EXERTIONAL RHABDOMYOLYSIS IN A DEER

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The term myopathy describes a non inflammatory degeneration of skeletel muscles which is characterized clinically by muscle weakness and pathologically by hyaline degeneration of the muscle fibers (Radostits *et al.*, 1994). Jarrett *et al.* (1964) observed capture myopathy (exertional rhabdomyolysis) and provided the first published description of the Pathology of this condition in Hunter's hartebeest, Damaliscus hunteri. Subsequently, numerous reports have described a similar condition in other animals (Hofmeyer *et al.*, 1973; Chalmers and Barrett, 1982). It can occur as a sequel to paralytic myoglobinuria, unaccustomed exercise or insufficient training, chasing by other animals, running wildly for several minutes and as capture myopathy during capture of wildlife (Bartsch *et al.*, 1977). It has been observed in as short a time as six days and as long as 30 days following stressful handling (Strafuss and Kennedy, 1973). The present report describes exertional rhabdomyolysis in a Chinkara deer.

Case history

A 7 years old female deer (Chinkara) weighing about 20 kg was brought to the clinic of the Department of Animal Reproduction, University of Agriculture, Faisalabad with the history that the deer had parturated four days ago and two days following parturition, she had been chased incessantly by a male deer for about one hour. Following this the doe became recumbent and was depressed and off-feed.

Clinical examination

Clinical examination revealed that all the vital parameters were within their normal range. On vaginal examination there was no laceration in the vagina and the cervix was closed. However, the animal was depressed, off feed and was unable to stand. The thigh and back muscles were swollen, firm and painful. A diagnosis of a capture myopathy was reached (Fowler, 1986).

Treatment

After diagnosing the condition, the following treatment was given:

Drug	Dose	Route
1. Dexamethasone Sod. Phosphate (4mg/ml,Decadran MSD).	2 ml	I/m
2. Novacoc (Richter Pharma, Austria) a therapeutic cocktail tonic with anelgesic, antipyretic and energetic properties.	50 ml	I/v
3. Ringer's solution	150 ml	I/v
4. Amivicom (Selmore, Korea) amino acid, vitamin and electrolyte properties.	3 ml	I/m
5. Norfloxacin 10% (Docterjin, Selmore, Korea)	3 ml	I/m

The treatment was continued for two days. Animal showed recovery and started eating. Three days following the cessation of treatment, the animal again showed the same symptoms and was brought to the clinic. The temperature was 105 °F alongwith all above mentioned clinical symptoms. The previously given treatment was reviewed as follows.

1.	Sod. Bicarbonate	50ml/day I/v x 2 days
2.	Novalgin	2.5ml b.i.d I/m x 3 days
3.	Novacoc	50ml/day x 2 days
4.	Amivicom	3ml I/v x 5 days
5.	Norfloxacin 10%	3ml/day I/m x 5 days

The dear recovered completely as the treatment was completed.

DISCUSSION

Capture myopathy (CM) is a muscle disease associated with the stress of various types (Wallach *et al.*, 1967). The disease is characterized by degeneration and necrosis of striated and cardiac muscles. Predisposing factors include fear, anxiety, over exertion, repeated handling, failure to allow an exhausted animal to rest before transportation, prolonged transportation and constant muscle tensions (Young and Bronkhorst, 1971; Strafuss and Kennedy, 1973; Chalmers and Barrett, 1977). Harthoorn (1974) reported that an acidemia could be another factor involved in muscular myopathy. Profound muscular exertion results in a metabolic conversion from aerobic to anaerobic oxidation. Lactic acid builds up more rapidly than it can be metabolized, producing a marked

