REVIEW ARTICLE

Capture Myopathy in Wildlife: Pathophysiology, Clinical Management and Therapeutic Interventions – A Review

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Abstract

Capture myopathy constitutes a severe pathological condition manifesting in wild fauna following pursuit, entrapment, physical restraint, live stranding events, or transportation procedures. This multi-systemic disorder exhibits significant morbidity and mortality rates across affected species. While all wild vertebrates demonstrate susceptibility, certain taxonomic groups exhibit sensitive predisposition to this condition. Diagnostic protocols rely predominantly on behavioural assessment, comprehensive case history analysis, and clinical symptoms; however, definitive diagnosis frequently requires post-mortem examination. Therapeutic interventions primarily encompass fluid resuscitation, analgesic management, and rehabilitative physiotherapy protocols.

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Introduction

Capture myopathy (CM) synonymously termed exertional rhabdomyolysis, white muscle disease, or stress-induced myopathy represents a non-infectious, metabolic syndrome of wild and domestic animals characterized by elevated morbidity and mortality indices (Paterson, 2014; Dinesh *et al.*, 2020). Initially documented in 1964 in *Beatragus hunteri* (Hunter's antelope), CM has subsequently been reported across diverse taxonomic groups, encompassing terrestrial and marine mammals, avian species, and reptilian orders. The condition demonstrates highest prevalence among ungulate species and long-legged wading birds (Jarrett *et al.*, 1964; Paterson, 2014; Breed *et al.*, 2019). CM is a physiological response to extreme stress in animals. It can occur naturally when an animal attempts to escape predators or flees from a perceived threat (Businga *et al,* 2007; Breed *et al.,* 2019). Unfortunately, CM is frequently observed during capture, handling, and translocation activities carried out for conservation purposes (Breed *et al.,* 2019). This condition is a significant factor contributing to the low success rates of wildlife conservation translocation efforts (Dickens *et al.,* 2010). Analogous pathophysiological processes occur in humans, typically associated with intensive physical exertion during endurance athletics, or severe traumatic crush injuries (Paterson, 2014). It is also a non-infectious disease characterized by severe muscle degeneration, metabolic acidosis, and myoglobinuria. Once clinical signs appear, the disease is difficult to treat and usually has a poor prognosis. Among mammals, prey species particularly ungulates are considered the most susceptible to capture myopathy (Rokde *et al.*, 2023). Interference with ecosystem such as deforestation, construction of roads, buildings, check dams, agriculture, the formation of ecological mosaics, tourism, etc., around the wildlife habitat changed the diurnal habits of wild ruminants in to nocturnal (Bhaydiya *et al.*, 2021). Heavy mortality in wild ungulates due to capture myopathy is often underreported, particularly during capture, handling, and translocation operations.

In wild populations, CM commonly arises from extended pursuit, physical capture, prolonged restraint, live-stranding events (particularly among marine mammals), or stress associated with transportation (Cattet *et al.,* 2008; Herráez *et al.,* 2013; Breed *et al.,* 2019; Dinesh *et al.,* 2020; Hurtado *et al.,* 2021). An evolutionary hypothesis once proposed that CM might offer adaptive benefits by promoting rapid prey mortality, thereby reducing energy expenditure for predators. However, current scientific consensus holds that CM is primarily an iatrogenic condition induced by human activities rather than a naturally occurring ecological phenomenon (Breed *et al.,* 2019). Ecological studies have now shown that oxidative status can be used as an indicator of fitness components in wild animals. It can also help predict their chances of reproduction and survival in their natural

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habitats. In ruminant species spotted deer and barking deer oxidative markers in muscle tissues showed a non-significant increase in affected individuals. Capture myopathy, resulting from exertion or stress-induced muscle degeneration in captured wild animals, is often fatal (Rokde *et al.*, 2023).

Capture myopathy (CM) is classified into four distinct clinical syndromes based on temporal presentation and symptoms:

1. ***Hyperacute or Capture shock syndrome*** – characterized by acute mortality during or immediately following capture procedures.
2. ***Acute or Ataxic myoglobinuric syndrome*** – manifests hours to days post-capture with evident myonecrosis affecting both skeletal and cardiac musculature.
3. ***Ataxic myoglobinuric syndrome, subacute or Ruptured muscle syndrome*** – animals may initially appear asymptomatic but subsequently experience acute muscle rupture, particularly affecting posterior limb musculature, resulting in abnormal postural configurations and frequently permanent functional impairment.
4. ***Delayed or Per-acute chronic debility syndrome*** – the least prevalent form, observed in previously pursued animals. Upon renewed chase stress, affected animals demonstrate acute collapse and mortality with characteristic mydriasis.

