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The Dark Side of Conservation - Understanding Capture Myopathy

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Abstract

Capture myopathy in wild animals refers to the development of muscle damage and associated complications resulting from the stress and exertion experienced during capture, handling, or other human interventions. This condition is commonly observed in various wildlife species, particularly during capture for research, relocation, rehabilitation, or management purposes. The underlying mechanisms and clinical manifestations of capture myopathy may vary depending on factors such as species, environmental conditions, and the specific circumstances of capture. This mini-review provides a broad overview of capture myopathy in wildlife associated with conservation measures.

Keywords: Ungulates, Myopathy, Rhabdomyolysis, Conservation

Introduction

Due to the near extinction of around 25% of carnivores and ungulates in the last decades globally due to human activity and climate change (Cardillo *et al*., 2005) [5], the introduction or reintroducing species through translocation to areas in their former range is widespread and increasingly practised by conservationists (Di Marco *et al*., 2014) [6].

Capture myopathy (CM) is a metabolic disease of wild and domestic animals that leads to significant morbidity and mortality rates. CM is a pathophysiological manifestation of animal stress. It can occur naturally when an animal attempts to avoid predation or run from danger in situations of great stress (Breed *et al*., 2019; Businga, Langenberg, & Carlson, 2007) [2, 3]. Unfortunately, it is often documented during capture and translocation procedures during conservation measures (Breed *et al*., 2019) [2]. CM is one of the causes of the low success rates of the translocation of wildlife (Dickens, Delehanty, & Michael Romero, 2010) [7].

It has been reported in several species of mammals, birds and reptiles (Di Marco *et al*., 2014) [6]. There are many contributing factors for CM which include species (more prey, particularly ungulates and bovids) (Ali *et al*., 2023) [1], environmental factors (high temperature, rain, high humidity), capture-related (technique, injuries, prolonged chases excessive handling, prolonged restraint, crating), other diseases (Landau, Kenney, Deuster, & Campbell, 2012) [12], nutrition (vitamin E, selenium deficiency, obesity), use of some anesthetic drugs (opioid-based combinations), and signalment (very old or young animals, males, pregnant females) (Dinesh *et al*., 2020; Krauer, 2024) [8, 11].

This mini review provides a broad overview of capture myopathy in wildlife associated with conservation measures.

Pathophysiology

CP represents a multifactorial syndrome involving the interplay of ischemia, metabolic disturbances, oxidative stress, inflammation, and muscle fiber necrosis (Breed *et al*., 2019; Krauer, 2024; Vanholder, Sever, Erek, & Lameire, 2000) [2, 11, 17]. During CP, muscle damage (rhabdomyolysis) occurs with the release of myoglobin and creatine kinase into the bloodstream. At the same time, blood lactate concentration increases, leading to a decrease in pH and acidosis and a rise in body temperature (Meyer, Fick, Matthee, Mitchell, & Fuller, 2008) [14]. The increased metabolic activity and oxygen demand during exertion can produce reactive oxygen species (ROS) within the muscle cells that can cause oxidative damage to cellular structures (Vanholder *et al*., 2000) [17]. The stress response triggered during capture or handling can lead to the release of stress hormones

such as cortisol, which can disrupt electrolyte balance in the body (Krauer, 2024; Vanholder *et al*., 2000) [11, 17]. Acute kidney failure occurs due to myoglobinuric acute kidney injury that is induced by prolonged vasoconstriction, intraluminal cast formation and haem–protein-induced cytotoxicity (Vanholder *et al*., 2000) [17]. Creatine kinase is elevated ten times the species' upper reference limit, and it is possible to observe myoglobinuria, hyperkalemia, and coagulopathy (Vanholder *et al*., 2000) [17]. Eventually, multiple organ failure and death follow the myoglobin-induced acute kidney failure (Fig 1).

