



Serum Biochemical Alterations Associated with Exertional Myopathy in Free-ranging Spotted Deer (*Axis axis*) and Barking Deer (*Muntiacus muntjak*)

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ABSTRACT

Background: Exertional myopathy (EM), also referred to as capture myopathy, is a fatal, stress-induced, non-infectious metabolic disorder that affects a wide range of wild mammals and birds following events involving intense physical exertion or psychological stress. In India, species such as spotted deer (*Axis axis*) and barking deer (*Muntiacus muntjak*) are frequently subjected to stressful conditions including rescue, capture, translocation, physical restraint and transportation, which predispose them to EM. However, limited research exists on the clinicopathological aspects of EM in these free-ranging cervid species.

Methods: The present study aimed to investigate the serum biochemical alterations associated with EM in spotted deer and barking deer under field conditions. Free-ranging cervids (spotted deer, $n = 26$; barking deer, $n = 7$) with a history of trauma, dog attack, vehicular collision, physical restraint, or capture were assessed during rehabilitation. Serum samples were analysed for key muscle injury biomarkers, including creatine kinase (CK), lactate dehydrogenase (LDH) and aspartate aminotransferase (AST).

Result: Significantly elevated levels of LDH and AST were consistently observed in animals exhibiting clinical signs suggestive of EM, whereas CK showed variable elevations. Among the three markers, LDH emerged as the most reliable indicator of muscular damage. This study provides the first systematic documentation of serum biochemical changes associated with EM in free-ranging spotted and barking deer. The findings highlight the diagnostic utility of LDH, CK and AST as biomarkers for EM and emphasize the need for precautionary measures to reduce stress during wildlife handling operations to prevent exertional fatalities in vulnerable cervid populations.

Key words: Barking deer, Capture, Cervid, Exertional myopathy, Serum biomarkers, Spotted deer.

INTRODUCTION

Rapid anthropogenic expansion, including agricultural intensification, urbanization and infrastructure development, has led to widespread habitat degradation and fragmentation, posing significant threats to global biodiversity (Bencin *et al.*, 2019; Pimm *et al.*, 2014). In the Indian subcontinent, these environmental pressures have increasingly brought wild animals into conflict with human activities, resulting in elevated incidences of wildlife injuries, displacement and mortality (Kumar *et al.*, 2019; Padmakumar and Shanthakumar, 2023). Among the most affected are free-ranging cervids, which often encounter threats such as vehicular collisions, attacks by feral dogs and trauma during rescue or translocation procedures (Deem *et al.*, 2001; Gentsch *et al.*, 2018).

To mitigate such human-wildlife conflicts and support conservation programs including rescue, translocation and rehabilitation, wild animals are frequently captured and physically restrained (Massei *et al.*, 2010; Somu and Palanisamy, 2023). While necessary, these interventions subject animals to acute physical exertion and psychological stress, predisposing them to a potentially fatal stress-related condition known as EM, or capture myopathy (Spraker, 1993; La Grange *et al.*, 2010; Dinesh *et al.*, 2020). EM is a non-infectious, metabolic disorder characterized by extensive skeletal muscle degeneration

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(rhabdomyolysis), metabolic acidosis, myoglobinuria and multi-organ dysfunction, often culminating in high mortality rates if not promptly diagnosed and managed (Paterson, 2007; Dinesh *et al.*, 2020; Krauer *et al.*, 2024).

The pathophysiology of EM involves a cascade of stress-induced physiological responses, including

sympathetic overactivation, hypoxia and lactic acidosis, leading to cellular injury, mitochondrial dysfunction and widespread myofiber necrosis (Breed *et al.*, 2019). Clinically, EM manifests as muscle stiffness, tremors, ataxia, tachycardia, hyperthermia, dyspnea and dark-colored urine due to myoglobinuria, with prognosis often poor once overt signs appear (Krauer *et al.*, 2024).

