measure.

Cases of AM tend to recur on the same premises in successive years. Risk factors associated to pasture characteristics are humidity, sloping, accumulated dead leaves, the presence of a waterway and the spreading of manure. Pastures are typically described as natural and being overgrazed. Implementation of routine management practices based on the identified risk factors aims at reducing the likelihood of AM. Prevention is primordial since no specific therapy is available and the fatality rate is high.

The management of diseased horses is directed towards supportive nursing and nutritional care. Treatment includes the drugs routinely used in the treatment of any form of rhabdomyolysis and fluid therapy. Aggressive antioxidant therapy is also recommended since it has proved to increase the chance for survival. Due to alterations in the oxidative metabolism, affected horses depend on carbohydrate metabolism for their energy supply and it is therefore important to feed these cases carbohydrate rich diets several times a day. The nutritional needs of dysphagic patients can be partially met via intravenous administration of balanced electrolyte and glucose solutions. If affected horses are unable to urinate, manual evacuation of the bladder may be necessary.

Medical management of nonrecumbent horses should be encouraged since remaining standing, most of the time is associated to a greater chance of survival. The outcome is related also to the severity of respiratory impairment. When AM declares in a pasture, pasture companions should be stabled and checked for at least 4 days, the maximal latency period of AM.

Following the identification of a horse with history and clinical signs consistent with AM, measures must be instituted immediately to confirm the diagnosis, handle the case adequately, establish a prognosis and to prevent the disease in pasture companions. The diagnostic work-up is described in a concurrent abstract within this proceeding (Votion 2011).

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References and further reading


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