6. BERBERINE -INDUCED UPREGULATION OF CIRCULATING ENDOTHELIAL PROGENITOR CELLS IMPROVES ENDOTHELIAL FUNCTION AND ARTERIAL ELASTICITY IN HEALTHY SUBJECTS

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4. CAROTID FLOW VELOCITY DIFFERENCE AS A SURROGATE FOR ISCHEMIC STROKE

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Background: Carotid flow peak velocity is increased in carotid artery stenosis. Carotid intima-media thickness (IMT) have been associated with an increased risk of ischemic stroke. We performed this study to evaluate the relationship between carotid flow velocity and carotid IMT in ischemic stroke without significant carotid stenosis.

Methods: A total of 559 patients with acute ischemic stroke were enrolled. We evaluated the association of carotid IMT and carotid flow velocity difference from peak systolic to end diastolic velocity with age, sex, potential vascular risk factors, and cardiac function by echocardiography.

Results: Age and common carotid IMT was significantly associated with carotid flow velocity difference (r = -0.106, p < 0.014). We did not find a significant relationship between carotid flow velocity difference and gender or traditional cardiovascular risk factors such as hypertension, diabetes, smoking and dyslipidemia. Left ventricle end diastolic dimension and diastolic parameter e/e' from echocardiography was also related with carotid blood flow velocity (r = 0.107, p = 0.015, r = 0.108, p = 0.016). Linear regression analysis demonstrate that carotid flow velocity difference was independently associated with carotid IMT (β = -0.094, p = 0.040) and e/e'β = 0.116, p = 0.011).

Conclusion: The present results indicated that carotid flow velocity difference was independently associated with carotid IMT and left ventricle diastolic function. We further suggest that carotid flow velocity difference is another surrogate for ischemic stroke in the spite of absence of significant carotid stenosis.

5. THE EFFECT OF CILOSTAZOL TO ARTERIAL STIFFNESS IN THE HYPERTENSIVE PATIENT

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Background: Cilostazol is a potent antiplatelet agent that selectively inhibits phosphodiesterase III, and also it has beneficial effect to peripheral artery disease. Clinical results of cilostazol study demonstrate that cilostazol flow velocity difference from peak systolic to end diastolic velocity with age, sex, potential vascular risk factors, and cardiac function by echocardiography did not change. C2 increased significantly (6.21 ± 2.80 ml per mm Hg/100 vs 4.06 ± 2.67ml per mm Hg/100, P < 0.01) and C1 remained unchanged (10.79 ± 3.27 ml per Hg/10 vs 10.06 ± 2.08 ml per mm Hg/10, P = 0.05). The increment of carotid PWV was statistically significant (r = 0.52, P < 0.05) and the increment of PWV was statistically significant (r = 0.68, P < 0.01). Multiple regression analysis indicated that only the EPC number was related with the determination of PWV (R² = 0.27, P = 0.01) and ∆C2 (R² = 0.46, P < 0.01).

Conclusion: BR-induced upregulation of the number and function of circulating EPCs contributes to improvement of endothelial function and small artery elasticity in healthy subjects.

7. KOREAN PANAX RED GINSENG IMPROVES ENDOTHELIAL DYSFUNCTION AND ARTERIAL STIFFNESS IN PATIENTS WITH CORONARY ARTERY DISEASE PROBABLY BY DECREASING RHO-ASSOCIATED KINASE ACTIVITY OF PERIPHERAL BLOOD MONONUCLEAR CELLS

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Background: Korean Panax Red Ginseng(KPRG) is known to enhance endothelium-dependent vasorelaxation by activating eNOS. Activation of Rho-associated kinase (ROCK) is implicated in endothelial dysfunction in part through destabilization of eNOS mRNA. This study, a randomized, double-blind, placebo-controlled, crossover trial, is aimed to analyze the effect of KPRG on endothelial function, arterial stiffness, and ROY activity of peripheral blood mononuclear cells (PBMC) in patients with coronary artery disease (CAD).

Methods: Patients (n=10, 12 males, 62.5 yrs) with stable angina (multi-vessel disease: n=10) were treated alternatively with either KPRG (3g/d) or placebo (starch, 3g/d) for 10 weeks (washout period for 4 wks). Measurement of flow-mediated dilation (FMD) and nitroglycerine mediated dilation (NMD) on the brachial artery and collection of peripheral venous blood for the biochemical study were performed at day 0 (baseline) and after completion (endpoint) of each treatment. Activity of ROCK of PBMC was assessed by analyzing phospho-Thr853 in the myosin binding subunit (MBS) of myosine light chain phoshapatase with use of Western blot, and was expressed as the % ratio of phospho-Thr853-MBS/total MBS at endpoint versus baseline. Patients were classified into low (-5%) FMD group (n = 11) and high (-5%) FMD group (n = 9).

Results: Treatment with KPRG significantly increased FMD in low FMD group (3.49 ± 0.35% vs 5.50 ± 0.72%, p = 0.013), but not in high FMD group. Brachial ankle pulse wave velocity (ba-PWV), measured by VP2000, tends to be decreased by KPRG (1770 ± 185 vs. 1499 ± 95, P = 0.05). There was not significant changes in nitroglycerine mediated dilation after each drug treatment in both patient groups. Treatment with KPRG significantly decreased ROCK activity in PBMC of CAD patients (n = 6) by 23.9 ± 3.01% as compared with the baseline (p < 0.05). Placebo treatment did not change any of these indices significantly.

Conclusion: In conclusion, KPRG improves endothelial dysfunction and peripheral arterial stiffness in patients with CAD probably by decreasing ROCK activity of PBMC.