Endothelial Dysfunction Caused by Circulating Microparticles from Patients with Metabolic Syndrome

Submitted by Emmanuel Lemoine on Wed, 12/11/2013 - 17:07

Titre	Endothelial Dysfunction Caused by Circulating Microparticles from Patients with Metabolic Syndrome
Type de publication	Article de revue
Auteur	Agouni, Abdelali [1], Lagrue-Lak-Hal, Anne-Hélène [2], Ducluzeau-Fieloux, Pierre-Henri [3], Mostefai, Hadj Ahmed [4], Draunet-Busson, Catherine [5], Lefthériotis, Georges [6], Heymes, Christophe [7], Martinez, Maria Carmen [8], Andriantsitohaina, Ramaroson [9]
Editeur	American Society for Investigative Pathology
Type	Article scientifique dans une revue à comité de lecture
Année	2008
Date	2008/10
Numéro	4
Pagination	1210 - 1219
Volume	173
Titre de la revue	The American Journal of Pathology
ISSN	0002-9440

Résumé en anglais	Microparticles are membrane vesicles that are released during cell activation and apoptosis. Elevated levels of microparticles occur in many cardiovascular diseases; therefore, we characterized circulating microparticles from both metabolic syndrome (MS) patients and healthy patients. We evaluated microparticle effects on endothelial function; however, links between circulating microparticles and endothelial dysfunction have not yet been demonstrated. Circulating microparticles and their cellular origins were examined by flow cytometry of blood samples from patients and healthy subjects. Microparticles were used either to treat human endothelial cells in vitro or to assess endothelium function in mice after intravenous injection. MS patients had increased circulating levels of microparticles compared with healthy patients, including microparticles from platelet, endothelial, erythrocyte, and procoagulant origins. In vitro treatment of endothelial cells with microparticles from MS patients reduced both nitric oxide (NO) and superoxide anion production, resulting in protein tyrosine nitration. These effects were associated with enhanced phosphorylation of endothelial NO synthase at the site of inhibition. The reduction of O2− was linked to both reduced expression of p47phox of NADPH oxidase and overexpression of extracellular superoxide dismutase. The decrease in NO production was triggered by nonplatelet-derived microparticles. In vivo injection of MS microparticles into mice impaired endothelium-dependent relaxation and decreased endothelial NO synthase expression. These data provide evidence that circulating microparticles from MS patients influence endothelial dysfunction.

URL de la notice	http://okina.univ-angers.fr/publications/ua218 [10]

DOI	10.2353/ajpath.2008.080228 [11]
Lien vers le document	http://dx.doi.org/10.2353/ajpath.2008.080228 [11]

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