Jpn. J. Vet. Res. **43**(2): 99–103 (1995)

SUSPECTED NUTRITIONAL MYOPATHY IN TWO CAPTIVE BENNETT'S WALLABIES (*MACROPUS RUFOGRISEUS*)

Ignacia S. BRAGA III, Satoshi TANAKA, Kenji OCHIAI and Chitoshi ITAKURA

(Accepted for publication: Aug. 8, 1995)

ABSTRACT

Between January and April, 1992, two captive Bennett's wallabies (*Macropus rufogriseus*) from a wildlife park in Iwamizawa City, Hokkaido, Japan, were diagnosed as having nutritional myopathy. One wallaby, four-year-old adult male, had mild fresh lesions, confined to the skeletal muscles, with poor cell reaction. The other wallaby, 8-month-old female joey, had marked and extensive lesions with cellular reactions and regenerative muscle fibers in the locomotor muscles. Involvement of other striated muscles such as those of the tongue, diaphragm and heart was mild with minimal cellular reaction. Age susceptibility may account for the difference in the extent of severity of the lesions between these two animals.

Key Words: Bennett's wallaby (*Macropus rufogriseus*), nutritional myopathy, vitamin E

Myodegeneration, caused by either vitamin E and/ or selenium deficiency, and/ or capture or "stress," is one of the most common problems in zoo animals affecting mainly hoofed stock and marsupials. Vitamin E deficiency appears to be the main cause of the disease in marsupials, especially in captive macropods[2,7].

A pair of Bennett's wallabies (*Macropus rufogriseus*) imported from Australia (site unknown) in October, 1990, was kept in a wildlife park in Iwamizawa City (latitude 43°11′N, longitude 141°41′E), Hokkaido, Japan. They were maintained on a diet consisting of apples, carrots, cabbages and bread with no supplements.

Two captive Bennett's wallabies were submitted separately between January 27 and April 20, 1992 for necropsy to determine the cause of death. Tissue samples for histologic examination were fixed in 10% neutral buffered formalin, processed routinely and embedded in paraffin. Sections were stained with hematoxylin and eosin (H&E).

Laboratory of Comparative Pathology, Graduate School of Veterinary Medicine, Hokkaido University, Sapporo 060, Japan

Special stains were done on selected sections to demonstrate glycogen (periodic acid-Schiff, PAS), muscle and collagen fibers (Masson trichrome), and muscle striations (phosphotungstic acid-hematoxylin, PTAH).

The male wallaby of the original pair (case 1), approximately 4 years old, weighing 9.5 kg, exhibited a reduction in feed intake and swelling of the right side of the face on April 17, 1992. The facial swelling was tentatively diagnosed as actinomycosis and the animal was given terramycine® (tetracycline-HCl). He died 3 days later on April 20.

A female joey (case 2) was born to the original pair in May, 1991. When she was about 8 months old, inappetence, depression and reluctance to move were observed about 3 days prior to death (January 27, 1992)

At necropsy, case 1 had focal congestive edema of the lungs, mild dilatation of both heart ventricles and multiple gastric ulcers, but no muscular lesions were noted. Other findings included accumulation of dark colored urine in the urinary bladder. In case 2, the extensive pallor and softening of the lumbar, pelvic and hindlimb muscles were observed with pulmonary congestive edema.

Histologically, lesions seen in the affected muscles were essentially the same in both animals although variable in severity. In case 1, the muscle lesions were observed only in the cheek and lips, and *Muscularis flexor digitorum superficialis* of the distal hindlimb. The lesions were composed of scattered hyaline degeneration and necrosis of muscle fibers that were occasionally associated with mild cellular reaction. No histological lesions were observed in the heart, diaphragm, and intercostal thigh muscles.