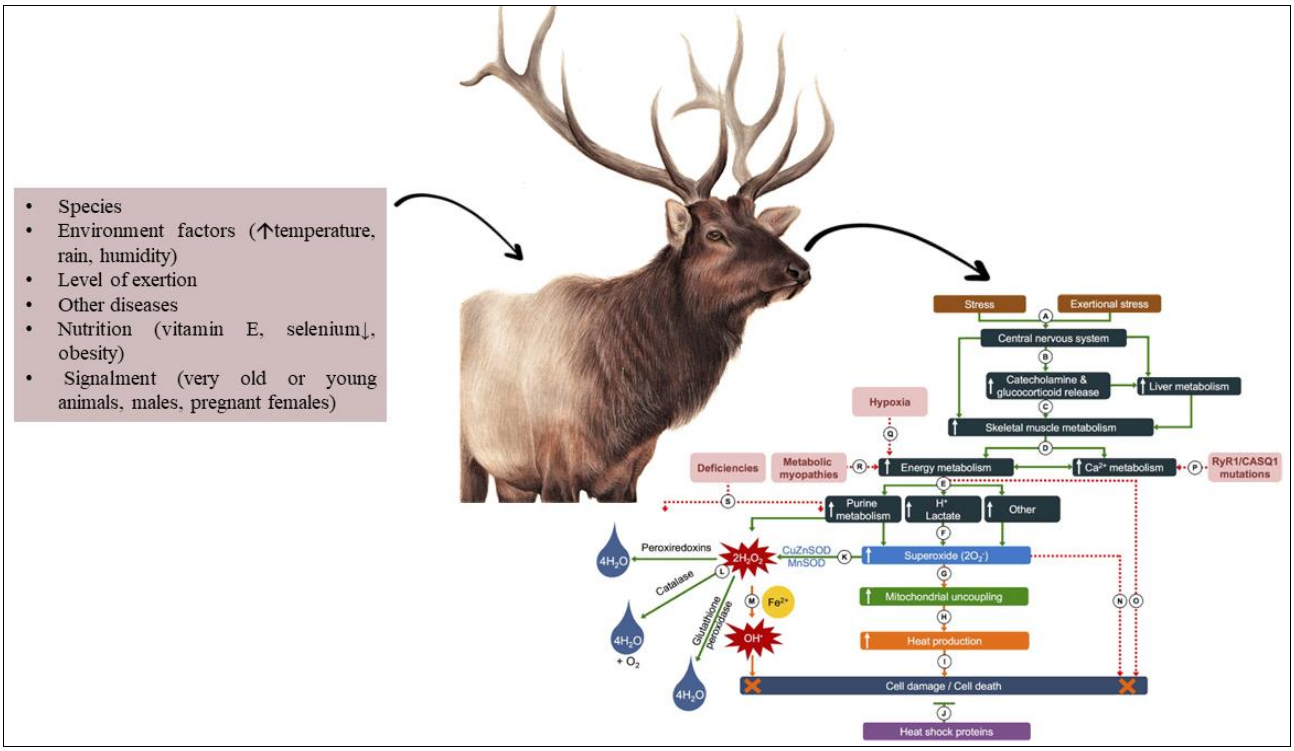

Fig 1: Schematic representation of possible pathomechanisms of capture myopathy in wild animals (Breed *et al*., 2019) [2]. (A) Stimuli in the form of fear and exertional stress (typical fight or flight response), with the central nervous system reacting to the stimuli. (B)Increase in sympathetic nervous activation and increased adrenalin, noradrenalin, dopamine and glucocorticoid secretion and release, as well as increased liver metabolism and skeletal muscle activity. (C) Increased catecholamine secretion upregulates skeletal muscle metabolism. (D) Increased ATP production from glycogen breakdown and phosphagen pathways in response to the demand from skeletal muscle contraction—myosin ATPase activity, active Ca2+-resorption into sarcoplasmic reticulum and the Na+K+ATPase pumps. (E) The increased demand for ATP replenishment results in elevated purine metabolism increased lactate and H+ production, and other pathways resulting in (F) increased generation of reactive oxygen species (ROS), such as superoxide (O2–). (G) The increase in O2– results in more significant uncoupling of oxidative phosphorylation and (H) increases heat production from the skeletal muscle. (I) An elevation in muscle temperature increases the risk of muscle fibre damage and necrosis (J) but is counteracted by the protective effect of heat shock proteins. (K)O2– is converted to hydrogen peroxide (H2O2) by superoxide dismutase (SOD), which requires zinc, copper and manganese to function optimally. (L) Three pathways neutralize the H2O2 to water (peroxiredoxins and glutathione peroxidase that requires selenium to function optimally) and oxygen (catalase). (M)Ifnot neutralized, H2O2 may be converted to hydroxyradical molecules (OH·) through the Fenton reaction (involving iron) that can cause severe cellular damage. (N) Excess ROS, especially in O2, may cause cellular damage. (O) A lack of ATP replenishment as a result of excessive metabolism [e.g. glycogen depletion or (Q) hypoxia] prevents the myosin–actin cross-bridges from detaching (a form of rigour). It leads to damaged muscle fibres through the mechanical stretch. Mutations in receptors involved in (P)Ca2+ regulation or (R) ATP production can result in muscle damage through the exact mechanism proposed in (O). (S) Mineral deficiencies (co-factors) within the oxidative stress pathway enzymes can lead to diminished antioxidant capacities, leading to excess ROS that may injure cell membranes.

Pathology and clinical signs

Clinical signs of capture myopathy can vary depending on the species and the cause of exertion. They may develop hours, days, or up to two months following capture. The most affected are the large muscles of the limbs, pectoral, intercostal, and cardiac muscles. Lesions are bilateral and symmetrical (Dinesh *et al*., 2020) [8].

