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CAPTURE MYOPATHY IN WILDLIFE: PATHOMORPHOLOGICAL AND HISTOENZYMIC STUDIES

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SUMMARY

The study was designed to elucidate and correlate histopathological and histoenzymic alterations in capture myopathy in wildlife (spotted deer, mouse deer, barking deer and one-horned rhinoceros). Histopathology showed haemorrhage and disruption, hyalinization and loss of striations in cardiac and skeletal muscles. In chronic cases oedema was also noted. Histoenzymic study exhibited the evidence of alkaline phosphatase, adenosine-triphosphatase and dehydrogenases in these lesions. X-ray micronalysis indicated the distribution of various elements in cardiac muscles.

Key words:Wildlife, capture myopathy, histopathology, histoenzymic study.

INTRODUCTION

Capture myopathy also known as straining disease is primarily a disease of recently captured wildlife in which the muscles are mainly affected[2]. The condition has been extensively studied abroad[3]. In the present study attempts have been made to correlate the histopathological changes with histoenzymic and X-ray microanalytical findings in affected muscles.

MATERIALS AND METHODS

Fourteen stressed and one unstressed (say control) animals of the same species which died due to ailment unassociated with capture myopathy belonging to the State Zoo, Guwahati, Assam were utilised in the present study. The history, clinical signs/symptoms, necropsy findings, histopathological observations and comparative X-ray microanalysis were taken into consideration while confirming the diagnosis of capture myopathy.

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Necropsy of all the animals was conducted and gross lesions were recorded. As muscles are primarily involved, pieces of skeletal and cardiac muscles of all the 14 affected and control animals were collected in 10% neutral buffered formalin and routinely processed for histopathological studies. Unfixed cryosections of muscles of both the affected and control animals were utilised to demonstrate alkaline phosphatase (AK pase), calcium activated myoadenosine-tri-phosphatse (MAT pase), succinate (SDH), malate (MDH) and lactate (LDH) - dehydrogenases by standard methods[2,9]. For comparative X-ray microanalysis, pieces of cardiac muscle of affected and control deer were air dried and coated with carbon for X-ray microanalysis by Energy Dispersive X-ray microanalyzer - Link (EDAX) through a scanning electron microscope (JEDL)

RESULTS AND DISCUSSION

All the 14 "affected" animals were having the history of recent capture. The details are an follows:

Table - 1 : Details of animals suffering from capture myopathy

Different species with no.	Mode of capture	Period of captivity before death
Spotted deer *(Axis axis)* - 4	Manually captured in Zoo with struggling	1-2 days
Barking deer *(Muntiacus muntiacus)* - 4	- do -	1-3 days
Mouse deer *(Tragulus meminna)* - 4	Mannually captured in Jungle and transported to Zoo	7-30 days
Rhinoceros *(Rhinoceros unicornis)* - 2	- do -	4-7 days

On necropsy, haemorrhage in different parts of the body, particularly on heart . was seen alongwith patchy pneumonic areas in different lobes of the lungs. Cut surfaces of cardiac and skeletal muscles exhibited variable sized foci of white streaks. Histopathology confirmed the gross lesions of haemorhage. In addition disruption, hyalinization and loss of striaitions could be observed in the muscle fibres and presence of oedema in the skeletal muscles in chronic cases. There changes supported earlier observations[1,6,7,11]

The histoenzymic localization of different enzymes in these lesions are presented in table - 2. In cardiac muscle, moderate to strong MAT pase activity was concentrated in periphery of muscle fibres, leaving the central area free. However, the enzymic activity in control cardiac muscle was diffusely distributed, showing a homogenous pattern. The MAT pase activity was stronger in affected skeletal muscle than in affected cardiac muscle, and even more so in the blood vessels. In comparison, MAT pase activity was relatively stronger in unaffected cardiac and skeletal muscle fibres than the affected ones. AKpase activity in the lesions was not appreciable. As regards the activity of dehydrogenases, SDH, MDH and LDH were found in the lesions. Both SDH and MDH activities were weaker in affected cardiac and skeletal muscle fibres. However, it was reverse in case for LDH. The dehydrogenase reaction was diffusely scattered in the lesions but granular in normal muscle fibres. Thus it was concluded that degenerating skeletal muscles had lower AT pase and SDH contents than normal and regenerating muscles and supported earlier findings[8]. Relatively stronger activity of LDH in lesion might be due to greater metabolic acidosis through anaerobic glycolytic pathway, as evidenced histochemically by Harthorn and Young[5]. It has been opined that muscular lesions in capture myopathy could be the combination of direct action of lactic acid on muscle fibres and muscular spasms (due to change in PH[11] and forced exercise resulting in further damage[1].