Few studies in captive carnivores were carried out including antioxidants and stress in captive leopards (Sarode *et al.*, 2024), evaluation of hepatobiliary system in leopards (Rai *et al.*, 2018) and biochemical indices of captive tigers (Allwin *et al.*, 2019). Although EM has been extensively documented in various wild species, there remains a significant lack of focused research on its occurrence in free-ranging cervids of the Indian subcontinent. In particular, species such as spotted deer is predominantly represented by anecdotal reports or isolated case studies with limited investigations exploring the associated biochemical alterations and there are no scientific reports of such studies in barking deer.

This knowledge gap is alarming, especially given that these cervids often endure stressors such as capture, translocation, predator attacks and rehabilitation efforts all established triggers for EM. Moreover, as key herbivores in forest ecosystems and primary prey for apex carnivores, a decline in their populations due to under diagnosed EM could disrupt ecological dynamics and conservation goals (Rubenstein, 2014; Rathore *et al.*, 2012).

Serum biochemical studies of free-ranging gaur (*Bos gaurus gaurus*) were carried out by Shrivastav *et al.* (2014) during reintroduction efforts to establish baseline physiological reference values, highlighting the importance of health assessment of large herbivores as an essential conservation tool for monitoring population fitness. Limited research has been conducted on exertional (capture) myopathy in spotted deer (*Axis axis*) and barking deer (*Muntiacus muntjak*), despite their significant ecological importance; notably, no prior studies have documented capture myopathy in barking deer, highlighting a critical gap in wildlife health and conservation research. The present study aims to address this critical void by systematically evaluating the serum biochemical parameters associated with EM in free-ranging spotted deer and barking deer under field conditions. By documenting alterations in key muscle injury biomarkers such as creatine kinase (CK), lactate dehydrogenase (LDH) and aspartate aminotransferase (AST). These findings are anticipated to enhance species-specific understanding of EM pathogenesis and support evidence-based clinical decision-making during wildlife rescue, rehabilitation and conservation translocation efforts.

MATERIALS AND METHODS

Sample collection

The work was conducted in the School of Wildlife Forensic and Health, Jabalpur. The samples were collected from the

two free-ranging herbivore species including spotted deer (n=26) and barking deer (n=7) with history of trauma, accident, physical capture, ptialism with blood tinged, dyspnea, depression, lameness, ataxia, increased heart rate, shivering, dog chase and bite marks from tiger reserves and in an around Jabalpur forest division Madhya Pradesh. These biochemical markers have different half-lives (some short, some long), hence blood samples were collected at an earliest after onset of injury. All the animals were physically restrained for treatment and rehabilitation procedure. Blood samples 2-4 ml of were collected from properly restrained animals in vacutainers without anticoagulant from the jugular vein aseptically. The samples were transferred in cold chain to laboratory for further analysis.

Serum biochemical analysis

The serum was separated in the laboratory and serum samples were stored at -20°C prior to analysis. Muscle injury markers such as creatine kinase (CK), lactate dehydrogenase (LDH) and aspartate aminotransferase (AST) were examined in serum samples using a semi-automatic biochemical analyzer with commercially available kits. Two-sample independent t-test was applied to compare the mean responses of the affected and unaffected animals in both the species.

RESULTS AND DISCUSSION

Stress, exertion and crush injury are well-documented causes of rhabdomyolysis. However, other major factors contribute to the development of exertion myopathy in wildlife, including procedures that involve long periods of restraint, struggling with unnatural positioning and lengthy pursuit during capture (Ashraf *et al.*, 2019; Herráez *et al.*, 2007). Diagnosis is based on clinical history, observed symptoms, clinical pathology and gross and microscopic examination of tissues (Dinesh *et al.*, 2020). In the present study, wild animals suffering from myopathy showed elevated muscle enzyme levels (Table 1). In spotted deer, the median creatine kinase (CK) level was 166.43 IU/L (range: 66.88-406.22 IU/L), the median lactate dehydrogenase (LDH) level was 2205.06 IU/L (range: 1346.86-3011.86 IU/L) and the median aspartate aminotransferase (AST) level was 196.53 IU/L (range: 91.73-386.02 IU/L) (Fig 3). In barking deer, the median CK level was 78.20 IU/L (range: 22.46-168.24 IU/L) (Fig 4), the median LDH level was 1898.64 IU/L (range: 1384.78-2609.58 IU/L) (Fig 5) and the median AST level was 209.96 IU/L (range: 114.08-316.36 IU/L) (Fig 6). The creatine kinase (CK) was significantly elevated in the blood of animals with a history of muscle injury and myopathy in spotted deer (Fig 1) and barking deer (Fig 2). The CK mean values were lower than those of Baric *et al.* (2011) observed in red deer (*Cervus elaphus*). Although the mean values of CK were lower than the values observed in some of the previous studies, there was a large variation in our study reflecting overloaded or skeletal muscle injury. It is well known that cervids are very susceptible to stress; therefore, there are

