This short article is to inform colleagues that a frequently fatal disease of grazing horses, atypical myoglobinuria/ myopathy (AM), whose aetiology is not known, has made a re-appearance this autumn and winter in the UK. The condition, first noted in the UK in 1939 and first reported in 1942, was identified again in 1985 and 1986 as the cause of multiple deaths in grazing horses in the UK. Since then it has only been seen very sporadically in the UK. It has also been recognised in European countries - Germany in 1995, Belgium in 2000 and France in 2002. Some of these countries experienced large numbers of deaths from AM (over 100 in Germany in the autumn of 1995 during one large outbreak in the north of the country). It is more common in young horses and has an association with inclement weather.

**Clinical Diagnosis**

Clinically AM appears in one or more horses at grass as a sudden onset of muscle weakness, lethargy, recumbency and death. Muscle tremors and tachycardia may occur and some horses are dyspnoeic. Exercise is not a pre-requisite. Horses are afebrile, and appetite may be retained but some show dysphagia. Deterioration is usually rapid. Clinical chemistry usually shows markedly elevated CK, AST and LDH: SDH is variably elevated. Serum calcium may be very low and should be checked. Urine samples can be discoloured due to myoglobinuria.

**Action:** On suspicion of a case/cases it is recommended that the affected horse and its co-grazers be removed from the paddock and assessed clinically and biochemically. Affected horses are given intensive supportive treatment.

**Concurrent AM and equine grass sickness (EGS):** A recent publication from Belgium records two separate cases of sudden recumbency at pasture in which lesions believed to be consistent with both AM and EGS were discovered. Similarities in the risk factors for both conditions were highlighted. The authors concluded their cases suggest common predisposing causes rather than a common aetiology.

**Pathological diagnosis**

**Sampling:** In AM, in view of the potential for multiple cases it is recommended that sudden deaths at pasture and horses dying after a short illness showing weakness and recumbency be subjected to a detailed necropsy. The necropsy should aim to screen for all causes of weakness, sudden recumbency and death (including recent trauma, acute infections including equine herpes virus (EHV), and visceral ruptures and displacements). It is recommended that, as well as gross and routine histological examination of all the main organs, the necropsy includes tissue harvesting to enable histological assessment of muscle at many sites particularly postural, respiratory and cardiac muscle, autonomic ganglia (e.g. cranial cervical and coeliacomesenteric) and samples of CNS from brain and spinal cord. Where acute infections cannot be ruled out appropriate microbiological screening measures should be a priority.

**Gross Findings:** There are no consistent gross changes in AM but in some there may be obvious discoulouration of striated and cardiac muscle, and distension of the bladder with dark myoglobin-stained urine.

**Histological findings:** Affected striated muscle shows segmental myodegeneration at different stages from simple loss of cross striations and eosinophilia, to sarcoplasmic condensation and discontinuity. The sarcolemmal sheath usually stays intact but may be distorted by swellings and indentations. Intercostal muscles, diaphragm and postural...
muscles are commonly affected. A cell response to the degenerate fibres is mainly by macrophages but some also attract neutrophils. Hepatocytes often show vacuolar change, as do some renal tubular epithelial cells and the adrenal cortex zona fasiculata. Scattered renal tubular pink to red granular casts are seen in the kidneys.

**Final diagnosis**
This is best determined with the benefit of the combined results of clinical, biochemical and pathological findings. Whilst they can be highly suggestive of a diagnosis, neither the clinical nor the gross pathologic changes are pathognomic of AM and so histological assessments are essential. As some myopathic changes can occur secondary to recumbency the appearance of the intercostals and diaphragmatic muscle are particularly relevant. Exclusion of an underlying central neuropathy is also important, EHV paralysis in particular.

AM is a poorly understood condition and has the potential to cause substantial losses. The literature suggests a mortality rate of 89%. An Atypical Myopathy Alert Group (AMAG) is collecting data on AM cases throughout Europe with a view to improving knowledge of the condition. More information can be found about atypical myopathy at [www.myopathieatypique.be](http://www.myopathieatypique.be)

![](image)

**Figure 1:** Striated muscle showing acute myodegeneration. H&E x 100

**References on AM:**
3. Personal communication, Dominique Votion