British Deer Veterinary Association (BDVA)

Position statement on capture myopathy in wild deer in the UK

Capture myopathy (or post capture myopathy) is a complicated syndrome that affects deer that have been stressed, trapped, confined, darted or netted. It is not a simply a matter of exertional rhabdomyolysis and/or lactic acidosis, although muscle tissue pathology is a prominent feature. There are profound and generalised metabolic disturbances. The syndrome may occur during or soon after capture or entrapment, although the onset may be delayed for up to 48 hours after the deer is released or taken into captivity. Clinical signs may be unilateral or bilateral and include weakness, ataxia, paralysis, hyperventilation and death. There may be non-weight bearing lameness, with reduced or absent sensation, proprioception and motor function. If the deer is seen to urinate, myoglobinuria may be evident. Some deer species appear more prone to this condition than others: fallow deer, axis deer and roe deer are commonly affected; red deer, sika deer and muntjac seem to be less susceptible to clinical signs.

If blood samples are taken at the time of capture, muntjac may have very high creatine kinase and lactate dehydrogenase levels without ever manifesting clinical signs of myopathy (Chapman & Green 2025). No link has been clearly established between any particular biochemical markers at the time of capture and the risk of development of myopathy.

Because the syndrome may have a delayed onset, the BDVA advises caution when contemplating the release of wild deer that have been entrapped or tangled or confined and have struggled forcibly to free themselves. There is substantial risk that these animals are likely to suffer immediate or delayed capture myopathy and are unlikely to survive long term. Welfare of the individual is compromised with the development of signs of myopathy. Any deer that show any evidence of incapacity, including being handleable when freed from entrapment, are likely to be severely compromised and should be humanely euthanised on welfare grounds. If such deer are released into the wild, if possible, they should be monitored for an hour or two. If they run swiftly away and disappear when released, the risk of myopathy may be low, but if they are sluggish, ataxic or if they move a short distance and lie down, capture myopathy and subsequent death are highly likely. These animals should not be left to linger with myopathy, they should be humanely dispatched by means of a rifle.

There will be some deer where there are no obvious signs suggestive of capture myopathy and after careful assessment (including how long they have been restrained) these may be candidates for immediate release.

Moving deer into some form of enclosure in order to monitor them may make capture myopathy more likely because it prolongs stress.

The treatment of capture myopathy is generally unrewarding and would require moving the deer into an enclosed system (with the need to consider various factors including as age, sex, species and drug residues). Mild cases may respond to slow administration of sodium bicarbonate (5 meq/kg) to combat acidosis and may benefit from diazepam (0.1-0.3 mg/kg) as a muscle relaxant. However, close human contact and the act of administering the medication may be so stressful to the deer that such therapy only worsens the syndrome.

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Chapman NG & Green P (2025) In: Deer Veterinary Medicine (in press), Wiley-Blackwell.