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Association of common genetic polymorphisms with plasma lipid and coronary artery disease in West Bengal population

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Both apolipoprotein B (APOB) and low-density lipoprotein receptor (LDL-R) play crucial role in LDL uptake by cells. The association of *apob Msp*I polymorphism and *ldl-r Ava*II polymorphism with coronary artery disease (CAD) has already been reported in other populations. Genetic variations in these two gene locus is associated with CAD in West Bengal population was investigated here. Blood samples were collected from angiographically proven 254 CAD patients and age matched 246 healthy people (controls) from different districts of West Bengal, India. Serum lipids including total cholesterol (TC), triglycerides (TG), high density lipid-cholesterol (HDL-C), low density lipid-cholesterol (LDL-C) and very low density lipid-cholesterol (VLDL-C) were evaluated in all the subjects. Genotyping was performed by PCR-RFLP combined with gel electrophoresis. The lipid-profile analysis revealed that TC, TG, LDL-C and VLDL-C were significantly high ($p < 0.001$) in patients than controls. Genotyping study showed that homozygous A^+A^+ genotype was significantly more prevalent (22% vs 10%, $p = 0.0011$) among patient group in this population than control. This genotype was also associated with higher LDL-C and TC levels. But there was no significant association of genotypes with serum lipid concentration was evident in APOB gene. The A^+A^+ genotype could be a genetic marker for CAD.

Keywords: APOB, LDL-R, CAD, PCR-RFLP, West Bengal

Introduction

Coronary artery disease (CAD) is the most common form of cardiovascular diseases (CVD)[1]. World Health Organisation (WHO) has already declared that CVD is the major reason of death in the planet. An estimated 17.3 million people died from CVDs in 2008, representing 30% of all global deaths and over 80% of the world's deaths from CVDs occur in low- and middle - income countries like India. Moreover, among these deaths, an estimated 7.3 million were due to CAD. So, we need to devise the highly potential diagnostic and therapeutic techniques which will efficiently help to reduce the number of death worldwide. In order to do that we first need to understand the pathology of the disease. CAD occurs when coronary arteries are narrowed which actually reduces or blocks the blood supply through the narrow arteries to the heart. The main cause of narrowing of the coronary arteries is the formation of atherosclerotic plaque[3]. There are several mechanisms in the body that leads to the plaque formation inside arteries. As they help to develop atherosclerotic plaques, these mechanisms are considered as risk factors for CAD. Retention of lipoproteins especially low-density lipoprotein (LDL) in the blood is one of the risk factors for developing CAD[3,4]. Among other reasons, improper clearance of LDL-C from blood is reported as one of the major causes of LDL retention[5,6]. Moreover, it has been also documented that the genetic polymorphisms at candidate gene locus actually modulate the LDL retention in blood as well as the formation of atherosclerotic plaque[7,8]. In this study we have focused on the genetic polymorphism at gene locus of two very well known proteins responsible for cellular uptake of LDL. They are low density lipoprotein receptor (LDL-R) and apolipoprotein B (APOB).

LDL-R is a trans-membrane protein that modulates plasma levels of LDL-C by regulating the uptake of LDL-C particles by cells of liver and delivers

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