Table - 2 : Histoenzymic activity in cardiac and skeletal muscles

Animal species	Condition of animals	SDH	MDH	LDH	ATpase	AKpase
Spotted deer	Control	++++	+++	+++	++	-
	Affected	++/+++	+++	++++	++	-

Spotted deer	Normal	++++	+++/++++	++++	++	-
	Affected	+++/++++	+++	++++	++	-
Barking deer	Control	++++	+++	+++	+++	-
	Affected	++/+++	+++	++++	++	-
Rhinoceros	Control	++	++++	+++	++	-
	Affected	++	+++	+++	++	-
Rhinoceros	Control	++++	+++	+++	++	-
	Affected	+++	+++	+++/++++	++/+++	

Negative:-, weak: +, moderate: ++, strong: +++, intense: ++++

The EDAX study showed certain remarkable differences in the distribution of different elements in normal and affected cardiac muscles (Fig. 1, 2). The concentration of silicon, potassium and iron decreased while phosphorus, sulphur and calcium appreciably increased in affected cardiac muscle as against control. Aluminium and chloride were present in normal cardiac muscle but these elements could not be seen in affected cardiac muscle. The observations in respect of phosphorus, potassium and calcium were in agreement with the findings of Haigh[4]. The remarkable elevation of surplus concentration in affected cardiac muscle might be due to degenerative changes[10]. Disturbance in the metabolism of phosphate and sodium, respectively, might be the factors responsible for the complete absence of aluminium and chloride in the cardiac muscle of affected animals[4].

The role of selenium and vitamin E deficiency as a predisposing factor in the pathogenesis of capture myopathy has been suggested by a good number of workers [7,8,14] However, in the present EDAX study selenium was found to be absent in cardiac muscle of both the affected and control animals. This aspect needs further elucidations.

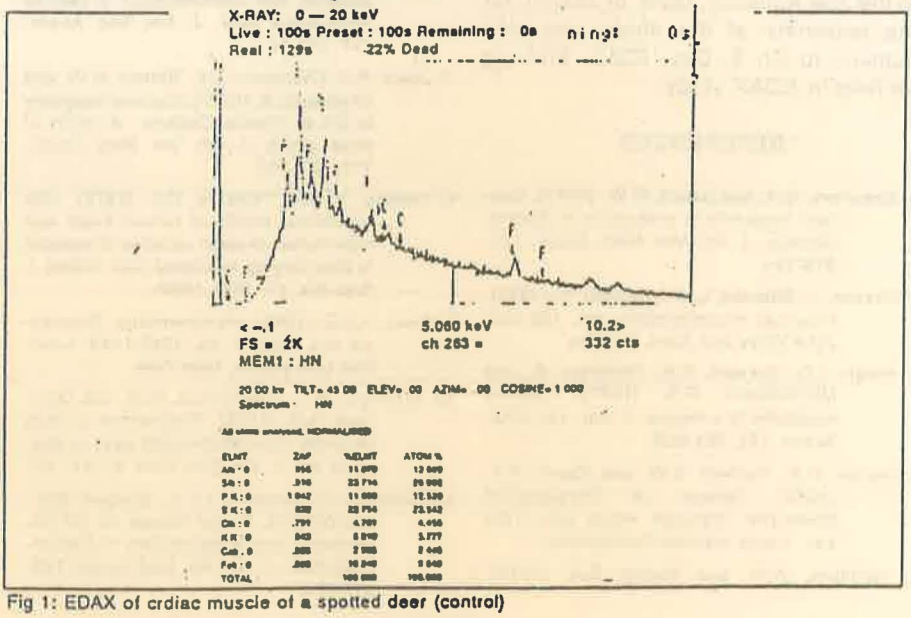

Fig 1: EDAX of crdiac muscle of a spotted deer (control)

