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Pharmacogenetic Study of Lipid-Lowering Therapy with Rosuvastatin in Coronary Artery Disease Patients

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Background & Hypothesis:

Polymorphic genes involved in the regulation of lipid metabolism may be responsible for interindividual differences in efficiency of hypolipidaemic therapy. In the present prospective and randomised study, we investigated the efficacy of rosuvastatin (10 mg/day) in lipid-lowering therapy in 62 patients with coronary artery disease (CAD) possessing different genotypes of lipoprotein lipase (LPL) gene.

Methods:

One-year therapy with rosuvastatin was carried out under the control of lipid metabolism parameters including total cholesterol, LDL-C, HDL-C, cholesterol unlinked to HDL, triglycerides, atherogenic index at the baseline and 4th, 8th, 24th and 48th week. The +495T >G polymorphism (*rs320*) of the LPL gene was genotyped in the patients through a real-time PCR TaqMan assay.

Results:

Rosuvastatin exerted a significant hypolipidaemic effect against all investigated lipid metabolism parameters by 24 weeks of therapy. Changes in parameters of lipid metabolism upon rosuvastatin treatment differed in CAD patients with genotype +495GG compared with the rest LPL genotypes. A genotype +495GG had a greater reduction in total cholesterol at week 8, LDL-C, cholesterol unlinked to HDL and atherogenic index at the 48 week of rosuvastatin therapy ($P < 0.01$) in comparison with the +495TT and TG genotypes.

Discussion & Conclusion:

The study findings suggest that the pronounced hypolipidaemic effect of rosuvastatin in homozygotes +495GG of the LPL gene could be associated with modulation of lipoprotein lipase activity, as it was previously reported for other statins. The study was supported by the Russian Research Foundation (No.-15-5-10010).