Endothelial Progenitor Cell-Mediated Vascular Repair System in Diabetes

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Abstract

Diabetes mellitus (DM) remains a leading cause of cardiovascular (CV) mortality and morbidity worldwide. Recent studies have shown the role of endothelial progenitor cells (EPCs) in endothelial repair following artery injury related to DM. The weak functional activities, lowed in vivo endothelial repair capacity and reduced number of EPCs directly contribute in the impaired endothelial reparation in DM. In fact, EPC dysfunction may not just worse functioning of vasculature and increase CV risk, but directly contributes in CD disease development in DM. There is a large body of evidence that restoring of EPCs number and functional ability may entail reducing DM-related complications and an improvement of CV outcomes. The purpose of this editorial is summary of our knowledge regarding EPCs dysfunction in DM.

Keywords: diabetes mellitus; endothelial progenitor cells, endothelial dysfunction; reparation

Introduction

Diabetes mellitus (DM) is a powerful risk factor of early atherosclerosis, cardiovascular (CV) disease, renal disease and heart failure [1]. There is a large body of evidence regarding that the hyperglycemia, lipotoxicity and hypoxia initiate the microvascular inflammation, induce endothelial dysfunction and impair endothelium
[2, 3]. Normally, the endothelial repair is result in several multifunctional relations, which in particularly include mobbing, differentiation and proliferation of endothelial progenitor cells (EPCs) derived from bone marrow and peripheral tissues [4, 5]. EPCs may cumulatively express on their surface various specific endothelial antigens like hematopoietic stem cells (CD34, CD133, AC133) and endothelial cell markers predominantly VEGF receptor-2, while expression of “non-classical” markers (CD45, Tei2 and Flt-1) could be found also [6]. Nevertheless, two main subsets of EPCs labeled as early EPCs and late outgrowth EPCs distinguish each other by their markers’ presentation and the role in angiogenesis and vascularization [7, 8]. Low number and weak functionality (i.e., reduced ability to proliferation, differentiation, adhesion, migration, incorporation into tubular structures, and survival) of EPC known as “impaired phenotype” were found in diabetics, whereas in the patients with increased number of EPCs was determined frequently [9, 10].

 Recent studies have shown that in subjects with known DM a dysfunction EPCs associated with neither their low numbers, nor their weak functions is a marker of CV risk and DM-related complications [11]. However, it has been found that subset of circulating CD34(+) cells expressing VEGFR2 and CD133 was a phenotypically and functionally distinct population of circulating EPCs that may influence on the reparation and angiogenesis [12, 13].

 DM is characterized reduced expression of angiopoetic factors and shaped an “impaired phenotype” of EPC signature as a result in mutual molecular mechanisms affected cellular signal systems, paracrine regulations and epigenetic modification. The dysfunction of EPCs correlates well with traditional CV risk factors, DM-related angiopathy and CV events [14-16]. Moreover, EPCs dysfunction may be a useful predictive tool for evaluating the CV risk including risk of death in general population and amongst individuals with known CV and T2DM [17].

 Finally, dysfunction of EPCs, which reflects worsening endothelial repair in T2DM, accompanies with CV / DM-related outcomes and may help to identify diabetics at increased CV risk. Large clinical studies are required to more pretty accurate evaluate the role of EPCs’ dysfunction as a predictive tool in diabetics.

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**References**


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