

ARTICLE

Capture myopathy

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Abstract

Capture myopathy is a condition that manifests after prolonged pursuit, entrapment, restraint, live-stranding and/or transport. Its effects are wide-ranging and often life-threatening. All wild animals are susceptible to this condition, though particular species seem to be predisposed. A diagnosis of capture myopathy revolves around the animal's natural and individual history as well as any clinical signs, although, in many cases, a definitive diagnosis cannot be made or is only determined post-mortem. Successful management often relies on aggressive fluid support, pain management and physical therapy.

BIO

Dr Rosenhagen is the full-time clinical veterinarian at PAWS, where she cares for injured and orphaned wildlife, trains veterinary students and interns and travels around the country to lecture and offer educational opportunities to other professionals in the field.

Background

CM, also known as exertional rhabdomyolysis, white muscle disease and stress myopathy, is a non-infectious metabolic disease with significant morbidity and mortality potential (Paterson 2014; Dinesh et al. 2020). It was first described in a Hunter's antelope (*Beatragus hunteri*) in 1964 and has since been reported in other terrestrial mammals, marine mammals, birds and reptiles, though ungulates and long-legged wading birds are overrepresented in the literature (Jarrett et al. 1964; Paterson 2014; Breed et al. 2019). A similar condition has been described in humans and is most often attributed to military training, endurance sports and severe crushing injuries (Paterson 2014). In animals, CM is caused by prolonged pursuit, entrapment, restraint, live-stranding (marine mammals) and/or transport (Cattet et al. 2008; Herráez et al. 2013; Breed et al. 2019; Dinesh et al. 2020; Hurtado et al. 2021). Some theories propose it evolved as a symbiotic relationship between predator and prey wherein an affected prey animal experiences a rapid death, and the predator conserves energy; however, most experts believe the condition is purely iatrogenic and does not occur in natural circumstances (Breed et al. 2019).

CM is classified into four syndromes based on history and clinical signs. *Capture shock syndrome* results in sudden death during capture or within a few hours after an event. *Ataxic myoglobinuric syndrome* manifests hours to days after the inciting event and may result in visible changes to skeletal and cardiac muscles. Animals

suffering from the third classification, *ruptured muscle syndrome*, may initially appear normal and then acutely rupture a muscle, especially in the hindquarters, resulting in abnormal limb position and severe permanent injury. The final form, *chronic debility syndrome*, is the rarest and occurs in animals with a history of previous pursuit. On a subsequent pursuit, the animal abruptly stops, its eyes dilate and it dies. In all syndromes, significant abnormalities in blood chemistry analytes have been reported. Of the above, ataxic myoglobinuric syndrome is the most reported and the one with the most documented management success (Spraker 1993).

Pathophysiology

The pathophysiology of CM involves several concurrent systemic changes in the affected animal, many of which are progressive without intervention. Overexertion or prolonged compression of skeletal muscles, particularly those of the limbs and pectorals, leads to necrosis and release of intracellular potassium and myoglobin into the blood (Cattet et al. 2008; Dinesh et al. 2020). Simultaneously, altered blood flow from muscle compression and/or insufficient nutrient and oxygen delivery to tissues from extreme demand results in diffuse tissue hypoxia, metabolic acidosis and cell dysfunction, exacerbating muscle cell degeneration and necrosis (Paterson 2014; Breed et al. 2019; Dinesh et al. 2020). Myoglobin is an intrinsically nephrotoxic protein and exerts damage by further constricting vessels and reducing blood flow

Keywords

Exertional rhabdomyolysis; white muscle disease; stress; wildlife rehabilitation

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Abbreviations

CM: capture myopathy;
CK: creatine kinase;
AST: aspartate aminotransferase;
NSAID: non-steroidal anti-inflammatory drug

Dates

Accepted: 20 November 2023
Published: 15 December 2023

to the kidneys as well as precipitating into casts in the renal tubules, leading to tubular necrosis. Myoglobin's effects are compounded by dehydration (Vanholder et al. 2000; Herráez et al. 2007; Breed et al. 2019). Abnormally, high levels of circulating potassium from cell destruction can lead to cardiac arrhythmias and asystole (Montané et al. 2002). In many cases, concurrent hyperthermia from associated exertion and stress leads to additional widespread tissue dysfunction and necrosis (Rae et al. 2008). Death may result from renal failure, cardiac failure or circulatory collapse.