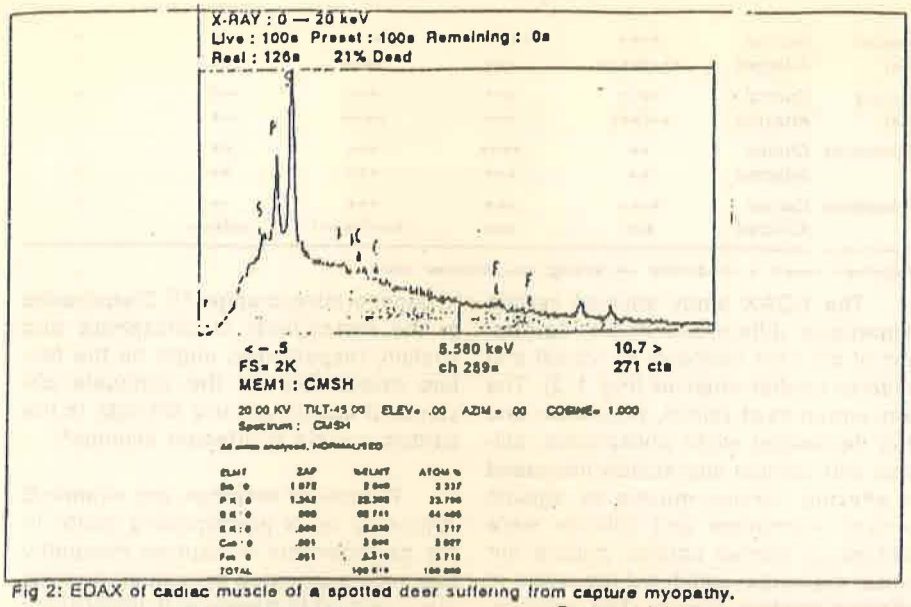

Fig 2: EDAX of cadiac muscle of a spotted deer suffering from capture myopathy.

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REFERENCES

1. Chalmers, G.A. and Barett, M.W. (1977). Capture myopathy in pronghrns in Alberta, Canada. J. Am. Vet. Med. Assoc. 171, 918-924.
2. Chayen, J.; Bitensky, L. and Butcher R. (1973). Practical Histochemistry. pp. 105-124. John Wiley and Sons, London.
3. Haigh, J.C., Stewart, R.R., Wobeser, G. and MacWilliams, P.S. (1977). Capture myopathy in a mosse. J. Am. Vet. Med. Assoc. 171, 924-926.
4. Harper, H.A., Rodwell, V.W. and Mayes, P.A. (1979). Review of Physiological Chemistry. Maruzen Asian edn, 17th edn. Lange Medical Publications.
5. Harthorn, A.M. and Young, E.A. (1974). Relationship between acid base balance and capture myopathy in Zebra *(Equus burchelli)* and an apparent therapy. Vet. Rec. 95, 337-342.
6. Kocan, A.A., Thedford, T.R., Glenn, B.L. and Shaw, M. G. (1980). Myopathy associated with immobilization in captive white-tailed deer. J. Am. Vet. Assoc. 177, 879-881.
7. Lewis, R.J., Chalmers, G.A., Berrett, M.W. and Bhatnagar, R. (1977). Capture myopathy in Elk in Alberta, Canada : A report of three cases. J. Am. Vet. Med. Assoc. 171, 937-942.
8. Narang, V. and Talesara, C.L. (1979). Histochemical profile of certain avian and mammalian skeletal muscles in relation to their varying functional load. Indian J. Exp. Bio. 17, 1087-1090.
9. Pears, A.G.E. (1980). Histochemistry. Theoretical and Applied, pp. 1342-1348. Churchill Livingstone, New York
10. Whanger, M.E., Tumbleson, M.G. and Dommert, A.R. (1969). Biochemical studies of serum from white-tailed deer in Missouri. Am. J. Vet. Clin. Path. 3, 121-125.
11. Wobeser, S., Bellamy, J.E.C., Boysen, B.G., MacWilliams, S. and Runge, W. (1976). Myopathy and myoglobinuria in a white-tailed deer. J. Am. Vet. Med. Assoc. 169, 971-974.