Journal of Stem Cells Research, Reviews & Reports

Austin Publishing Group

Open Access Full Text Article

Editorial

The Role of Endothelial Progenitor Cells in the Cardiovascular Disease Pathogenesis

Nicoleta Alexandru[1], Elisabeta Badila[2] and Adriana Georgescu[1]*

[1]Department of Pathophysiology and Pharmacology, Institute of Cellular Biology and Pathology, 'Nicolae Simionescu' of Romanian Academy, Romania

[2]Emergency Hospital Bucharest, Romania

***Corresponding author:** Adriana Georgescu, Department of Pathophysiology and Pharmacology, Institute of Cellular Biology and Pathology 'Nicolae Simionescu' of Romanian Academy 8, BP Hasdeu Street, PO Box 35-14, 050568-Bucharest, Romania, Tel: +4021 319 4518; Fax: +4021 319 4519; Email: adriana.georgescu@icbp.ro

Received: August 23, 2014; **Accepted:** August 23, 2014; **Published:** August 25, 2014

Editorial

The development of atherosclerosis and cardiovascular disease is the result of multiple intermediate processes where endothelial dysfunction and vascular inflammation play key contributing roles. Atherosclerosis is one of the most important and common cause of death and disability in the world and remains an occult but important precursor of significant cardiovascular events. Atherosclerosis as a chronic inflammatory disease of the arterial wall is closely related to subendothelial lipoprotein retention, endothelial activation and migration of immune cells to inflamed intima which result in formation of fatty streaks and subsequent atheromas [1,2].

Endothelial Progenitor Cells (EPCs) have attracted major interest as biomarkers and potential regulators for atherosclerotic vascular disease, but their involvement in the mechanisms of inflammatory processes and vascular repair remain controversial. EPCs are present in peripheral blood and can develop a functional endothelial phenotype. The number and function of circulating EPCs are altered in atherosclerosis, diabetes, chronic venous insufficiency and EPCs have been shown to promote postnatal angiogenesis and vasculogenesis [3-7]. In contrast, elevated EPC levels were seen in patients that suffered an acute myocardial infarction [8] and patients that underwent a percutaneous coronary intervention [9]. Also, in patients with coronary artery disease some studies report that the EPC number is increased [10,11]. Besides, patients with unstable angina and no evidence of cardiac necrosis exhibited increased circulating EPCs [3]. Systemic inflammation, in addition to recognized growth factors, could play a role in the peripheral mobilisation of EPCs in these patients. Likewise, it can be speculated that this may represent a protective, compensatory response.

In this context, EPC research has important clinical implications but is often impeded by methodological issues and a lack of consensus on phenotypic identification of these cells.

The number of circulating EPCs can be measured using fluorescence-activated cell-sorting analysis and standard gating techniques to detect surface marker expression [12]. Asahara et al. [13] was the first to isolate EPCs in human peripheral blood, using anti-CD34 monoclonal antibodies. Now EPCs are defined as either early and late EPCs based on their biological properties and their time of appearance in vitro culture. With the use of CD133, an antigen specifically identifying primitive stem cells, a novel means to precisely delineate mature from immature EPCs was possible [14]. Other markers used include vWF, VE-cadherin, Vascular Endothelial Growth Factor Receptor-2 (VEGFR-KDR) and binding by lectins and Acetylated Low-Density Lipoproteins (Ac-LDL) [14-16].

The EPC function can, in part, be measured by assessing their colony forming capacity in vitro [17]. Modalities to increase the number or improve the function of EPCs may be promising in the treatment of cardiovascular disease. Among these are physical exercise, administration of erythropoietin and treatment with statins [18] and irbesartan [6,19], which enhance both the number and functionality of EPCs. In addition, systemic transfusion of EPCs improves the endothelial and platelet function and reduces the atherosclerosis in the animal models [20,21]. EPCs have the ability to reduce platelet activation and to modulate their pro-inflammatory and antithrombogenic properties in hypertension associated with hypercholesterolemia. These findings revealed a new biological role of circulating EPCs in platelet function regulation, and contributed to a better understanding of their crosstalk, and the mechanisms of atherosclerosis [21].

However, much work remains to be done to clarify the role of EPCs in cardiovascular disease. Presently, there is a huge interest in the EPC assessment as advanced markers for cardiovascular disease pathogenesis evaluation and drug therapy monitoring.