All CM variants exhibit significant biochemical abnormalities. Among these classifications, ataxic myoglobinuric syndrome demonstrates highest reported incidence and optimal therapeutic responsiveness (Spraker, 1993).

Pathophysiology

The pathophysiological mechanisms underlying capture myopathy involve complex multi-systemic alterations that demonstrate progressive deterioration without immediate therapeutic intervention. Excessive muscular exertion or sustained mechanical pressure on skeletal muscle groups particularly affecting appendicular and pectoral regions initiates myocyte necrosis with subsequent release of intracellular potassium and myoglobin into systemic circulation (Cattet *et al.*, 2008; Dinesh *et al.*, 2020). During capture myopathy (CM), muscle damage (rhabdomyolysis) occurs, resulting in the release of myoglobin and creatine kinase into the bloodstream. Concurrently, blood lactate levels increase, leading to a drop in pH (acidosis) and a rise in body temperature (Meyer *et al.,* 2008). Concurrently, compromised vascular perfusion resulting from muscular compression or inadequate oxygen and nutrient delivery due to excessive metabolic demands precipitates diffuse tissue hypoxia, metabolic acidosis, and cellular dysfunction. These pathological conditions accelerate myocyte degeneration and necrosis (Paterson, 2014; Breed *et al.*, 2019).

Myoglobin, a haeme-containing muscle protein, exhibits inherent nephrotoxic properties. It contributes to renal injury through vasoconstriction mechanisms and reduced renal perfusion. Additionally, myoglobin precipitates obstructive tubular casts, leading to tubular necrosis. These deleterious effects are exacerbated in dehydrated subjects (Breed *et al.*, 2019). Acute kidney failure in capture myopathy results from myoglobinuric acute kidney injury, which is triggered by prolonged vasoconstriction, intraluminal cast formation, and haem-protein-induced cytotoxicity (Vanholder *et al.,* 2000).

Hyperkalemia resulting from extensive myolysis can precipitate cardiac dysrhythmias or asystole (Montané *et al.*, 2002). Hyperthermia secondary to intense exertion and physiological stress frequently exacerbates tissue injury, contributing to multiorgan dysfunction and necrosis (Rae *et al.,* 2008). Creatine kinase is elevated 10-times the species' upper reference limit, and it is possible to observe myoglobinuria, hyperkalemia, and coagulopathy (Vanholder *et al.,* 2000). Ultimately, myoglobin-induced acute kidney failure can progress to multiple organ failure and death. Ultimate mortality may result from acute renal failure, cardiac arrest, or cardiovascular collapse.

Predisposing Factors

Capture myopathy (CM) has been reported in a wide range of species, including mammals, birds, and reptiles (Di Marco *et al.,* 2014). Multiple factors contribute to the development of CM. Species susceptibility plays a key role, with prey species particularly ungulates and bovids and long-legged wading birds, being more vulnerable (Ali *et al.,* 2023). Adverse environmental conditions such as high temperatures, rainfall, and elevated humidity levels can exacerbate the risk. Capture-related factors, including the technique used, sustained pursuit, physical injuries, excessive handling, prolonged restraint, and crating, are also significant contributors. Additionally, underlying health conditions (Landau *et al.,* 2012), nutritional imbalances (Vitamin E or selenium deficiency, obesity), certain anaesthetic protocols (especially those involving opioid-based drug combinations), and animal characteristics such as age (very young or old), sex, and physiological state (pregnancy) further influence the likelihood of CM (Krauer, 2024). While any vertebrate species may develop CM under extreme physiological stress, multiple risk factors have been identified that increase susceptibility. Factors including high-velocity pursuit, prolonged chase duration, entanglement scenarios, or unnatural transport positioning can precipitate CM. Pre-existing morbidities such as parasitic infestations or renal dysfunction increase susceptibility. Medications inducing excitation, muscular rigidity, respiratory depression, or peripheral vasoconstriction may contribute to CM development. Neonatal, geriatric, or gravid animals exhibit elevated risk profiles (Paterson, 2014; Breed *et al.*, 2019; Dinesh *et al.*, 2020).

