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Capture Myopathy in a Red-Necked Wallaby (*Macropus rufogriseus*)

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Abstract : This case report describes capture myopathy in an 8-month-old female red-necked wallaby (*Macropus rufogriseus*) that died after a 14-day history of depression, lameness and astasia following a bathing protocol for dermatitis. In a blood test performed the day after the wallaby fell down, serum chemistry showed a normal range of LDH but elevated CK, ALT and AST. Upon histopathologic examination after the animal's death, we found degeneration and necrosis of myofibers in skeletal muscle and myocardium. According to history that the wallaby was pressed by other individuals, suspected vitamin E insufficiency in the diet, physical stress during bathing as well as results of a blood test and microscopic examination, we diagnosed this case as capture myopathy. Although capture myopathy is a common problem in marsupials, this is the first reported case in Korea and understanding this case will help to manage future cases of capture myopathy in captive settings in Korea.

Key words **:** capture myopathy, red-necked wallaby, stress, vitamin E, rhabdomyolysis.

Introduction

Capture myopathy, also called exertional rhabdomyolysis, is a metabolic muscle disease that is characterized by degenerative or necrotizing damage to the muscles from an increased myocyte production of lactic acid when oxygen is depleted and anaerobic metabolism occurs (2,6). The cause of disease is associated with extreme exertion or stress during the capture process (3,6). Clinical signs of muscle damage include ataxia, paresis and paralysis, and a cascade of complications such as lactic acidosis, renal failure, cardiac arrhythmia and death may occur (2).

Capture myopathy has been documented in many animals including ungulates, carnivores, rodents, primates, pinnipeds, birds and marsupials (6). Myodegeneration, caused by capture, stress and deficiency of vitamin E or selenium, is one of the most common problems in zoo animals and mainly affects hoofed stock and marsupials (1).

This case report describes capture myopathy in a red-necked wallaby that was raised in a zoo. The report identifies clinical signs observed during the case as well as pathologic findings.

Case

An 8-month-old intact female red-necked wallaby (*Macropus rufogriseus*) was raised in a zoo. The wallaby was fed a daily total of 800 g of food that included vegetables such as carrots, cabbage, sweet potatoes and apples as well as alfalfa hay and pellets that were not formulated specifically for marsupials. The wallaby emerged from the pouch at 2 months of age exhibiting seborrheic dermatitis, and many scales were observed on her skin (Fig 1). We cultured hairs taken from skin lesions in an enriched dermatophyte medium (InTray™ DM, BioMed Diagnostics, USA). After 4 days we observed a reddish change in the colonies' growth (Fig 2A). Under microscopic examination we found macroconidia and microconidia with separated hyphae (Fig 2B), but we did not conduct further testing to identify the genus of the fungus. To treat the dermatitis we bathed the animal with ketoconazole shampoo (NITOZOL SHPO, Korea Pharma, Korea) twice at 12 days interval. After 3 days of bathing, the wallaby showed clinical signs of ptyalism, dyspnea, depression, lameness and ataxia.

Fig 1. Severe seborrheic dermatitis with the formation of yellow-brown crusts.

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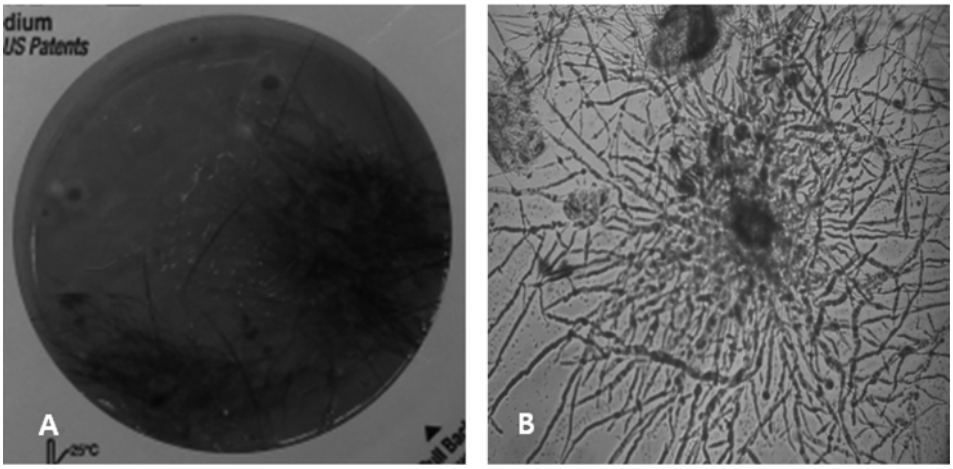

