Editorial

iMedPub Journals
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Journal of Clinical and Molecular Endocrinology
ISSN 2572-5432

2016
Vol1.No.3: e105

DOI: 10.21767/2572-5432.100e105

Impaired Endothelial Repair System in Diabetes: the Role of Endothelial Progenitor Cells

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Rec date: Oct 26, 2016; **Acc date:** Oct 28, 2016; **Pub date:** Nov 1, 2016

Citation: Berezin AE (2016) Impaired Endothelial Repair System in Diabetes: the Role of Endothelial Progenitor Cells . J Clin Mol Endocrinol 1: e105.

Abstract

Diabetes Mellitus (DM) remains a leading cause of Cardiovascular (CV) mortality and morbidity worldwide. Recent studies have revealed that reduced number and weak function of Endothelial Progenitor Cells (EPCs) directly contribute in the impaired endothelial reparation in diabetics.

Moreover, EPC dysfunction may worse functioning of vasculature and increase CV risk. There is evidence that restoring of EPCs number and functional ability may entail reducing DM-related complications and an improvement of CV outcomes. The aim of the editorial is summary of knowledge regarding EPCs dysfunction in diabetics.

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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