Clinical Manifestations

The clinical signs of capture myopathy can vary depending on the species affected and the intensity or duration of exertion. Symptoms may manifest within hours, days, or even up to two months following the capture event. The most commonly affected muscle groups include the large muscles of the limbs, as well as the pectoral, intercostal, and cardiac muscles. Notably, the lesions tend to be bilateral and symmetrical in distribution. The majority of clinical signs associated with CM result from severe exertional stress, myalgia secondary to muscle trauma, and metabolic derangements, particularly acidosis. Frequently documented clinical presentations including lethargy and depression, muscular rigidity and stiffness, recumbency with inability to maintain upright posture, ataxia and incordination, paresis or complete paralysis, hyperthermia, obtundation and altered mental status and tachypnea. The most common clinical signs of capture myopathy include depression, lethargy, unresponsiveness to human presence, muscle stiffness or weakness, tremors, ataxia, firm or exaggerated stepping, tachycardia, open-mouth and rapid breathing, hyperthermia, and the presence of red-brown urine (Krauer, 2024). Certain cases may result in acute mortality without preceding clinical symptomatology (Businga *et al.*, 2007; Herráez *et al.*, 2007; Ward *et al.*, 2011; Paterson, 2014; Lubbe *et al.*, 2021). While these represent the most frequently observed manifestations, CM presentations demonstrate considerable variation depending on species-specific factors and individual case history. Unfortunately, once clinical signs become apparent, the prognosis for successful recovery remains generally guarded (Paterson, 2014; Breed *et al.*, 2019).

Diagnostic Protocols

Ante-mortem diagnosis of capture myopathy relies predominantly on species identification, comprehensive case history, and clinical observation (Paterson, 2014). While no single diagnostic test provides definitive confirmation, serum biochemistry and urinalysis constitute valuable diagnostic modalities (Rokde *et al.*, 2023).

The most diagnostically significant biomarkers are the enzymes creatine kinase (CK) and aspartate aminotransferase (AST), both intracellular components of skeletal and cardiac myocytes. Elevated CK concentrations represent the most sensitive and specific indicator of myocyte damage in both avian and mammalian species (Businga *et al.*, 2007; Ward *et al.*, 2011). Significant elevations in both CK and AST, combined with compatible clinical signs, provide strong diagnostic support for CM (Businga *et al.*, 2007; McEntire and Sanchez, 2017).

Hyperkalemia may be observed secondary to myocyte breakdown, although this finding may result from alternative etiologies. Myoglobinuria, characterized by dark brown-pigmented urine, indicates severe myonecrosis and suggests potential or active renal injury or failure (Paterson, 2014). In avian species, elevated uric acid concentrations may occur but can result from unrelated pathological processes. Therefore, such findings require cautious interpretation (Ward *et al.*, 2011).

Post-Mortem Findings

Necropsy findings may vary but frequently include dark, edematous kidneys, haemorrhage within skeletal musculature, pale or discolored regions in skeletal or cardiac muscle, frank haematemesis or melena, dark-pigmented urine in the urinary bladder (Herráez *et al.,* 2007; Cattet *et al.,* 2008; Paterson, 2014; Breed *et al.,* 2019; Dinesh *et al.,* 2020). In certain cases of acute mortality, gross post-mortem findings may be minimal or absent, complicating diagnostic determination (Herráez *et al.*, 2007).

Therapeutic Management

Effective therapeutic management of capture myopathy in captive care settings requires early recognition and frequently aggressive intervention protocols. Initial treatment focuses on stress minimization, thermoregulation, restoration of tissue perfusion, and analgesic management (Paterson, 2014). During recovery phases, passive or active physiotherapy is often required to restore function in damaged musculature (McEntire and Sanchez, 2017).

Immediate Response and Stabilization

Upon suspected CM diagnosis, all handling, restraint, pursuit, or transport activities must be immediately discontinued (Ward *et al.*, 2011). The affected animal should be placed in a thermoneutral, quiet, and dimly illuminated environment to minimize stress while preparing for therapeutic intervention. This controlled environment enhances treatment efficacy. Supplemental oxygen therapy is recommended when available (Paterson, 2014).