Predisposing factors

While any animal may be at risk of CM in the right circumstances, a compilation of predisposing factors has been identified that seem to increase an animal's probability of succumbing to this condition. The mnemonic SECONDS is used to describe the factors (Paterson 2014; Breed et al. 2019; Dinesh et al. 2020).

- S—species: prey animals in general, ungulate and long-legged wading bird specifically
- E—environment: extreme heat and humidity; uneven or slippery terrain
- C—capture conditions: high chase speeds, prolonged pursuit, entanglement in nets or traps or unnatural positioning during prolonged transport
- O—other disease: underlying disease or infection, such as endoparasitism or pre-existing renal dysfunction
- N—nutrition: obesity; animals deficient in vitamin E and/or selenium
- D—drugs: pharmaceuticals that cause secondary excitement, muscle rigidity, hypoventilation or altered peripheral perfusion
- S—signalment: very young, old or pregnant animals

Clinical signs

Most clinical signs associated with CM are secondary to extreme exertion, pain from severe muscle trauma and physiologic efforts to correct metabolic acidosis. Lethargy, muscle stiffness, recumbency, ataxia, paresis or paralysis, hyperthermia, obtunded mentation and tachypnoea are reported most often, though acute death without clinical signs may also occur (Jarrett et al. 1964; Businga et al. 2007; Herráez et al. 2007; Ward et al. 2011; Paterson 2014; McEntire & Sanchez 2017; Dinesh et al. 2020; Lubbe et al. 2021). While the abovementioned signs are the most common, the list of clinical signs reported in animals suffering from CM is extensive and varies by species and individual history. Unfortunately, once clinical

signs manifest, the prognosis for successful treatment is generally poor (Paterson 2014; Breed et al. 2019).

Diagnosis

An antemortem CM diagnosis relies strongly on the animal's natural and individual history as well as any clinical signs that have manifested (Paterson 2014). Blood chemistry and urine tests are also beneficial. CK and AST are intracellular enzymes predominantly found within skeletal and cardiac muscle. Increased CK is the most sensitive and specific index of muscle damage in birds and mammals (Businga et al. 2007; Ward et al. 2011); significant increases in both CK and AST in conjunction with clinical suspicion are strongly supportive of CM (Businga et al. 2007; McEntire & Sanchez 2017). Increased levels of circulating potassium may also be present, though potassium may increase secondary to other aetiologies. The presence of myoglobinuria, grossly apparent as brown-coloured urine, may be indicative of severe muscle trauma and impending or active renal damage or failure (Paterson 2014). Uric acid increases in birds may be variable and secondary to unrelated aetiologies and should thus be interpreted with caution (Ward et al. 2011). Post-mortem findings include dark, swollen kidneys, haemorrhage within the skeletal muscle, pale areas within skeletal or cardiac muscles, frank blood or blood-tinged contents in the lumen of the intestine and/or dark urine within the urinary bladder (Herráez et al. 2007; Cattet et al. 2008; Breed et al. 2019; Paterson 2014; Dinesh et al. 2020). In cases of acute death, a gross necropsy may be unremarkable (Herráez et al. 2007).

Treatment

Successful management of CM in a captive care setting relies on timely and often aggressive therapy. Early treatment revolves around limiting ongoing stress and struggle, controlling body temperature, improving perfusion to compromised tissues and managing pain (Paterson 2014). Later, management frequently includes passive or active physical therapy to restore function to damaged muscles (McEntire & Sanchez 2017).

In acute cases where CM is likely or suspected, ongoing pursuit, restraint or transport must be ceased as soon as possible (Ward et al. 2011). For animals in captive care situations, the animal should be placed in a cool, dark and quiet area, while the caretaker gathers supplies for active management. This will afford the animal time to calm down while improving the efficiency of treatment when it is implemented. Supplemental oxygen may also be beneficial when available (Paterson 2014). In cases of

confirmed hyperthermia, brief, hands-off cooling efforts are critical, including application of isopropyl alcohol on unfurred/featherless areas of the body, use of a fan, adding a towel-covered cold pack to the enclosure and/or cool or room temperature subcutaneous fluids (Breed et al. 2019). Once the animal is deemed stable enough for additional handling, treatment may be initiated, though regular monitoring throughout the procedure is necessary to monitor for signs of decompensation.

Initial stabilization efforts should involve fluid therapy and analgesia at a minimum; additional treatments may include muscle relaxers, sedatives/anxiolytics, vitamin E and selenium supplementation, and/or sodium bicarbonate (Paterson 2014; Breed et al. 2019).