several reports about variations in CK concentrations in cervids (Camargo *et al.*, 2013). Elevation of plasma creatine kinase (CK) levels is considered to be a sensitive and specific index of muscle damage in mammals (Bailey *et al.*, 1997).

In the present study, lactate dehydrogenase (LDH) concentrations were significantly higher in spotted deer

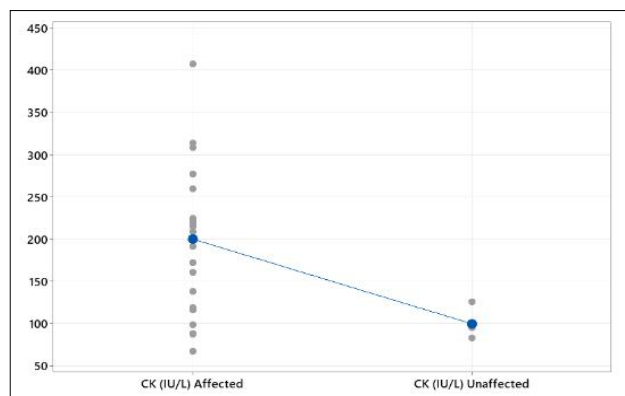


Fig 1: Creatine kinase (CK) levels in affected and unaffected spotted deer.

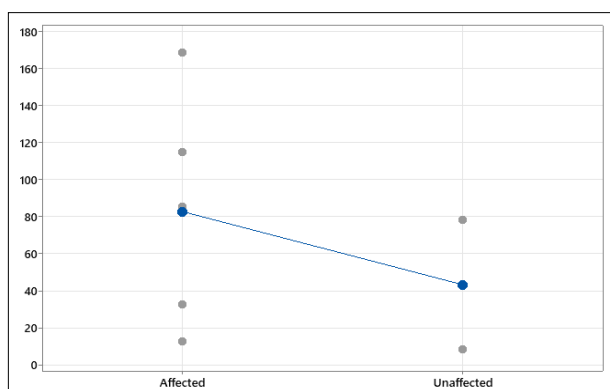


Fig 2: Creatine kinase (CK) levels in affected and unaffected barking deer.

(Fig 3) and insignificantly rose in barking deer (Fig 4). Deer frightened by capture showed markedly raised LDH (Jones and Price, 1992). Raised LDH in capture myopathy had been reported in different species (Wobeser *et al.*, 1976; Businga *et al.*, 2007; Paterson *et al.*, 2007). The raised LDH concentration in the present study is in agreement with the previous study of muscle disorders with myonecrosis (Green-Barber *et al.*, 2017). Duncan and Prasse (1986) also reported the higher serum levels of CK and AST in muscle damage.

The AST in the present study was found to be insignificantly higher in both spotted deer (Fig 5) and barking deer (Fig 6) in comparison to the reference values of AST in spotted deer 42.88 ± 5.97 IU/L (Gupta *et al.* (2007) and barking deer in the present study. Chapple *et al.* (1991) also reported elevated concentrations of CK and AST in spotted deer during myopathy. Elevated AST levels have been previously reported during capture myopathy across various species (Businga *et al.*, 2007; Paterson *et al.*, 2007; Green-Barber *et al.*, 2017). However, because a significant portion of AST originates from mitochondria, a substantial increase is likely to indicate irreversible cellular damage (Sodikoff, 2001). Serum AST concentrations increase more slowly in comparison to serum CK concentrations after an initial muscles damage. Serum AST has a longer half-life and persists longer at elevated concentrations compared with CK (Hartup *et al.*, 1999).

