Dealing with atypical myopathy

What causes the seasonal pasture myopathy and how should cases be managed?

Outbreak characteristics
The earliest UK cases of atypical myopathy, formerly known as atypical myoglobinuria, were described in the veterinary literature in the early 1940s, but the condition was probably encountered long before this according to historical veterinary texts in the preceding decades.

In the early 1980s, vets in Scotland reported an outbreak of myopathy among grazing horses and investigators of this outbreak defined atypical myopathy as a specific disease.

The clinical signs reported in these earliest cases were associated with postural and respiratory muscle failure, and horses demonstrated biochemical evidence of a myopathy (elevated creatine kinase and aspartate aminotransferase) and myoglobinuria.

The first large atypical myopathy outbreak in continental Europe occurred in Germany in the late 1990s and since this time most northern European countries have recognised outbreaks with reported numbers of affected horses and mortality rates varying from year to year.

Veterinarians in the Midwestern USA and eastern Canada have reported a ‘seasonal pasture myopathy’ since the 1960s, which has been described as a degenerative muscle disease of grazing horses with the same characteristics and aetiology as ‘European’ atypical myopathy.

Likely cause
Over the decades, ingested environmental toxins had been considered the likely causative agent in atypical myopathy due to the trend for multiple co-grazing horses to be affected, and because epidemiological studies revealed common pasture characteristics (sparse grazing, trees surrounding pasture) and feeding practices such as lack of supplementary forage.

Recent studies have demonstrated that horses with atypical myopathy (and seasonal pasture myopathy) have an acquired multiple acyl-CoA dehydrogenase deficiency. Analysis of urine and blood from affected cases demonstrated the presence of hypoglycin A metabolites (Votion et al., 2014). European horses probably acquire atypical myopathy via ingestion of hypoglycin A in the seeds (in autumn/winter) and seedlings (spring) of the tree Acer pseudoplatanus (European sycamore).

When seeds from sycamore trees on the pastures of affected horses were analysed, variable concentrations of the toxin were found within and between trees on the same pasture (Unger et al., 2014). The factors affecting the concentration of hypoglycin A in an individual seed or tree are yet to be established.

Epidemiological studies have identified horse management practices associated with an increased risk of atypical myopathy. These factors probably influence the likelihood of toxin exposure, e.g. inclement weather causing seeds to fall onto pasture; or ingestion, e.g. inactive horses, lack of provision of supplementary forage, time spent on pasture v. stabled.

Clinical presentation
Atypical myopathy results from a deficiency of the fatty acid oxidation pathway, which is the primary energy source in type I muscle fibres. Hence, clinical signs relate to postural and respiratory muscle damage and failure and a cardiomyopathy is also present in many cases.

Clinical signs include lethargy, stiffness, reluctance to move, muscle tremors, and increased recumbency and tachypnoea. Tachycardia is often present due to anxiety, pain, hypovolaemia, and intrinsic cardiac pathology. Some horses have a persistent low head carriage, resulting in pharyngeal and generalised head oedema, partial respiratory obstruction and exacerbation of the respiratory distress. Increased vocalisation, head-tossing and oesophageal obstruction are also seen less frequently.

Frequently atypical myopathy cases have myoglobinuria that is recognised practically as dark red/brown and concentrated urine with high specific gravity (SG) and a positive dipstick for blood (haemoglobin and myoglobin are not distinguishable on urine dipstick testing).

A horse with cardiovascular compromise or hypovolaemic shock from another cause (strangulating intestinal obstruction/enterocolitis) will have concentrated dark