In case 2, severe and extensive lesions were found in the locomotor muscles such as the muscles of the back, pelvis and hindlimb. The lesions were hyaline degeneration, granular and floccular degeneration, and focal or multifocal segmental necrosis of muscle fibers (Fig. 1). Degenerating muscle fibers lost their striations and were not stained with PTAH and Masson trichrome. Dystrophic calcification of necrotic muscle fibers was sometimes seen. In severely affected muscles, there were marked macrophage reaction and occasional infiltrations of lymphocytes, neutrophils and eosinophils. Regenerative muscle fibers with central nuclei, nuclear rows (Fig. 2), plump vesicular nuclei containing prominent nucleoli, and cytoplasmic basophilia were present. Histological lesions observed in the muscles of the tongue, diaphragm, lateral shoulder muscles (*Muscularis supraspinatus* and *Muscularis deltoideus*) and heart (Fig. 3) were mild, focal, and fresh, and sometimes associated with macrophage infiltration.

Nutritional myopathy, also known as nutritional myodegeneration or white muscle disease, is caused principally by deficiencies in selenium and vitamin E. The condition is commonly observed in pigs and herbivores, but is sometimes seen in herbivorous and omnivorous zoo animals[1].

The disease in case 1 was asymptomatic, therefore the lesions were mild,

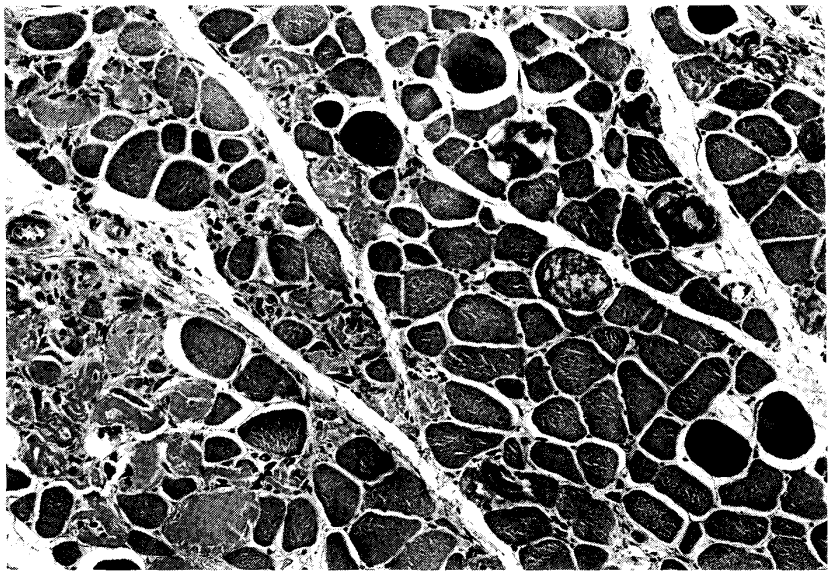

Figure 1. *Muscularis longissimus dorsi.* Hyalinization and necrosis of muscle fibers with a number of small regenrative muscle fibers. Case 2. H&E. ×250.

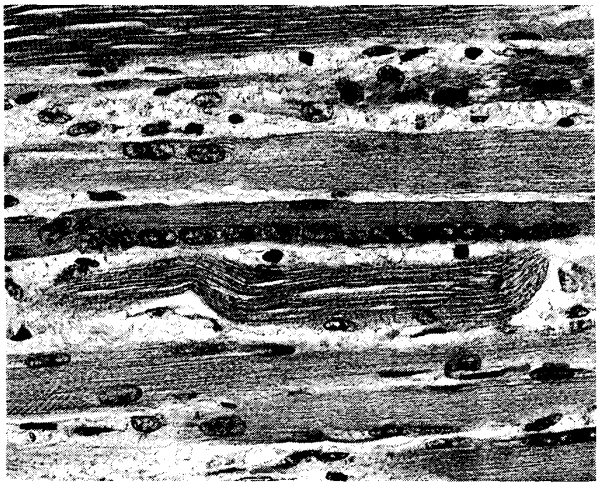

Figure 2. *Muscularis iliotibialis lateralis.* Regenerating muscle fibers with nuclear chain surrounded by degenerating muscle fibers. Case 2. H&E. ×280.