Increases in serum enzymes CK, AST and LDH are usually due to an increase in muscular cell permeability and muscular damage resulting from physical stress (Businga *et al.*, 2007; Montané *et al.*, 2002; Wallace *et al.*, 1987). Fig 7 and 8 depicts all three muscle injury markers were appreciably increased in concentration in the studied deer with muscle damage. The elevated concentration of CK, LDH and AST was observed in the individual animal indicated that all three markers show a uniform increase with the advent of muscle injury. In the present study, the muscle damage was mainly observed due to chasing by feral dogs, dog bites, improper restraint and transportation.

Table 1: Serum biochemical alterations associated with exertional myopathy in free-ranging spotted deer (*Axis axis*) and barking deer (*Muntiacus muntjak*).

		Spotted deer (<i>Axis axis</i>)			Barking deer (<i>Muntiacus muntjak</i>)		
		CK (IU/L)	LDH (IU/L)	AST (IU/L)	CK (IU/L)	LDH (IU/L)	AST (IU/L)
Mean		180.45	2252.74	196.53	75.64	1949.03	197.73
Standard error		17.71	89.96	14.48	20.17	163.71	31.19
Standard deviation		90.34	458.72	73.85	53.38	433.14	82.54
Range		339.34	1665	294.29	145.78	1224.8	202.28
Minimum		66.88	1346.86	91.73	22.46	1384.78	114.08
Maximum		406.22	3011.86	386.02	168.24	2609.58	316.36
Significance	Affected	204.76 ^a ±	2423.29 ^a ±	204.83 ^a ±	2085.57 ^a ±	182.25 ^a ±	2085.57 ^a ±
variation±SE		19.98	82.25	17.47	162.40	67.05	162.40
	Unaffected	99.58 ^b ±	1654.26 ^b ±	169.09 ^a ±	1592.32 ^a ±	209.14 ^a ±	1592.32 ^a ±
		7.08	84.23	23.16	124.80	45.90	124.80

These enzymes appeared elevated in many stressed wild ungulates and in those suffering from capture myopathy (Vassart *et al.*, 1992). Hartup *et al.* (1999) observed similar degrees AST and CK elevation in translocated otters (*Lutra canadensis*). Fitte (2017) evaluated serum enzymes (CK and AST) in blesbok (*Damaliscus pygargus Phillips*) specific to skeletal muscles and observed a higher concentration of these enzymes in chased animals in comparison to a control group of animals. Moreover, as AST, LDH and CK levels tend to increase progressively following muscle injury, a mild elevation in these parameters may be attributed to the time interval between the traumatic event and blood collection. Additionally, physical restraint and the injection procedure using a hand-held syringe can themselves induce stress, potentially contributing to these changes. In this case, a marked increase in AST, along with elevated levels of BUN, LDH and CK, was consistent with the findings typically associated with capture myopathy.

All animals were properly physically restrained for treatment and rehabilitation procedure and samples were

also collected during the procedure. On the other hand, biochemical analyses showed increased levels of CK and LDH, which are often related to stress-linked muscle damage (Stringer *et al.*, 2011). In general, serum CK levels increase rapidly following a stress episode and along with potassium (K⁺), it is regarded as a critical early indicator of rhabdomyolysis associated with capture operations (Kaneko, 1997). A value more than four times the normal upper limit of CK was found in the blood of the Corsican red deer (*Cervus elaphus corsicanus*) examined, indicating that muscular lesions were active prior to death. The present results indicate that the concentration of all three muscle injury markers in spotted deer and barking deer serum can be used to establish myopathy-related causes. Comparison of these biochemical markers in both species of animals in the present study indicated that the values of AST, LDH and CK are higher in chital as compared to barking deer. The parameters observed in the present work are in agreement with the findings of earlier workers and also support the findings of an increased concentration of muscle injury markers in the serum of affected animals.

