



Increased numbers of Atypical Myopathy cases reported throughout Northern Europe

Liege University's Equine Atypical Myopathy Alert Group has reported an unusually high prevalence of this unusual, life-threatening condition (also called atypical myoglobinuria) throughout northern Europe this autumn. Up to 30th October, 114 cases were reported from England (7 cases), Belgium, Germany, Netherlands, France and Switzerland. At Rossdale & Partners, we have now seen four cases referred from the Newmarket area, one from Bedfordshire and one from Norfolk.

Little is known about the cause of EAM but it has been suggested that it is caused by the ingestion of a pasture-derived clostridial toxin and, in this respect, may be similar to Equine Dysautonomia (Grass Sickness). Some researchers have suggested that there may be an association with eating bark or certain leaves. The condition affects individual and groups of horses at pasture and has a high mortality rate. Up to 89% of affected horses are reported to die or to require euthanasia. Young horses that are in poor to normal bodily condition and are kept on relatively poor quality pasture (often with no supplementary feed) are particularly prone to the condition. The trigger factor appears to be a sudden adverse change in weather conditions, such as a frost or heavy rain (again similar to grass sickness). A number of horses within a particular group are often affected over a short period of time.

Clinical signs include a sudden onset of muscle weakness or stiffness, in a horse or horses at pasture, but unrelated to excessive or unusual exercise. These signs usually progress to collapse and in some cases the affected horse is found recumbent. Rectal temperature, heart rate and respiratory rate may be normal and the horse may appear quite bright and responsive, even willing to eat and drink while recumbent. If the diaphragm is affected there may be dyspnoea (difficult or laboured respiration) and some cases do show colic-like symptoms such as paddling or stretching the limbs and neck. Urine is characteristically dark red-brown coloured and turbid.



Urine sample collected from a case of EAM showing typical dark-red cloudy appearance

The diagnosis is confirmed on the basis of clinical signs and laboratory tests. Serum muscle enzymes (CK, AST, LD) are typically extremely high. Other laboratory abnormalities include metabolic acidosis, hypocalcaemia, hyperglycaemia, hyperlipaemia, hyperlactataemia and increased serum levels of cardiac troponin I. Attending veterinary surgeons should submit at least both clotted (serum) and fluoride oxalate blood samples for laboratory analysis.

Intensive intravenous fluid therapy, anti-inflammatory medication and nursing must be instigated as soon as possible, if the case is to have a chance of survival. The underlying



Collapsed foal with EAM receiving intensive care at Rossdales Equine Hospital Foal Care Unit

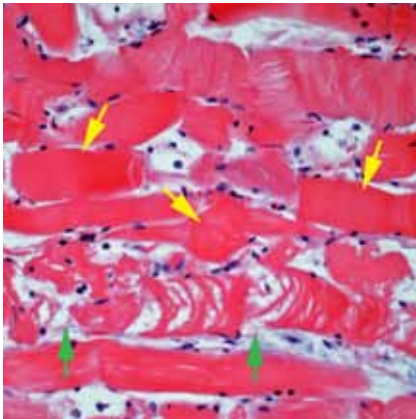
pathology is associated with interference to the uptake and usage of glucose by the muscles. For this reason, the administration of glucose, insulin, carnitine and Vitamin C may be of benefit. There is significant risk of toxic damage to the kidneys and liver. Some cases develop respiratory and cardiac collapse if the associated muscles are significantly affected.

At post mortem examination, dark red-brown urine is present in the bladder. There may not be any obvious gross abnormalities to the muscles, or some muscle may show pale streaking. Microscopically, there is myodegeneration, usually affecting the respiratory and postural muscles most severely and sometimes also affecting cardiac muscle. The kidneys usually show accumulation of proteinaceous material (myoglobin) within their tubules and may show signs of tubular degeneration and mineralisation.

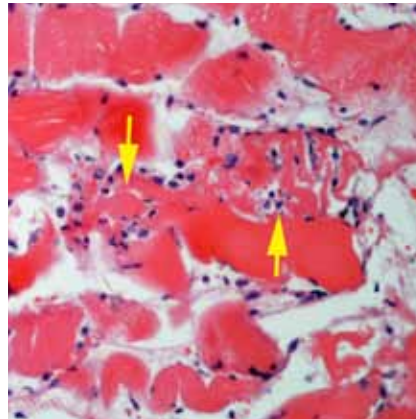


The same foal two days later, still receiving intensive care, but standing and showing signs of gradual clinical improvement

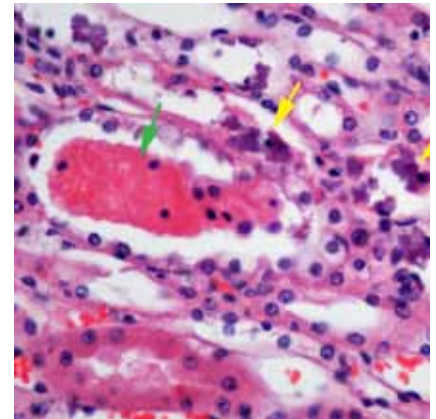
Where there has been a positive diagnosis, other horses in the same paddock should be moved either into stables or a barn, or at least onto different pasture. They should be given supplementary food and have blood taken for muscle enzyme



Microscopic picture of a muscle tissue section taken at postmortem examination from a horse that died with signs of EAM, showing muscle fibre structure disruption and degeneration (green arrows) and signs of muscle fibre swelling and loss of striation (yellow arrows)



Microscopic picture of a muscle tissue section taken at postmortem examination from a horse that died with signs of EAM, showing muscle fibre structure disruption and degenerate fibres (yellow arrows) with signs of leucocyte cell infiltration, indicating early repair



Microscopic picture of a kidney tissue section taken at postmortem examination from a horse that died with signs of EAM, showing a myoglobin 'cast' (green arrow) within a kidney tubule and mineralisation (crystalline calcium deposits - yellow arrows) forming at areas of tubular damage

screening. Any horses with elevated serum CK (creatine kinase), AST (aspartate aminotransferase) and LDH (lactate dehydrogenase) levels should receive appropriate treatment until clinical normality and these muscle enzyme results have returned to normal.

Prevention should include access to pasture with adequate grass and providing supplementary feed, particularly during periods of sudden adverse change in weather conditions, such as a frost or heavy rain. Clean, easily accessible water should be provided from a flowing or easily replenished source.

Owners of horses, particularly young stock living out, should be alerted to this often fatal condition, which seems to be occurring more commonly this autumn.

The Atypical Myopathy Alert Group of Liege University has published extensively and, because of this year's increased incidence, is currently conducting an epidemiological study. For more information on preventative measures and to report suspected cases please go to www.myopathieatypique.be