The association of low molecular heparin and galectin-3 on the cell migration and proliferation of vascular endothelial cell from mesenchymal stem cells.

Yang Ding, Xiaoqiang Li^{*}, Aimin Qian, Hong-Fei Sang, Chenglong Li

Department of Vascular Surgery, the Second Affiliated Hospital of Soochow University, Suzhou, PR China

Abstract

Objective: To explore the effect of the association of low molecular heparin and Galectin-3 on the cell migration and proliferation of vascular endothelial cell from mesenchymal stem cells.

Methods: Depending on the administration, this study is divided into four groups: low molecular weight heparin group, adding 20 μ g/ml low molecular weight heparin into the cells; Galectin-3 group, adding 5 μ g/ml Galectin-3 into the cells; combination group, adding 20 μ g/ml low molecular weight heparin and 5 μ g/ml of Galectin-3 into the cells; control group, equal volume of phosphate buffer saline buffer into the cells. Then we explored the effect of the association of low molecular heparin and Galectin-3 on the cell migration and proliferation of vascular endothelial cell from mesench matched thematched the cells.

Results: The optical density at 490 nm (OD_{490}) for LMWH, Galectin-3, combined and control groups were 0.285 ± 0.018 , 0.297 ± 0.041 , 0.351 ± 0.016 , and 0.233 ± 0.005 , respectively, and the combined group could significantly increase the cell proliferation than another group (P×0.05). Cultured for 24 h, the cell migration rate of low molecular weight heparin group and Galectine 9 group were 42.02 ± 7.62 and 45.82 ± 3.96 , respectively, whereas the cell migration rate of combined group and control group were 68.53 ± 11.22 and 34.21 ± 3.99 , respectively, suggesting that combined group had the largest cell migration (P<0.05).

Conclusion: The association of low molecular housin and Galectin-3 could significantly improve the cell migration and proliferation of vascular endothely/cell from mesenchymal stem cells.

Keywords: Low molecular heparin, Galecting, Necular endothelial cell, Cell migration, Cell proliferation.

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Introduction

With the aging of society, the incidence of outonic peripheral arterial disease (PAD) increases year by year. PAD seriously affects patient's physical health and quality of life [1-3]. Among various types of PAD, arterial occlusive disease of low extremity and diabetic foot are the most serious, which are difficult to cure [4,5]. Recently years, PAD have aroused widespread attention due to it can lead to limb ischemia, necrosis, eventually amputation, or even death in patients [6,7]. It is reported that stem cell transplantation can be applied to treat PAD [8]. Similarly, it has been proved that transplanted bone marrow mesenchymal stem cells (MSCs) with great regenerative potential may differentiate into vascular endothelial cells and smooth muscle cells, and then to repair the damaged tissue [9]. At same time, through autocrine and paracrine pathway, it synthesizes and secretes vascular growth factor to promote angiogenesis. Another study shows that bone marrow MSCs can promote cell proliferation, suppress apoptosis, and through anti-inflammatory to promote angiogenesis [10,11]. Thus, vascular endothelial cells is the key factor in stem cell transplantation. Vascular endothelial cells that constitute the blood vessel wall act as a shield for harmful stimulus to blood. It is the sole anti-thrombotic cell type in

human body [12-14]. In addition, it can produce multiple active substances to protect blood vessel [15-17]. However, clinical investigation found that some problems remain unsolved. MSCs-derived vascular endothelial cells play an important role on angiogenesis and repair of damaged vascular tissue, but so far, the migration and proliferation of MSCsderived vascular endothelial cells may mainly influence the ability of angiogenesis and vascular repair. Therefore, how to improve the migration and proliferation of MSCsderived vascular endothelial cells is the focus of clinical research for treating PAD.

Heparin is commonly used as anticoagulant drugs clinically to prevent postoperative thrombosis [17-20]. Low molecular weight heparin (about 5 kd) is generated through hydrolysis of heparin mainly. It has been widely used in clinical practice because it has many advantages, such as high efficiency, ineligible affinity to platelet and better stability. Galectin-3 belongs to glycoprotein. Depending on its glycol-domain, it specifically binds intracellular glycoproteins, cell surface molecules, glycosylated extracellular matrix proteins and membrane proteins *via* the lectin-glyco-interaction to participate in a variety of physiological and pathological