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Korean Panax ginseng decreases Rho-associated kinase activity of peripheral blood mononuclear cells and ameliorates endothelial dysfunction in patients with coronary artery disease

Ick-Mo Chung[1], Joo Weon Lim[2], Hae-Yun Chung[3], Jeong Yeon Seo[3], Hyeyoung Kim[3]

[1]*Division of Cardiology, Ewha Womans University School of Medicine;*
[2]*Institute of Gastroenterology, College of Medicine, Yonsei University;*
[3]*Food and Nutrition, College of Human Ecology, Yonsei University*

Korean Panax Ginseng (KPG) is known to increase nitric oxide (NO) bioavailability in vitro study. Given the role of oxidative stress in pathogenesis of atherosclerosis, KPG may ameliorate oxidative stress in atherosclerosis, thereby improving endothelial function. However the effect of KPG on the pathogenesis of atherosclerosis is not known yet. Rho-associated kinase (ROCK) that can be activated by GTP-binding RhoA may decrease NO bioavailability through destabilization of eNOS mRNA. This study, a randomized, double-blind, placebo-controlled, crossover trial, is aimed to analyze the effect of KPG on endothelial function and ROCK activity of peripheral blood mononuclear cells (PBMC) in patients with coronary artery disease (CAD). Patients (n=20, 12 males, 62.5±2.2 yrs) with CAD diagnosed by coronary angiography were treated alternatively with either KPG (3g/d) or placebo (3g/d) for 10 wks (washout period for 4 wks). Measurement of flow mediated dilation (FMD) at the brachial artery, as an index of endothelial function, and collection of blood for biochemical analysis were performed before and after treatment with each drug. Activity of ROCK was assayed in PBMC by analyzing phospho-Thr853 in the myosin binding subunit (MBS) of myosine light chain (MLC) phosphatase with use of Western blot, and was expressed as the ratio (%) of phospho-Thr853-MBS/MBS at endpoint compared with baseline. Patients were classified into low (<5%) FMD group (n=11) and high (≥5%) FMD group (n=9). Treatment with KPG 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 (7.12±0.61 vs. 5.44±0.70, p>0.05). Treatment with placebo did not change FMD significantly in both patients groups. There was no significant changes in nitroglycerine mediated dilation after each drug treatment in both patient groups. Treatment with KPG, but not placebo, significantly decreased ROCK activity in PBMC of CAD patients (n=6) by 23.9±3.01 as compared with the baseline (p<0.05). In conclusions, KPG improves endothelial dysfunction in patients with CAD probably by decreasing ROCK activity of PBMC. Further study is needed to estimate the therapeutic effect of KPG on CAD.