In animals with confirmed hyperthermia, immediate, non-contact cooling methods are essential, which include application of isopropyl alcohol to glabrous regions, utilization of fans for convective cooling, placement of towel-wrapped cryotherapy packs in the enclosure and administration of room-temperature or cool subcutaneous fluid therapy. Once the animal demonstrates sufficient stability for handling, therapeutic intervention should commence with continuous monitoring for signs of decompensation.

Fluid Therapy

Fluid therapy constitutes the cornerstone of CM management. Ideally, an indwelling intravenous catheter should be placed for continuous fluid infusion utilizing drip sets or infusion pumps. Fluid therapy restores tissue perfusion, enhances oxygen delivery, facilitates carbon dioxide elimination, and buffers metabolic acidosis by reducing lactate accumulation and normalizing blood pH (McEntire and Sanchez, 2017). Adequate hydration improves renal perfusion, promoting

myoglobin and potassium elimination while preventing or mitigating renal injury. Alternative fluid delivery methods include bolus administration via butterfly catheter when intravenous access is suboptimal, or subcutaneous administration when vascular access is not feasible. Balanced crystalloid solutions are typically employed. In cases of suspected hyperkalemia, 0.9% sodium chloride solution (without potassium supplementation) represents a safer alternative.

Analgesic Management

Inadequate pain control contributes to continued physiological stress and condition deterioration. Analgesic options include:

1. ***Opioid analgesics:*** Provide superior analgesia, though potent μ-opioid agonists (morphine, fentanyl, hydromorphone) may induce respiratory depression and hyperthermia. Careful dosing and monitoring are essential (Plumb, 2005).
2. ***Non-steroidal anti-inflammatory drugs (NSAIDs):*** Effective for pain and inflammation management but may compromise renal function, particularly in dehydrated subjects. Should be used judiciously and only when renal function is stable (Plumb, 2005; Dinesh *et al.*, 2020).
3. ***Muscle relaxants:*** Muscle relaxants are beneficial in the management of capture myopathy, as they help relieve muscular rigidity and the associated pain. By reducing muscle tension, these drugs can improve the animal's comfort and mobility during recovery. They may also help prevent further muscle damage caused by sustained contractions. The use of muscle relaxants should be carefully monitored and customized to the species and condition of the animal to ensure both efficacy and safety.

Sedation and Anxiolytic Therapy

For animals demonstrating continued distress or excessive activity, sedatives or anxiolytics may be beneficial (Paterson, 2014; Breed *et al.*, 2019). Benzodiazepines provide both sedation and muscle relaxation but may cause paradoxical excitation in young or otherwise healthy animals when used as monotherapy. A multimodal pharmacological approach is recommended to optimize therapeutic effects (Plumb, 2005; Ward *et al.*, 2011).

Vitamin E and Selenium Supplementation

Deficiencies in vitamin E and selenium constitute recognized risk factors for CM development (Paterson, 2014). Although their definitive therapeutic role remains uncertain, these nutrients enhance endogenous antioxidant capacity. Despite limited evidence for therapeutic efficacy (Liu *et al.*, 2019), many practitioners administer single doses of both compounds at intake for potential benefits (Businga *et al.*, 2007; Hurtado *et al.*, 2021).

Sodium Bicarbonate Therapy

Intravenous sodium bicarbonate has historically been utilized to correct metabolic acidosis. However, due to variable therapeutic efficacy and risk of pH overcorrection without appropriate blood gas monitoring, its clinical use is declining (Breed *et al.*, 2019; Dinesh *et al.*, 2020). In most wildlife rehabilitation facilities lacking blood gas analysis capabilities, oxygen therapy and fluid resuscitation represent preferred methods for managing acidosis.

Rehabilitation and Long-Term Care

Animals surviving the acute phase of CM require ongoing supportive care, particularly physiotherapy, to prevent decubitus ulceration in recumbent animals and stimulate controlled muscular recovery. Muscle regeneration may require weeks to months, particularly in severe cases. Wildlife caretakers must evaluate the feasibility of extended care, especially for species poorly adapted to prolonged captivity (McEntire and Sanchez, 2017).

