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Relationship between the G75A polymorphism in the apolipoprotein A1 (*ApoA1*) gene and the lipid regulatory effects of pravastatin in patients with hyperlipidemia

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ABSTRACT. In this study, we investigated the relationship between the G75A polymorphism in the apolipoprotein A1 (*ApoA1*) gene and the lipid regulatory effect of pravastatin in patients with hyperlipidemia. A total of 179 patients were divided into two groups: the pravastatin (N = 97) and policosanol (N = 82) treatment groups. The total cholesterol (TC), triglyceride, low-density lipoprotein (LDL-c), high-density lipoprotein, ApoA, and ApoB concentrations in the serum were measured using an automatic biochemical analyzer before and after treatment for 12 weeks. The genotypes of the *ApoA1* G75A SNP were detected by polymerase chain reaction-restriction fragment length polymorphism, and were subsequently statistically analyzed. Pravastatin treatment induced a significant decrease in the TC, LDL-c, and ApoB levels in patients expressing the *ApoA1* AA+GA genotype ($P < 0.05$), and not in those expressing the GG genotype ($P > 0.05$). However, policosanol

treatment induced a non-significant decrease in the serum TC levels ($P > 0.05$) and a significant decrease in the ApoB levels ($P < 0.05$), and did not induce a decrease in the LDL-c ($P > 0.05$) levels in patients with the AA+GA genotype. Policosanol also induced a significant decrease in the TC and LDL-c levels in patients with the GG genotype ($P < 0.05$). The various genotypes of the *ApoA1* G75A SNP influence the efficacy of lipid regulation by pravastatin and policosanol in patients with hyperlipidemia.

Key words: Hyperlipidemia; Apolipoprotein A1; Total cholesterol; Pravastatin; Policosanol; Gene polymorphism

