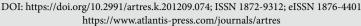


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Conference Abstract

P.65 Increased Biomarkers of Endothelial Dysfunction and Thrombotic Microenvironment in Patients with Autoimmune Rheumatic Disorders Free from Cardiovascular Comorbidities

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ABSTRACT

Purpose/Background/Objectives: Cardiovascular risk is increased in patients with autoimmune rheumatic disorders [1]. Endothelial and platelet MVs (EMVs, PMVs) are small vesicles $(0.1-1\,\mu\text{m})$ released from plasma membrane and represent novel markers of endothelial dysfunction and thrombosis. Their levels increase substantially in patients with cardiovascular diseases [2,3]. We tested whether EMVs and PMVs are increased in patients with autoimmune rheumatic disorders in the absence of cardiovascular comorbidities.

Methods: Consecutive patients with rheumatoid arthritis or systemic lupus erythematosus were studied, provided they were free from cardiovascular comorbidities (hypertension, diabetes, heart disease, history of cardiovascular or cerebrovascular events). We additionally used (a) a control group consisting of healthy volunteers and (b) a reference group including patients with stable coronary artery disease (CAD). MVs were measured by a standardized flow cytometry protocol [2,3].

Results: We studied 74 participants: 17 patients with autoimmune rheumatic diseases; 34 healthy volunteers, and 23 stable CAD patients. Patients with rheumatic diseases presented increased levels of both EMVs (283.3 \pm 195.0/ μ L vs 168.5 \pm 54.8/ μ L, p = 0.029) and PMVs (374.0 \pm 275.3/ μ L vs 225.7 \pm 101.1/ μ L, p = 0.046) compared to controls. In addition, they presented similar levels of EMVs compared to CAD patients (283.3 \pm 195.0/ μ L vs 297.0 \pm 211.8/ μ L, p = 0.846), whereas PMVs were substantially elevated in the latter (374.0 \pm 275.3/ μ L vs 1034.8 \pm 374.0/ μ L, p = 0.029).

Conclusions: Endothelial dysfunction and thrombotic predisposition, shown by increased levels of EMVs and PMVs, respectively, may be evidenced in patients with autoimmune rheumatic diseases, even in the absence of cardiovascular comorbidities and before the establishment of clinically evident cardiovascular complications. In these patients, levels of EMVs appear to be comparable with those of stable CAD patients.

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