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Impairment of Normal Endothelial Cell Function Plays a Major Role in the Initiation and Advancement of Cardiovascular Disease

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Abstract

Endothelial dysfunction is a significant factor in the development and progression of cardiovascular disease. Microparticles (MPs) derived from endothelial cells are considered to be biological markers of endothelial injury and repair, and could potentially serve as useful predictors of clinical outcomes. Apoptotic endothelial cell-derived MPs have been shown to contribute to microvascular inflammation and worsening of endothelial integrity, whereas endothelial MPs originating from activated endothelial cells may promote vascular repair and vasodilation.

Keywords: Cardiovascular disease; Apoptotic cells; Microparticles; Endothelial dysfunction; Endothelial cells

Introduction

Endothelial dysfunction refers to the impairment of normal endothelial cell function, which can lead to various pathological conditions. The endothelium is a thin layer of cells that lines the inner surface of blood vessels and plays a crucial role in regulating vascular homeostasis by producing and releasing factors that control blood flow, inflammation, and thrombosis. Endothelial dysfunction can be caused by various factors, such as oxidative stress, inflammation, hypertension, hyperlipidemia, diabetes, smoking, and aging. The consequences of endothelial dysfunction include impaired vasodilation, increased vascular permeability, increased thrombogenicity, and chronic inflammation, which can contribute to the development of cardiovascular diseases, such as atherosclerosis, hypertension, heart failure, and stroke. Recent studies have suggested that endothelial cells release distinct types of MPs, with different phenotypes and quantities, depending on whether the cells are activated or undergoing apoptosis. This suggests that the MP pattern could provide valuable information about the nature of endothelial injury. In particular, it has been proposed that the impaired immune pattern of circulating endothelial cell-derived MPs could serve as a personalized marker of vascular remodeling or endothelial dysfunction in individuals with cardiovascular disease. Overall, the use of MPs as biomarkers in cardiovascular disease has promising potential for personalized diagnosis and therapy. However, further research is needed to fully understand the mechanisms underlying endothelial dysfunction and the role of MPs in this process, as well as to determine the clinical utility of MP analysis in predicting cardiovascular outcomes. Endothelial-derived microparticles (MPs) are a group of plasma membrane vesicles with varying sizes (ranging from 100-1000 nm) that are produced by cells from the vascular endothelium or circulating endothelial cells, either through apoptosis or activation [1]. They are generated when cell membranes are shed or blebbed, which happens in both normal physiological conditions (such as stress and micro environmental stimulation) and pathological conditions (such as thrombosis, endotoxinemia, inflammation, malignancy, ischemic/hypoxic injury, and endothelial shear stress). Endothelial-derived MPs are normally present in low concentrations in the blood plasma [2]. Recent research has suggested that endothelial-derived MPs can serve as powerful paracrine regulators, influencing the functions of target cells that affect cell differentiation, tissue growth and repair, vasculogenesis, vasodilation, inflammation, apoptosis, infection, and malignancy [3-5]. They achieve this by exchanging information between cells, transferring active molecules, microRNA, peptides, hormones, inflammatory factors, growth factors, and other signaling molecules [6,7]. It is believed that activated and apoptotic endothelial cells release phenotypically and quantitatively distinct MPs, and that the pattern of MPs can provide useful information on the nature of endothelial injury. However, the full scope of biological functions of circulating endothelial MPs is still not fully understood and requires further investigation. There is a substantial amount of evidence linking immune patterns of MPs originating from endothelial cells to the development of various diseases, such as cardiovascular and rheumatic diseases, cancer, sepsis, eclampsia, autoimmune and metabolic states [8-11]. In cardiovascular diseases, such as heart failure, increased levels of apoptotic endothelial cell-derived MPs have been frequently observed and linked to endothelial dysfunction [12]. These MPs are capable of directly causing microvascular inflammation and deteriorating endothelial integrity. Furthermore, depletion of potentially pro-angiogenic endothelial MPs originating from activated endothelial cells is considered a marker of endothelial injury and insufficient repair activity. Recent clinical studies have indicated that a high ratio of CD31+/annexin V+ MPs to CD62E+ MPs is an indicator of an impaired immune phenotype of endothelial cell-derived MPs, which can be used to identify the pattern of MPs in patients with dysmetabolic disorders like diabetes mellitus and metabolic syndrome. Therefore, levels of circulating apoptotic MPs from endothelial cells can be used as biomarkers to predict the presence of cardiorenal disease, ventricular arrhythmia, and sudden cardiac death. Because endothelial dysfunction plays a role in the development of cardiovascular diseases, MPs originating from endothelial cells are potentially useful biological markers of endothelial injury and repair, and may be helpful in predicting clinical outcomes and guiding biomarker-based therapy. Elevated levels of apoptotic endothelial cell-derived MPs have been shown to predict the development of cardiovascular diseases and clinical outcomes. However, the role of decreased levels of circulating activated endothelial cell-derived MPs is not yet clear. It is possible that

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the development of cardiovascular diseases is the result of an imbalance between apoptotic endothelial-derived MPs and activated endothelial cell-derived MPs, which may be contributed to by both disease-specific and traditional cardiovascular risk factors, leading to an impaired immune MP phenotype. Recent clinical studies have demonstrated that a high ratio of apoptotic endothelial cell-derived MPs to activated endothelial cell-derived MPs and/or endothelial progenitor cells can predict cardiac failure-related death, all-cause mortality, and the risk of recurrent hospitalization due to stable or acutely decompensated cardiac failure.

Discussion

It is currently unclear whether the impaired phenotype of circulating MPs can be used to predict cardiovascular events in patients without previously documented cardiovascular and metabolic diseases. It has been suggested that the pre-existing apoptotic phenotype of endothelial MPs could serve as an individual marker beyond other cardiovascular risk factors, such as dyslipidemia, obesity, diabetes, and hypertension. If this hypothesis is true, the immune phenotype of endothelial-cell-derived MPs could be used for risk stratification among individuals in the general population.

Conclusion

To sum up, the impaired immune pattern of circulating endothelial cell-derived MPs may be regarded as a personalized marker of vascular remodeling or endothelial dysfunction among individuals with cardiovascular disease. However, the evidence of the predictive value of this marker for patients in the general population is currently limited. Further clinical trials with greater statistical power are needed to clarify these findings and their clinical significance.

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Conflict of Interest

Author declares no conflict of interest.

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