IJAE
Vol. 120, n. 1 (Supplement): 20, 2015
ITALIAN JOURNAL OF ANATOMY AND EMBRYOLOGY

Atherosclerosis and beneficial effects of parnaparin

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In recent years atherosclerosis has become a serious health challenge given its causal association with cardiovascular disease and stroke, which predictably leads to most of the morbidity and mortality worldwide [1]. The mechanism by which in this pathology there is an extensive proliferation of vascular smooth muscle cells (VSMCs) and their migration to the intima remain poorly understood. Recently, low molecular weight heparin (LMWH) has been shown to inhibit the proliferation of VSMCs and, in particular, a kind of these heparins, parnaparin (PNP), acts on the development and clinical course of atherosclerosis in animal models [2].

We hypothesized that PNP might modulate VSMCs migration and thereby decrease vascular remodelling in atherosclerosis. Vascular remodelling was evaluated in aortas of an animal model of atherosclerosis, (ApoE)-deficient mice (ApoE -/-) with normal or western diet. We treated animals with PNP to evaluate its protective effect. Thereafter, we evaluated morphological changes and the expression of proteins linked to atherogenesis and atheroprogression in particular an enzyme involved in oxidative stress, iNOS, some inflammatory mediators, such as TNF-α, IL-1 and IL-6, and markers of VSMCs alterations (PAI-1 and TSP1).

Our results demonstrated that PNP downregulates VSMCs proliferation and migration, mediated by PAI-1 and TSP1, and reduces inflammation and oxidative stress in vessels.

These data suggested that LMWH, in particular PNP, could be a theoretically practical tool in the prevention of atherosclerotic vascular modification.

References

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Keywords
Atherosclerosis; low molecular weight heparin; parnaparin; intimal hyperplasia