local acidosis. In severe cases, it may lead to systemic acidosis. Harthoorn (1974), Gericke and Belonje (1975), Gericke and Hofmeyer (1976) reported elevated values of serum enzymes, including glutamic oxaloacetic transaminase (SGOT), glutamic pyruvic transaminase (SGPT), lactic dehydrogenase and creatine phosphokinase (SCPK) in CM-affected animals (Chalmers and Barrett, 1977; Lewis *et al.*, 1977) which unfortunately, has not been determined in the reported case. Its determination could have supported the diagnosis of the condition. Because of the clinical and pathologic similarity of CM to various myopathies of domestic animals, the diagnosis depends, to a large degree on a composite of history and clinical pathology. Therefore, in this disease, a history of pursuit capture and physical restraint is important in the differentiation of CM from similar myopathies of domestic livestock (Hadlow, 1973; Radostits *et al.*, 1994). In the reported case there was a history of over exertion due to incessant chasing by male dear for few hours alongwith swelling and pain to thighs and back muscles. The high rise of temperature may have been due to accumulation of lactic acid which responded positively to sodium bicarbonate infusion. Clinical signs in severe cases include peracute death following cardiac failure caused by cardiac necrosis. The muscles of the back, rump and upper leg may be swollen, firm and hot. Myoglobinuria, as well as dyspnea and tachycardia may be present in acute stages. Secondary trauma of exposed surfaces of the limbs may be brought about through the animal's struggle to stand. Numerous reports on CM treatment indicated that injections of preparations containing Se, vit. E and corticosteroids alongwith intravenous infusion of sodium bicarbonate resulted in immediate and dramatic clinical improvement (Harthoorn and Young, 1974; Hofmeyer, 1974). In the present case same treatment protocol was used and the doe responded positively. Prevention should be paramount goal and for this purpose attempt should be made to minimize all contributory factors. If it is felt that severe stress has been inflicted, treat intensively for acidosis with intravenous sodium bicarbonate. Keep the animal well oxygenated. Once muscle necrosis has occurred, general nursing care, plus hot packs to the affected muscles may give some relief but the prognosis is poor.

REFERENCES

Bartsch, R.C., E. McConnell, G.D. Imes and J.M. Schmidt, 1977. A review of exertional rhabdomyolysis in wild and domestic animals. Vet. Pathol., 14: 314-319.

Chalmers, G.A. and M.W. Barrett, 1982. Capture myopathy. In: Non Infectious Diseases of Wild Life. Edited by G. L. Hoff and J. W. Davis (eds). U.S.A. pp. 84-94.

Chalmers, G.A. and M.W. Barrett, 1977. Capture myopathy in pronghorns in Alberta, Canada. J. Am. Vet. Med. Assoc., 171: 918-923.

Fowler, M.E., 1986. Zoo and Wild Animal Medicine. W.B. Saunders Co. Philadelphia, USA.

Gericke, M.D. and J.M. Hofmeyer, 1976. Aetiology and treatment of capture stress and myopathy in springbok, Antidorcas marsupialis. S. Afr. J. Sci., 72: 28.

Gericke, M.D. and P.C. Belonje, 1975. Aspects of forced exercise and the therapy thereof in sheep. J. S. Afr. Vet. Med. Assoc., 46: 353-357.

Hadlow, W.J., 1973. Myopathies of animals. In: C.M. Pearson and F.K. Mostofi (eds). The Striated Muscle. Williams & Wilkins, Baltimore, USA.

Harthoorn, A., 1974. A relationship between acid-base balance and capture myopathy. Vet. Rec., 94: 337-338.

Harthoorn, A.M. and E. Young, 1974. A relationship between acid base balance and capture myopathy in zebra, Equus burchelli and an apparent therapy. Vet. Rec. 95: 337-342.

Hofmeyer, J.M., 1974. Developments in the capture and airlift of roan antelope, Hippotragus equinus equinus, under narcosis to the Etosha National Park, Madoqua 1(8): 37-48.

Hofmeyer, J.M., G.M. Louw and J.S. du Preez, 1973. Incipient capture myopathy as revealed by blood chemistry of chased zebras. Madoqua 1(7): 45-50.

Jarrett, W.F.H., F.W. Jennings, M. Murray and A.M. Harthoorn, 1964. Muscular dystrophy in a wild Hunter's antelope. East Afr. Wild. J., 2: 158-159.

Lewis, R.J., G.A. Chalmers, M.W. Barrett and R. Bhatuagar, 1977. Capture myopathy in elk in Alberta, Canada: A report of three cases. J. Am. Vet. Med. Assoc., 171: 927-932.

Radostitis, O.M., D.C. Blood and C.C. Gay, 1994. Veterinary Medicine. Bailliere Tindall, U.K.

Strafuss, A.C and G.A. Kennedy, 1973. Degeneration myopathy in a giraffe. J. Am. Vet. Med. Assoc., 163: 551-554.

Wallach, J.D., R. Fruch and M. Leutz, 1967. The use of M99 as an immobilizing and anesthetic agent during capture, wild animals. J. Am. Vet. Med. Assoc., 151: 870-873.

Young, E. and P.J.L. Bronkhorst, 1971. Overstraining disease in game. Afr. Wild Life, 25: 51-52.