The most common clinical signs are depression, lethargy, unresponsiveness to human presence, muscle stiffness or weakness, tremors, ataxia, firm stepping, tachycardia, open-mouth and rapid breathing, hyperthermia and red-brown urine (Krauer, 2024) [11]. Once clinical signs appear the prognosis is generally poor (Rosenhagen, 2023) [16].

There are four different syndromes of CM described: hyperacute or capture shock syndrome, acute or ataxic myoglobinuric syndrome, subacute or ruptured muscle syndrome, chronic debility or delayed peracute syndrome (Breed *et al*., 2019; Dinesh *et al*., 2020) [2, 8] (Table 1).

Table 1: Characteristics of the four different syndromes of Capture Myopathy regarding time appearance, clinical signs and biochemical values (Breed *et al*., 2019; Câmara *et al*., 2020) [2, 4]

	Time and prognosis	Clinical signs	Biochemical
Hyperacute or capture shock syndrome	During immobilization or within a short time after Death 1–6 h post capture	Depression, hyperpnea/ tachypnea, tachycardia, elevated body temperature, weak thready pulses	Elevations in serum aspartate aminotransferase (AST), creatinine phosphokinase (CK), and lactate dehydrogenase (LDH) enzyme
Acute or ataxic myoglobinuric syndrome	Common Hours to days post capture Animals with moderate to severe symptoms have higher mortality.	Severe ataxia, torticollis, myoglobinuria	Elevated serum enzymes (AST, CK, and LDH) and blood urea nitrogen (BUN)
Subacute or ruptured muscle syndrome	4–48 hours post capture	Marked drop in the hindquarters and hyperflexion of the hock due to unilateral or bilateral rupture of the gastrocnemius muscle	Elevated AST, CK, LDH BUN may be within normal limits or slightly elevated.
Chronic debility or delayed peracute syndrome	Rare, associated with a second stress event during 24h post capture	Calm if undisturbed. If manipulated, try to run but stay still abruptly; eyes begin to dilate, and death occurs within several minutes.	Elevated AST, CK, and LDH

At *post-mortem* examination, light-colored skeletal muscles, more evident in the limbs, and discoloration of cardiac muscle (Fig 1), severe to minor intestinal and hepatic congestion, pulmonary congestion and edema can be observed. The kidneys are dark, with slight lobulation on the cortical surface (Fig 2), and in the bladder, the urine has a dark brown color. Histologic findings may include necrosis in skeletal muscle, brain, liver, heart, adrenal glands, lymph nodes, spleen, pancreas, and renal tubules, and nephrosis due to accumulation of myoglobin (Dinesh *et al*., 2020) [8].