Fig 2. After 4 days of incubation. (A) Reddish change with colony growth in Intray™ DM media. (B) Grouped microconidia and spiral hyphae (× 100).

Table 1. Result of CBC and serum chemistry

Item	Result	Reference range*
WBC (× 10^3/μl)	10.74	5.728 ± 2.226
RBC (× 10^6/μl)	4.79	5.38 ± 0.74
PCV (%)	33.2	47.7 ± 7.4
CK (IU/L)	> 2000	1371 ± 1819
ALT (IU/L)	> 1000	45 ± 26
AST (IU/L)	697	88 ± 71

*ISIS March 2002, International species information system; 12101 Johnny Cake Ridge Road, Apple valley, MN 55124, USA

When forced to stand, she remained standing for only 2 to 3 hours and then fell down again.

We performed a complete blood cell count (CBC; Hemavet 950, Drew Scientific Group, USA) and a serum chemistry (Fuji Dry Chem3500s, FUJI PHOTO FILM CO., Japan). The results of the CBC and serum chemistry showed increased WBC, decreased PCV and elevated CK, ALT and AST (Table 1), other levels were within normal ranges. The wallaby did not respond to fluid therapy. She continued to show lameness and astasia, and she died after 14 days of treatment.

We conducted a postmortem examination of the wallaby. Skeletal muscles and myocardium were fixed in 10% buffered formalin, routinely processed, embedded in paraffin, sectioned at 5 μm thick and stained with hematoxylin and eosin (Fig 3).

On microscopic examination, degeneration and necrosis of myofibers were found in the femoral muscle. Muscle fibers were swollen, segmented and lost their striation. Infiltration of macrophages and fibrosis were seen in the lesion (Fig 3). A focal myocardial necrosis and mineralization were found in the heart lesion.

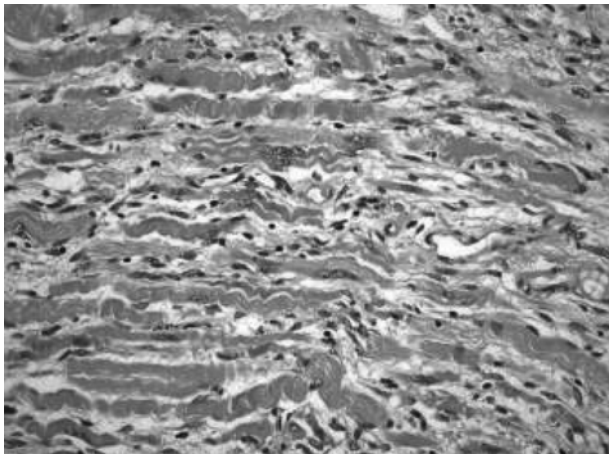

Fig 3. Femoral muscle. Degeneration and necrosis of myofibers. Infiltration of macrophage and fibrosis (H&E × 400).

Discussion

Stress, exertion and crush injury are well-documented causes of rhabdomyolysis. However, other major factors contribute to the development of capture myopathy in wildlife, including procedures that involve long periods of restraint, struggling from unnatural positioning and lengthy pursuit during capture (6). The wallaby in this case study had severe dermatitis when she emerged from the pouch, and she was physically pressed by other individuals that lived together. We suspect that severe stress from capture and restraint during bathing caused capture myopathy in this situation. In addition, we believe that myopathy was intensified because of a vitamin E insufficiency in the animal's diet. Macropods have an absolute need for dietary vitamin E. Complete marsupial feeds such as Mazuri Marsupial Pellet (PMI Nutrition International, Inc., Brentwood, MO) are supplemented. However, nonmarsupial herbivore pellets necessitate vitamin E supplementation at 200 mg to 600 mg of vitamin E per day for adult macropods (5). The wallaby was fed a diet of vegetables and nonmarsupial pellets, and no vitamin E or selenium supplements were provided.

