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CORRESPONDING AUTHOR

Serena Ceriotti

serena.ceriotti@unimi.it

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Pulmonary vascular remodeling occurs in severe equine asthma.

S. Ceriotti^{a*}, M. Bullone^b, A. Vargas^b, M. Leclère^b, F. Ferrucci^a, J. Lavoie^b.

^aDepartment of Health, Animal Science and Food Safety, faculty of Veterinary Medicine, Università degli Studi di Milano, via Celoria 10, 20133 Milano, Italy

^bDepartment of Clinical Sciences, faculty of Veterinary Medicine, Université de Montréal, 3200 Rue Sicotte, Saint-Hyacinthe, Québec, Canada, J2S 6C7

Abstract

Severe equine asthma (heaves) is a chronic genetic-environmental condition characterized by airway neutrophilic inflammation leading to airway obstruction, lung tissue remodeling and hypoxemia (Bullone, 2015). Hypoxemia triggers pulmonary vasoconstriction, resulting in higher vascular resistance and development of pulmonary hypertension (PH). PH-associated cardiovascular complications could be present in the end-stage disease, as it happens in human type 3 PH (Tsangaris, 2012). Pulmonary vascular remodeling is involved in the pathogenesis of type 3 PH (Singh, 2016) and may represent a therapeutic target for preventing PH onset, both in human and in horses. In this study, we investigated the presence of pulmonary artery remodeling in severe equine asthma, as, to the best of our knowledge, this has not been yet studied. Lung biopsy specimens were collected via thoracoscopy from 6 asthmatic and 5 age-matched control horses. Histomorphometric assessment was performed on Movat-Russell stained histological sections, evaluating pulmonary artery wall area, intimal area, medial area and their correlations with the internal elastic lamina length (IEL) (Fernie, 1985). The total amount of smooth muscle within the artery wall and the density of proliferating smooth muscle cells were similarly evaluated using immunostaining for α -smooth muscle actin (α -SMA) and proliferating cell-associated nuclear antigen (PCNA). Increased pulmonary wall area, increased amount of smooth muscle and loss of correlation between intimal area and IEL were present in asthmatic horses, when compared to controls. In conclusion, pulmonary artery remodeling due to smooth muscle hypertrophy and intimal muscularization may be induced by persistence of hypoxic vasoconstriction stimulus and release of cytokines from inflammatory cells infiltrating the annexed airways (Barbera, 2003).

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