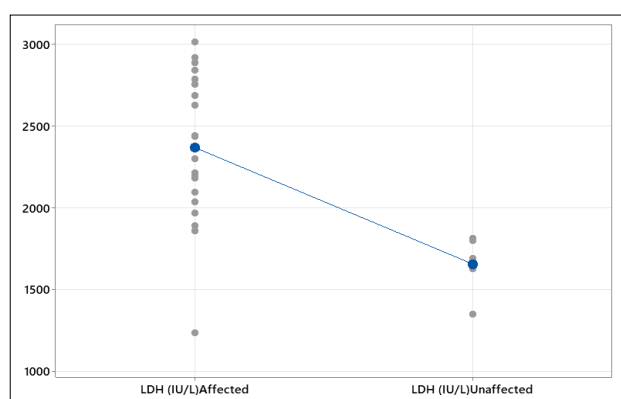


Fig 3: Lactate dehydrogenase (LDH) levels in affected and unaffected spotted deer.

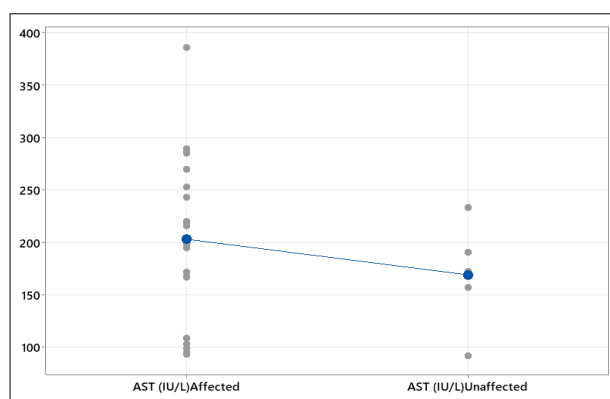


Fig 5: Aspartate aminotransferase (AST) levels in affected and unaffected spotted deer.

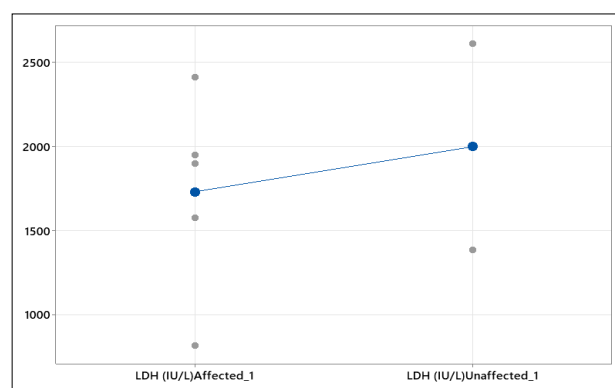


Fig 4: Lactate dehydrogenase (LDH) levels in affected and unaffected barking deer.

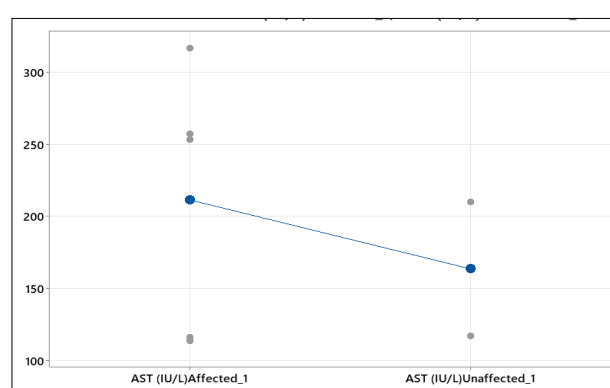


Fig 6: Serum aspartate aminotransferase (AST) levels in affected and unaffected Barking deer.