Rehabilitation protocols should be individualized based on species, clinical condition, and temperament, and may include: hydrotherapy or aquatic exercise, assisted standing with support slings, short-term sling housing systems, passive range-of-motion exercises administered by caretakers (Ward *et al.,* 2011; Paterson, 2014; McEntire and Sanchez, 2017).

Capture Myopathy and Challenge to Wildlife Conservation

Capture myopathy (CM) presents a significant challenge to wildlife conservation, as it can adversely affect the health, welfare, and survival of individual animals and entire populations. Conservation initiatives and scientific research frequently involve the capture and relocation of animals for purposes such as reintroduction, translocation, or population supplementation (Hartup *et al.,* 1999). However, the stress associated with capture and handling significantly elevates the risk of CM, potentially undermining the success of these programs and compromising animal welfare (Breed *et al.,* 2019; Herráez *et al.,* 2013). The threat is particularly serious for endangered or vulnerable species with small or fragmented populations, where the loss of even a few individuals due to capture-related injury or mortality can have substantial consequences for the population's long-term viability (Di Marco *et al.,* 2014). Even in cases where capture and release are initially successful, affected individuals may experience delayed mortality or suffer from reduced fitness due to CM-related complications (Rosenhagen, 2023). Therefore, long-term post-release monitoring is essential to evaluate the health and survival of relocated animals, understand the broader impacts of capture-induced stress on population dynamics, and guide adaptive management strategies (Miller *et al.,* 2013). Capture myopathy remains a major concern for wildlife management. Despite its significance, there is still

limited understanding of the underlying pathophysiology, triggers, and predisposing factors that contribute to the onset of CM. Mitigating the risk of this condition requires meticulous planning and the implementation of capture protocols that prioritize minimizing stress and physical exertion, along with strategies for early detection and timely intervention in affected individuals. In addition, efforts to improve habitat quality, address nutritional deficiencies, and promote the overall welfare of wild populations can play a critical role in reducing both the incidence and severity of capture myopathy. Expanding this body of knowledge is essential to safeguard animal welfare and enhance survival outcomes particularly for endangered species already facing population pressures (Garces *et al.,* 2024).

Present review article contributes to the much-needed knowledge on capture myopathy and wildlife conservation by providing detailed insights into pathophysiological mechanisms underlying capture myopathy involving complex multisystemic alterations that demonstrate progressive deterioration without immediate therapeutic intervention by understanding the factors influencing capture myopathy and the dynamics of pathological changes. Last but not least, more detailed study should be considered for long-term conservation.

Conclusion

As with any pathological condition, prevention represents a superior approach compared to therapeutic intervention. Preventive measures include limiting pursuit duration, handling procedures, and transport of wild animals, particularly in hazardous environments and high-risk species. Furthermore, controlling underlying pathological conditions prior to stressful events when possible, and restricting handling to experienced personnel, will further reduce capture myopathy (CM) risk. In general, comprehensive planning and communication protocols should be implemented during capture or care of wild animals. Despite optimal preventive measures and careful planning, CM remains a clinical possibility. Although the prognosis in affected animals is generally guarded, successful cases have been documented and continue to be reported. Therefore, practitioners are encouraged to pursue cases with aggressive and timely therapeutic interventions to provide animals with optimal recovery potential and successful return to natural habitats.

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Announcement - I

XII Annual Convention and International Conference of SVSBT-2025

XII Annual Convention of the Society for Veterinary Sciences & Biotechnology (SVSBT) and International Conference on "Bridging Science and Society: Biotechnology for Sustainable One Health" will be organized by College of Veterinary Sciences, LUVAS, Hisar-125004, Haryana, India, during 3rd to 5th December, 2025. The detailed Brochure cum Final Announcement showing Theme Areas / Sessions, Registration Fee, Bank Details for online payment and deadlines, etc. has been floated on the **society's website: https://www.svsbt.com/conference/registration.php** and also on the Whatsapp group and e-mails. The organizing committee invites abstracts of original and quality research work on theme areas of any of the eight technical sessions of seminar limited to 250-300 words for oral and poster presentations through conference website: https:www.svsbt.com//conference/ on or before 30th October, 2025 or, by e-mail to: svsbt2025@gmail.com for inclusion in the Souvenir cum Compendium to be published on the occasion.

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