



Letter to the editor

## Lymphoedema and agenesis of the popliteal lymph nodes in Golden Retriever dogs with Muscular Dystrophy (GRMD)

The absence of dystrophin caused by genetic disorders linked to the X chromosome is expressed in humans and dogs as a progressive and lethal disease characterized by weakness and muscle degeneration (1, 3). In skeletal muscle, injuries such as hypertrophy, atrophy, degeneration, necrosis and fibrosis are observed. Three Golden Retriever Muscular Dystrophy dogs (GRMD), from the kennel of the Associação de Amigos dos Portadores de Distrofia Muscular - AADM, Ribeirão Preto - SP, Brazil, naturally died, aged four, six and 18 months were submitted to necropsy in Department of Veterinary Pathology of the Faculdade de Ciências Agrárias e Veterinárias, Unesp, Jaboticabal, Brazil. The dogs presented, in the external inspection, very dry hair and muscular atrophy of the hind legs. In two animals we observed an increase in volume of bilateral hind limbs due to subcutaneous edema. In these, the necropsy showed subcutaneous edema (Fig. 1) and agenesis of the popliteal lymph nodes, indicative of lymphoedema. In the third animal (six month old) the popliteal lymph nodes were present and no edema was seen. In a study of hereditary ascendancy it was noted that all three dogs had the same paternal origin, and the breeding animal did not have these clinical features.



Figure 1: Subcutaneous edema in hind limb of a Golden Retriever dog with Muscular Dystrophy and agenesis of popliteal lymph nodes.

The primary lymphoedema (2) is characterized by excessive accumulation of fluid in the interstitial space due to defects in the development of lymphatic vessels and lymph nodes, which may affect only one individual (simple congenital lymphoedema) or a family (hereditary congenital lymphoedema). The

main changes in skeletal muscle are hyalinization, necrosis and calcification. Breeds like the Labrador and Golden Retriever are affected by this disease (2). Affected dogs do not always manifest the clinical feature, but can transmit the disorder to their descendants. So, it is necessary a thorough investigation of the genetic tree of these animals before the introduction to breeding for colonies of GRMD. Due to the injury to skeletal muscle, this disease probably aggravates the clinical symptoms and dystrophic lesions, accelerating muscle degeneration of the affected area, compromising the studies regarding to GRMD.

1. BERGMAN RL., INZANA KD., MONROE WE., SHELL LG., LIU LA., ENGVALL E., SHELTON GD. Dystrophin-deficient muscular dystrophy in a Labrador retriever. *J. Am. Anim. Hosp. Assoc.*, 2002, 38, 255-61.
2. FOSSUM TW., MILLER MW. Lymphedema: Etiopathogenesis. *J. Vet. Intern. Med.*, 1992, 6, 283-93.
3. NGUYEN F., CHEREL Y., GUIGAND L., GOUBAULT-LEROUX I., WYERS M. Muscle lesions associated with dystrophin deficiency in neonatal Golden Retriever puppies. *J. Comp. Pathol.*, 2002, 126, 100-8.

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