

Case Study

Capture myopathy in red deer and wild goat

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ABSTRACT

This syndrome is a shock-like hyper metabolic myopathy triggered in susceptible animals by stress. Capture myopathy (C.M.) is a commonly occurring condition in mammals following trapping and transportation. In this case 12 to 24 hours after transportation of red deer (*Cervus elaphus*) and wild goats (*Capra ibex*) clinical signs such as: muscular tremor, ataxia, recumbency, hyperthermia, tachycardia, hyperventilation and red brown urine observed. According to symptoms Capture myopathy was diagnosed Treatment was ineffective on one red deer and one wild goat. Necropsy findings of dead animals were included: hyperemia, petechial hemorrhage in pericardium and heart muscle, pale foci of leg and heart muscles and red brown urine in bladder. This case report represents the attention to Capture myopathy in wild animals and particular caution that should be exercised in capturing and handling of these animals.

Keywords: Capture myopathy, red deer, wild goat

INTRODUCTION*

Capture myopathy (C.M.) also known as exertional rhabdomyolysis, is a condition associated with stress which occurs in mammals and some species of birds. In some cases is associated with chemical immobilization (James *et al* 1991, Bartsch *et al* 1973). Capture myopathy became widely recognized in Africa in the late 1960s and early 1970s when many rare animals died during or soon after capture. This syndrome was first described in a wild Hunter's antelope in South Africa (Jarrett *et al* 1964). Subsequently it was observed in many other wild African ruminant species

(Chalmers & Barrett 1971)(Spraker, Adrian and Lance 1978). A similar condition to C.M. in Rocky mountain goats was described by Herbert and Cowan(1971).C.M. was described in white tailed deer (Beringer *et al* 1996) Moose (Haich *et al* 1971) pronghorn antelope (Chalmers & Barrett 1971) (5) Rocky mountain bighorn sheep (Spraker 1977) and elk (Lewis *et al* 1978). It was described in some species of birds such as Flamingo Greater sandbill crane several finches and Turkeys (Spraker 1987). In New Zealand the live capture of red deer for deer farms has been plagued with losses that have varied according to the methods, drugs, time of year and manner of handling the deer. Post losses have ranged from a few percent to up to 70%. This condition is initiated by stress, e.g. handling,

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managing, transport, mating, excitement and exercise. As the Capture myopathy is difficult to treat, every effort should be made to prevent the problem. Thus wild life personnel should exercise extreme care during trapping, handling and transportation of animals.

CASE HISTORY

Two red deer (*Cervus elaphus*) and two wild goats (*Capra ibex*) showed some clinical signs in 12-24 hours after transportation. Capturing, restraint and transportation time of animals was about 3 hours. The clinical signs included: depression, debilitation, lassitude, muscle stiffness, tremor, ataxia, firm stepping (muscle tremor is evident in muscles of back and legs that progress to muscle rigor, and the animal is reluctant to move), Tachycardia (up to 20 beats /min), open mouth breathing, rapid shallow breathing, laminitis, recumbency, mucosal hyperemia, anus temperature of 42 and red brown urination. Administration of vitamin E, Selenium, Flunixin meglumin and fluid therapy along the providing an individual pen, performed to animals. One red deer with mild symptoms recovered but one red deer and one wild goat died with sever symptoms about 30 hours after moving. Necropsy findings were hyperemia, petechial hemorrhage in pericardium and heart muscle, pale foci of leg and heart muscles, inflammation and darkness of kidneys and red brown urine in bladder.

DISCUSSION

Observed clinical signs in these cases are commonly occurring conditions in such animals following transportation. The clinical signs related to the exhaustion of several physiological mechanisms of the animal, including muscular exhaustion and response of the autonomic nervous system. Prolonged stimulation cause to tissue perfusion, tissue hypoxia, cellular necrosis and vascular collapse and shock may occur. Hypoxia results because myoglobin depletes its stores of oxygen and the hemoglobin cannot fulfill all the

needs of the contracting muscle. Prolonged muscular exertion can further lead to anaerobic metabolism (glycolysis) and build up of lactic in the muscle, resulting in cell death, acidosis and death due to shock. In cases that the irreversible shock does not progress, mild to severe muscle necrosis and renal nephrosis (due to hypoxia and toxic properties of the myoglobin) could be observed.

Predisposing condition for C.M. include strenuous pursuit during capture operation, genetic predisposition, transportation, handling, overexertion during periods of high ambient temperature and possibly deficiency in selenium and vitamin E. Some of these predisposing factors were present in these cases. At postmortem autopsy muscles of the back, thigh, lion and shoulder are the most extensively affected and should be examined grossly and histologically. In autopsy of these cases gross lesions of affected muscles (thigh and myocardium) were pale and wet foci. The gross lesions observed in these cases were similar to the gross lesions reported in Proghorns (Chalmers *et al* 1971). Capture myopathy is difficult to treat so prevention is crucial. However the treatment consist of removing of animal from impending stress, intravenous (IV) administration of tranquilizers, fluid therapy to correct acidosis, surface cooling and hyperventilation with oxygen. In these cases mentioned treatment improved the one wild goat health with mild symptoms and could be advisable (Clarence & Fraser 1991)(Murray *et al* 2003) (Willims & Trone 1996)(Otto *et al* 2000).

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