References

1. Libby P, Aikawa M. Stabilization of atherosclerotic plaques: new mechanisms and clinical targets. Nat Med. 2002; 8: 1257-1262.
2. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. N Engl J Med. 2005; 352: 1685-1695.
3. George J, Goldstein E, Abashidze S, Deutsch V, Shmilovich H, Finkelstein A, et al. Circulating endothelial progenitor cells in patients with unstable angina: association with systemic inflammation. Eur Heart J. 2004; 25: 1003–1008.
4. Hill JM, Zalos G, Halcox JP, Schenke WH, Waclawiw MA, Quyyumi AA, et al. Circulating endothelial progenitor cells, vascular function, and cardiovascular risk. N Engl J Med. 2003; 348: 593-600.
5. Vasa M., Fichtlscherer S., Aicher A. Number and migratory activity of circulating endothelial progenitor cells inversely correlate with risk factors for coronary artery disease. Circ Res. 2001; 89: E1-E7.
6. Georgescu A, Alexandru N, Andrei E, Titorencu I, Dragan E, Tarziu C, et al. Circulating microparticles and endothelial progenitor cells in atherosclerosis; pharmacological effects of irbesartan. J Thromb Haemostasis. 2012; 10: 680-691.
7. Georgescu A, Alexandru N, Popov D, Amuzescu M, Andrei E, Zamfir C, et al.

Citation: Alexandru N, Badila E and Georgescu A. The Role of Endothelial Progenitor Cells in the Cardiovascular Disease Pathogenesis. J Stem Cells Res, Rev & Rep. 2014;1(2): 1006.

Chronic venous insufficiency is associated with elevated level of circulating microparticles. J Thromb Haemost. 2009; 7: 1566-1575.

8. Shintani S, Murohara T, Ikeda H, Ueno T, Honma T, Katoh A, et al. Mobilization of endothelial progenitor cells in patients with acute myocardial infarction. Circulation. 2001; 103: 2776-2779.

9. Banerjee S, Brilakis E, Zhang S, Roesle M, Lindsey J, Philips B, et al. Endothelial progenitor cell mobilization after percutaneous coronary intervention. Atherosclerosis. 2006; 189: 70-75.

10. George J, Goldstein E, Abashidze S, Deutsch V, Shmilovich H, Finkelstein A, et al. Circulating endothelial progenitor cells in patients with unstable angina: association with systemic inflammation. Eur Heart J. 2004; 25: 1003-1008.

11. Guven H, Shepherd RM, Bach RG, Capoccia BJ, Link DC. The number of endothelial progenitor cell colonies in the blood is increased in patients with angiographically significant coronary artery disease. J Am Coll Cardiol. 2006; 48: 1579-1587.

12. Andrei E, Alexandru N, Dragan E, Georgescu A. Flow cytometric analysis of circulating microparticles and endothelial progenitor cells in plasma; a research tool for atherosclerosis and therapy. Exp Clin Cardiol. 2014; 20: 1554-1563.

13. Asahara T, Murohara T, Sullivan A, Silver M, van der Zee R, Li T, et al. Isolation of putative progenitor endothelial cells for angiogenesis. Science. 1997; 275: 964-967.

14. Peichev M, Naiyer AJ, Pereira D, Zhu Z, Lane WJ, Williams M, et al. Expression of VEGFR-2 and AC133 by circulating human CD34+ cells identifies a population of functional endothelial precursors. Blood. 2000; 95: 952–958.

15. Rafii S, Lyden D. Therapeutic stem and progenitor cell transplantation for organ vascularization and regeneration. Nat Med. 2003; 9: 702-712.

16. Asahara T, Takahashi T, Masuda H, Kalka C, Chen D, Iwaguro H, et al. VEGF contributes to postnatal neovascularization by bone marrow derived endothelial progenitor cells. EMBO J. 1999; 18: 3964 – 3972.

17. Hill JM, Zalos G, Halcox JP, Schenke WH, Waclawiw MA, Quyyumi AA, et al. Circulating endothelial progenitor cells, vascular function, and cardiovascular risk. N Engl J Med. 2003; 348: 593-600.

18. Dimmeler S, Aicher A, Vasa M, Mildner-Rihm C, Adler K, Tiemann M, et al. HMG-CoA reductase inhibitors (statins) increase endothelial progenitor cells via the PI 3-kinase/Akt pathway. J Clin Invest. 2001; 108: 391-397.

19. Badila E, Daraban AM, Ghiorghe S, Georgescu A, Alexandru N, Bartos D, Tîrziu C. Rethinking cardiovascular therapy - the effect of irbesartan on circulating microparticles and endothelial progenitor cells in patients with hypertension and dyslipidemia. Farmacia. 2014; 62: 93-106.

20. Ma ZL, Mai XL, Sun JH. Inhibited atherosclerotic plaque formation by local administration of magnetically labeled endothelial progenitor cells (EPCs) in a rabbit model. Atherosclerosis. 2009; 205: 80-86.

21. Alexandru N, Popov D, Dragan E, Andrei E, Georgescu A. Circulating endothelial progenitor cell and platelet microparticle impact on platelet activation in hypertension associated with hypercholesterolemia. PloS One. 2013; 8: e52058-e52068.

J Stem Cells Res, Rev & Rep - Volume 1 Issue 2 - 2014
ISSN : 2381-9073 | www.austinpublishinggroup.com

Citation: Alexandru N, Badila E and Georgescu A. The Role of Endothelial Progenitor Cells in the Cardiovascular Disease Pathogenesis. J Stem Cells Res, Rev & Rep. 2014;1(2): 1006.