The benefits of fluid therapy cannot be overstated. Ideally, an indwelling catheter is placed to administer a constant flow of fluids via pump or drip set. Intravenous fluids rapidly improve perfusion to tissues to increase oxygen delivery and carbon dioxide elimination, thereby reducing ongoing lactic acid production and increasing the pH in an acidotic animal. Additionally, adequate perfusion of the kidneys facilitates removal of myoglobin and potassium and may reduce or prevent renal damage (McEntire & Sanchez 2017). If an indwelling catheter cannot be placed, a bolus into the vasculature via butterfly catheter is also beneficial. Subcutaneous fluids, while suboptimal, will still provide needed fluid therapy when vascular access is not feasible due to patient or caretaker limitations. In the author's experience, balanced crystalloid solutions are appropriate, though in cases of presumed hyperkalemia, options without added potassium, such as 0.9% sodium chloride, may be a safer option.

Unmanaged pain in an afflicted animal will contribute to ongoing distress and anxiety, thus exacerbating the disease (Dinesh et al. 2020). Many drugs will provide analgesia, including opioids, NSAID and muscle relaxers. Opioids have the capacity to offer the most robust analgesia; however, potent mu opioid drugs, like morphine, fentanyl and hydromorphone, have the potential to lead to respiratory depression and hyperthermia, so drugs and dosages must be selected carefully (Plumb 2005). Decreased renal perfusion is a well-known possible side effect of NSAID use, so these drugs should likewise be used judiciously and ideally only when the animal is euhydrated, and renal function is stable (Plumb 2005; Dinesh et al. 2020).

Sedative or anxiolytic drug use may be beneficial for animals that display ongoing signs of distress and/or excessive activity while in care (Paterson 2014; Breed et al. 2019). In addition to reducing anxiety and causing sedation, benzodiazepine drugs have the added benefit of relaxing muscles; however, paradoxical excitability may be an unwanted side effect when these drugs are

used alone in young, otherwise healthy animals, so multimodal drug use is recommended (Plumb 2005; Ward et al. 2011).

Vitamin E and selenium deficiencies have long been implicated in increasing an animal's susceptibility to CM (Paterson 2014), and while both vitamin E and selenium are known to improve the effect of antioxidants in the body, there is very little data to support nutritional supplementation as playing a definitive role in managing or preventing this condition (Liu et al. 2019). Regardless, many caretakers, including the author, elect to administer a single injection of vitamin E and selenium at the time of intake in CM suspects for the potential antioxidant benefits (Businga et al. 2007; Paterson 2014; Dinesh et al. 2020; Hurtado et al. 2021).

Historically, intravenous sodium bicarbonate has been used to correct metabolic acidosis, but this medication is falling out of favour due to variable efficacy and the possibility of overcorrection of pH imbalances without careful monitoring of blood gases (Breed et al. 2019; Dinesh et al. 2020). In most wildlife rehabilitation settings, close monitoring of blood pH is not feasible, so supplemental oxygen and fluid therapy are often the preferred method to facilitate correction of metabolic acidosis states.

For animals that survive the initial acute CM episode, ongoing care should include physical therapy considerations to prevent pressure lesions in recumbent animals and encourage controlled muscle strengthening in debilitated animals (Ward et al. 2011; Paterson 2014; McEntire & Sanchez 2017). In severe cases, muscle regeneration may require weeks to months for adequate tissue repair and strengthening, which may be an important consideration for the wildlife rehabilitator before embarking on cases in species unlikely to endure long-term captivity and handling (McEntire & Sanchez 2017). The most appropriate physical therapy option will vary by the animal's tolerance, its natural history and clinical signs but may include swimming or hydrotherapy, assisted standing, short-term housing in slings and passive range of motion by the handler (Ward et al. 2011; Paterson 2014; McEntire & Sanchez 2017).

Conclusions

As with any disease, prevention is a far better goal than treatment. Preventative measures include limiting the duration of pursuit, handling and transport of wild animals, particularly in dangerous environments and high-risk species. Moreover, controlling underlying disease before a stressful event when possible, and limiting handling to experienced personnel, will further reduce the risk of CM (Paterson 2014; Dinesh et al. 2020).

In general, good planning and communication should always be implemented during the capture or care of wild animals. Even in the face of the best laid plans and prevention, CM remains a possibility. Despite the generally poor prognosis in affected animals, successful cases have been and continue to be reported, so the reader is encouraged to pursue cases with aggressive and timely care to offer the animal the strongest chance of recovery and return to the wild.

Disclosure statement

The author reports no conflict of interest.

Funding

No financial support was utilized.

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