EQUINE ATYPICAL MYOPATHY: MORPHOLOGICAL ALTERATIONS IN OXIDATIVE MUSCLES

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SUMMARY

Between September 2000 and December 2003, a series of horses kept on pasture were referred for medical and necropsic evaluation of a sudden ataxia/myoglobinuria syndrome. Clinical examination (stiffness, muscle pain, muscle fasciculations, abnormal gait, recumbency, myoglobinuria, tachycardia, sweating) and plasma CPK, LDH and AST levels were consistent with extensive myonecrosis and, together with anamnestic data, with so-called “Equine Atypical Myopathy” (EAM), a fatal disease of unknown etiology repeatedly reported in the literature since 1939. Macroscopic and microscopic (histology, histoenzymology, ultrastructure) lesions were evaluated in thirty-two 0.5- to 7-year-old horses.

Macroscopic lesions
Necropsic examination revealed large areas of muscle necrosis, the extent and severity of which varied between cases and muscles, but which were clearly more constant and severe in respiratory and postural muscles and in the myocardium.

Microscopic lesions
Histology highlighted a multifocal and monophasic process compatible with Zenker degeneration/necrosis that mostly and segmentally affected type-1 fibres. Histochemical evaluation revealed a weak and disorganized pattern of NADH tetrazolium reductase staining, the absence of calcium salts precipitates and a dramatic accumulation of lipid droplets. Ultrastructural examination often revealed fibres of which the sole modifications were altered mitochondria and sarcoplasmic lipidosis.

Discussion
Taken together, the data suggest that a primary alteration of mitochondria should be considered, although secondary mitochondrial abnormalities have yet to be ruled out. The morphologic features gathered here reveal that EAM shares most of the characteristics of toxic myopathies.