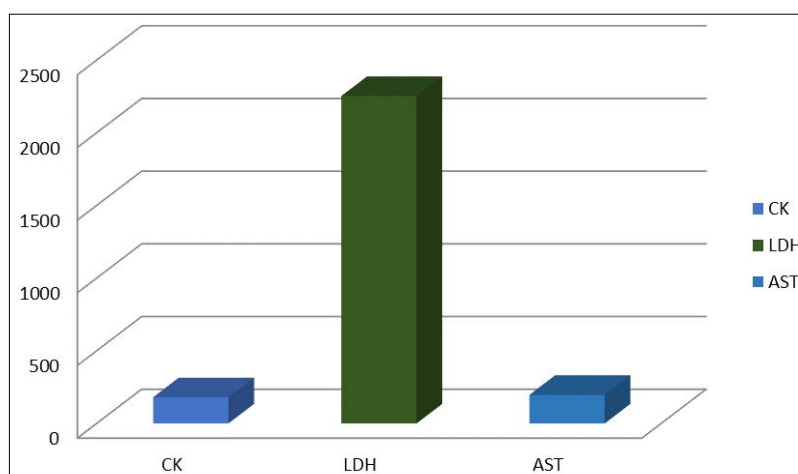


Fig 7: Mean concentration level of creatine kinase, lactate dehydrogenase and aspartate aminotransferase in chital.

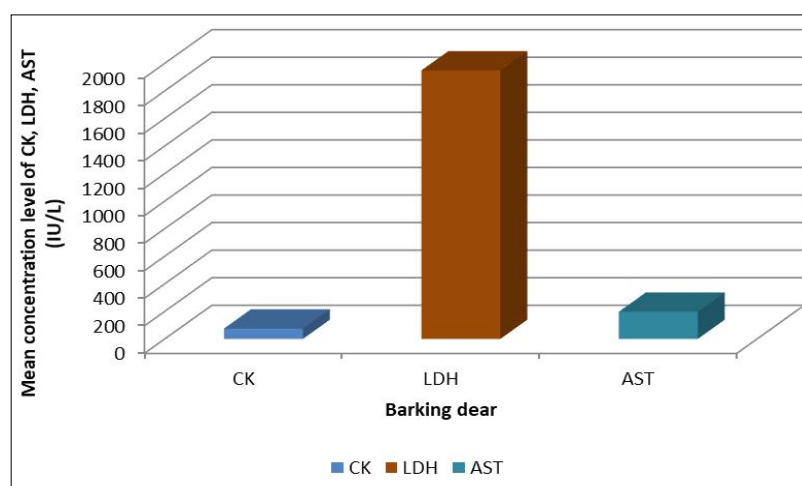


Fig 8: Mean concentration level of creatine kinase, lactate dehydrogenase and aspartate aminotransferase in barking deer.

CONCLUSION

The present study demonstrates that serum creatine kinase (CK), aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) serve as reliable biochemical indicators of muscle injury and exertional myopathy (EM) in free ranging spotted deer and barking deer. All three enzymes showed appreciable elevation in affected animals, with chital displaying comparatively higher values than barking deer. These findings highlight the marked susceptibility of cervids to stress and confirm that prolonged restraint, pursuit by feral dogs, physical trauma and improper handling are major contributors to muscle damage in free-ranging and rescued deer.

Overall, the study reinforces the diagnostic value of CK, AST and LDH in assessing stress-induced muscle injury in wild ungulates. Routine monitoring of these biomarkers during wildlife rescue, handling and rehabilitation can enhance clinical assessment, facilitate timely intervention and reduce mortality associated with

EM. As wild animal populations continue to decline due to habitat loss, deforestation, anthropogenic pressures and disease, minimizing preventable causes of mortality including exertional myopathy becomes increasingly vital. Implementing stress-mitigation strategies, humane capture practices and evidence-based rehabilitation protocols is therefore essential to improving wildlife welfare and supporting long-term conservation efforts.

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Disclaimers

The views and conclusions expressed in this article are solely those of the authors and do not necessarily represent the views of their affiliated institutions. The authors are responsible for the accuracy and completeness of the information provided, but do not accept any liability for any direct or indirect losses resulting from the use of this content.

Informed consent

All animal procedures for experiments were approved by the Committee of Experimental Animal care and handling techniques were approved by the University of Animal Care Committee.

Conflict of interest

The authors declare that there are no conflicts of interest regarding the publication of this article. No funding or sponsorship influenced the design of the study, data collection, analysis, decision to publish, or preparation of the manuscript.

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