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The mechanisms of endothelial microparticles impair endothelial function in heart valve disease and congenital heart diseases

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Abstract

Endothelial microparticles (EMPs) were subcellular fragments of the endothelial cell lipid bilayer and shed from endothelial cells by a variety of stimuli to activate endothelial cells. The increase of plasma EMPs were suggested as a new biomarker of endothelial dysfunction and play important roles in inflammation, coagulation, vascular dysfunction and angiogenesis. However, whether EMPs increase in heart valve disease and congenital heart diseases to impair endothelial function as well as it's mechanisms remain unknown. Here, 81 patients with mitral valve disease and 43 patients with congenital heart diseases as well as 45 healthy subjects were analyzed for the levels of plasma EMPs by flow cytometry. Human mitral valve endothelial cells, human cardiac microvascular endothelial cells and C57BL/6 mice were treated with EMPs. The expression and association of proteins related to endothelial function were determined. The generation of nitric oxide (NO) and superoxide anion ($O_2^{\bullet-}$) were measured. EMPs were significantly increased in patients with mitral valve disease and congenital heart diseases compared with that in healthy subjects. In cultured endothelial cells, EMPs dramatically decreased both Akt and eNOS phosphorylation and the association of Hsp90 with eNOS and increased caveolin-1 expression. EMPs also decreased NO production, but increased $O_2^{\bullet-}$ generation. In mice heart, EMPs increased caveolin-1 expression and P38 phosphorylation, decreased eNOS phosphorylation and NO production. Our data suggested that EMPs was increased in heart valve disease and congenital heart diseases. The increase of EMPs can, in turn, impair endothelial function in heart through impairing Akt/eNOS-Hsp90 signaling pathway.

Biography

Jing-song Ou has completed his M.D and Ph.D at the age of 35 years from Sun Yat-sen University of Medical Sciences and postdoctoral training from Medical College of Wisconsin. He is a Pear River Scholar Professor, Associate Chief of Division of Cardiac Surgery, and Vice Director of Guangdong Engineering Laboratory for Diagnosis and Treatment of Vascular Diseases. He has published more than 60 papers in reputed journals and serving as an editorial board member in CHEST China and Dataset Papers in Biology. He will also service as an editorial board member in American Journal of Physiology- Endocrinology and Metabolism beginning July 1, 2013.