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ANTIPHOSPHOLIPID ANTIBODIES AND EX-VIVO ENDOTHELIAL PERTURBATION

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Abstract

Introduction: Antiphospholipid (aPL)-mediated endothelial cell perturbation plays a role in antiphospholipid syndrome (APS)-associated vasculopathy. It's widely accepted that aPL antibodies activate endothelium both in vitro and in experimental animal models by inducing a pro-inflammatory/-coagulant phenotype. In spite of such large evidence, few contrasting studies raised the issue about the possibility to detect a comparable endothelial perturbation ex-vivo in APS patients. The aim of the study was to evaluate several ex-vivo indirect parameters of endothelial perturbation in APS patients.

Methods: We investigated plasma levels of soluble adhesion molecules (sICAM-1, s-VCAM-1, s-E-selectin), soluble thrombomodulin (sTM), von Willebrand factor (vWF) and tissue plasminogen activator (t-PA) by solid-phase assays, in 66 APS patients and 99 age and sex matched healthy subjects. In addition, circulating endothelial cells identified by flow cytometry and brachial artery flow mediated vasodilation were evaluated in 38 APS patients (30 primary APS and 8 SLE-associated APS) and in 47 healthy controls.

Results: We found that plasma levels of circulating TM, t-PA and VCAM-1 did not differ from controls, while a significant increase in sICAM-1 ($P < 0.001$), sE-selectin ($P < 0.001$) and vWF titres ($P < 0.05$) was found. Also circulating mature endothelial cells - with no sign of activation or undergoing apoptosis - were significantly higher in patients than in controls, both as percentage of CD146-positive cells and as absolute number ($P < 0.05$). Furthermore mean brachial artery flow mediated vasodilation responses were significantly impaired compared to healthy subjects ($P < 0.05$).

Conclusions: As a whole these findings do suggest that aPL antibodies per se are able to support a full-blown endothelial perturbation in vivo and support the two-hit hypothesis as suggested by experimental animal models of aPL-induced thrombosis.

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