Fig 2: A) variegated discoloration of the myocardium in a deer; B) darkened kidney in a deer due to myoglobinuria; C, D) discoloration skeletal muscle in roe deer

Diagnose, Treatment and Prevention

Diagnosis is based on clinical history, clinical signs, clinical pathology, and gross and microscopic pathology (Dinesh *et al*., 2020; Krauer, 2024) [8, 11]. There is no specific treatment for CM, and it generally has a low success rate (Breed *et al*., 2019; Câmara *et al*., 2020; Dinesh *et al*., 2020) [2, 4, 8]. Treatment is supportive using analgesia, dantrolene sodium (McKenzie, Valberg, Godden, Finno, & Murphy, 2004) [13], fluid therapy, muscle relaxants (benzodiazepines, methocarbamol), vitamin E and selenium supplementation, surface cooling, oxygen supplementation, and hyperbaric oxygen, sodium bicarbonate (Businga *et al*., 2007; Dinesh *et al*., 2020) [3, 8]. Prevention is always the best option. Species that are susceptible to CP should be handled with care and gently; captures should not be performed on hot days or cold water to minimize stress during capture and use anesthetic agents (e.g., Ketamine, Xylazine) if necessary (Breed *et al*., 2019; Dinesh *et al*., 2020) [2, 8]. Always use capture methods that minimise animal stress, struggling time, and handling (Breed *et al*., 2019; Meyer *et al*., 2008) [2, 14].

Capture myopathy poses and wildlife conservation efforts

CM can pose a significant challenge to wildlife conservation efforts, as it can impact the health, welfare, and survival of individual animals and populations. Conservation programs or scientific studies often involve capturing and relocating animals for reintroduction, translocation, or supplementation efforts (Hartup, Kollias, Jacobsen, Valentine, & Kimber, 1999) [9]. However, the stress of capture and handling can increase the risk of CM, potentially compromising the success of these programs and the animals' welfare (Breed *et al*., 2019; Herráez *et al*., 2013) [2, 10].

It can be particularly detrimental to endangered or vulnerable species with small or fragmented populations since the loss of even a few individuals due to capture-related injuries or mortality can have significant implications for the long-term viability of these populations (Di Marco *et al*., 2014) [6]. Even after successful capture and release, individuals may continue to be at risk of capture myopathy-related complications, such as delayed mortality or impaired fitness (Dinesh *et al*., 2020; Rosenhagen, 2023) [8, 16]. Long-term monitoring efforts are essential to assess the health and survival of released animals, identify potential impacts of capture-related stress on population dynamics, and inform adaptive management strategies (Miller *et al*., 2013) [15].

Conclusion

CM is a condition that leads to the death of many wild species, with no cure available. It is characterized by severe muscle rhabdomyolysis, kidney failure and elevated body temperatures. Although the capture and handing of susceptive species to CM is necessary as a conservation major, this tool negatively impacts these animals. CM represents a significant concern for wildlife professionals, researchers, and conservationists involved in capturing and

managing wild animals. Now, little is known regarding pathophysiology, triggers and predisposing factors that induce CP. Mitigating the risk of capture myopathy requires careful planning and implementation of capture protocols designed to minimize stress and exertion and strategies for early detection and intervention in affected individuals. Additionally, efforts to enhance habitat quality, address nutritional deficiencies, and promote the welfare of wild populations can help reduce the prevalence and severity of capture myopathy in wildlife. This knowledge is necessary in the future to ensure animal welfare and survival, particularly in already endangered species.

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