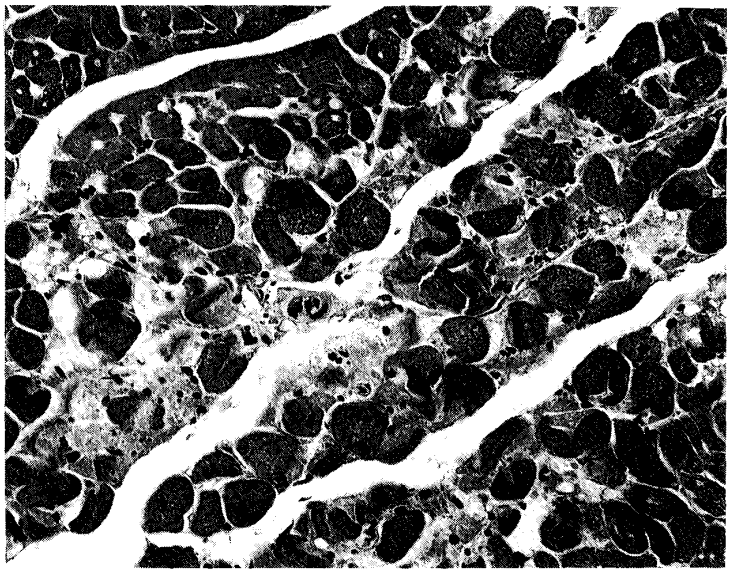

Figure 3. Cardiac muscle. Mild degenerating lesions with poor cellular reaction. Case 2. H&E. ×200.

affecting only a few muscles. In case 2, however, the lesion distribution was typical of nutritional myopathy found in marsupials, affecting primarily the muscles comprising the main bulk of the musculature of the wallaby, i.e., the lumbar, pelvic and hindlimb muscles. A previous report on the nutritional myopathy of the Bennett's wallaby[5] describes severe lesions in the masticatory muscles. In young animals, extensive lesions may be seen in the tongue[1].

Morphologic differences between the lesions of capture and nutritional myopathy are indiscernible[8], but a history of recent capture (within a month) would be highly suggestive of capture myopathy[6].

Nutritional myopathies in quokkas (*Setonix brachyurus*) are responsive to vitamin E treatment but not to selenium as in rabbits[2]. Kakulas[3] reported that the size of the enclosure ("enclosure factor") also is an important factor in the development of the condition. Therefore, animals in large enclosures do not suffer from it, indicating that prophylaxis rests mainly on management. Animals with restricted exercise, on the other hand, may require massive doses of vitamin E for prevention[4].

There were no history of recent capture, and other lesions suggesting the underlying cause of the myopathy in the present Bennett's wallabies were not seen. These facts are consistent with the view that the myopathy is due to vitamin E deficiency. The difference in the severity of the muscle lesions between the two animals may be attributed to the difference in age. Hulland[1] proposed that rapid post-natal growth is apparently a predisposing factor to the development of this condition.

REFERENCES

1) HULLAND, T. J. : Muscle and tendon. *In* : Jubb, K. V. F., Kennedy, P. C. & Palmere, N. eds., *Pathology of Domestic Animals*, 4th ed., pp. 228–235. Academic Press, San Diego, California, 1993.
2) KAKULAS, B. A. : Myopathy affecting the Rottnest quokka (*Setonix brachyurus*) reversed by α-tocopherol. *Nature* **191** : 402–403, 1961.
3) KAKULAS, B. A. : Influence of the size of enclosure on the development of myopathy in the captive Rottnest quokka. *Nature* **198** : 673–674, 1963.
4) MACKENZIE, W. F. & FLETCHER, K. : Megavitamin E responsive myopathy in Goodfellow tree kangaroos associated with confinement. *In* : Montali, R. J. & Migaki, G. eds., *the Comparative Pathology of Zoo Animals*, pp. 35–39. Smithsonian Institution Press, Washington, D. C., 1980.
5) MUNDAY, B. L. : Myonecrosis in free-living and recently-captured macropods. *J. Wildl. Dis.* **8** : 191–192, 1972.
6) MUNDAY, B. L. : Marsupial Diseases. *In* : Australian Wildlife, *the John Keep Refresher Course for Veterinarians. Proceedings* **104** : 352–353, 1988.
7) PRESIDENTE, P. J. A. : *The Management of Australian Mammals in Captivity*. pp. 55–66, 75–81, 133–143, 1882.
8) SCHMIDT, R. E. & HUBBARD, G. B. : Atlas of Zoo Animal Pathology, Vol. 1, pp. 201–210. CRC Press, Boca Raton, Florida, 1987.