Clinical signs of capture myopathy include pain, stiffness, inability to rise and oliguria (9). Acute capture myopathy leads to death in 3 to 4 hours because of rhabdomyolysis that leads to ventricular fibrillation. Subacute capture myopathy exhibits ataxia, torticollis and myoglobinuria caused by extensive muscle and kidney necrosis (4). Increases in serum enzyme CK, AST and LDH are usually due to an increase in muscular cell permeability and muscular damage resulting from physical stress (2,7,9). While not entirely muscle specific, elevations of these enzymes indicate muscle destruction (9). An increase of ALT could be useful in cases of capture myopathy (8). The animal in this case showed clinical signs of lameness and astasia due to muscular damage. Her serum chemistry results revealed normal LDH but elevated CK, AST and ALT. These serum chemistry results support that muscular damage occurred when the animal was captured and restrained for bathing. Histopathologically, we found degeneration and necrosis of myofibers in the femoral muscle, a focal myocardial necrosis and mineralization in the heart lesion. Skeletal muscle necrosis and myocardial necrosis are found in myopathy, and fibrosis and mineralization can occur in chronic cases (4). By history, blood serum chemistry and histopathologic change, we conclude that press of other individuals and stress during bathing led to capture myopathy, the main cause of death of this wallaby.

Treatments for capture myopathy are steroids, sodium bicarbonate, lactated Ringer's solution and selenium or vitamin E, but prognosis is usually poor (4,9). Consequently, we must make efforts to prevent this condition from occurring in the first place. Preventive measures include avoiding capture and restraint procedures during hot humid weather, having a well-trained restraint team and working quickly to minimize muscular exertion, using long tranquilizers and sedatives and ensuring animals are on a good plane of nutrition, particularly with respect to vitamin E.

Conclusion

This case report describes capture myopathy of an 8-month-old red-necked wallaby that was kept in a zoo. The wallaby died after showing clinical signs that included depression, lameness and astasia after bathing for dermatitis. Blood serum chemistry revealed elevated serum enzymes that indicated muscle damage, in histopathologic examination, degeneration and necrosis of myofibers were found. The wallaby was pressed by other individuals, had suspected vitamin E insufficiency and experienced physical stress during bathing these factors that caused capture myopathy.

Wildlife and zoo animals are at risk for capture myopathy. Because the prognosis is not good and the treatment procedure itself could be another stress factor, it is important to take measures to prevent this condition from occurring in the first place.

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붉은목왈라비에서 발생한 포획근병증

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요 약 : 본 증례는 8개월령의 암컷 붉은목왈라비에서 발생한 포획근병증으로, 피부병 치료를 위하여 약욕 후 침울, 파행, 기립불능증의 임상증상을 보이다 14일 후에 폐사하였다. 왈라비가 기립불능 증상을 보인 다음날 실시한 혈액검사에서, CK, ALT, AST의 상승을 확인하였다. 폐사 후 실시한 조직검사에서, 골격근과 심근의 근섬유가 퇴행되고, 괴사된 소견을 발견할 수 있었다. 동거 개체로부터의 압박과 비타민 E의 부족이 의심되는 급여력, 약욕시의 스트레스등과 혈액검사와 조직검사 결과 포획근병증으로 진단하였다. 포획근병증은 유대류에서 호발하는 질병으로 알려져 있으나 국내에서의 발병보고는 본 증례가 최초로, 포획근병증의 관리에 도움이 될 것이다.

주요어 : 포획근병증, 붉은목왈라비, 스트레스, 비타민 E, 횡문근융해.