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Association Analysis of Putative Loci with Coronary Artery Disease

Authors : Asma Naseer Cheema, Attya Bhatti, Jabar Ali, John Peter

Abstract : Background: High cholesterol levels, endothelial dysfunction, inefficient coagulation cascade and hyper inflammatory response all are the basis of coronary artery disease (CAD). Several studies are carried out to see the genetic influence of these factors on disease outcome. Objective: The objective of our study was to see the association of 10 putative loci with coronary artery disease in our population. Materials & Methods: We screened our population for 10 putative loci of CAD showing significant association ($p < 5x10-8$) with candidate genes (regulating the cholesterol metabolism, endothelial function, coagulation cascade and inflammatory response of body). Hardy-Weinberg equilibrium and linkage disequilibrium in cases and controls s were estimated separately. Approximately 5-10 ng of dried DNA in 384 well plate format was used to genotype each sample on the Sequenom iPLEX assay at University of Pittsburgh Genomics and Proteomics Core Laboratories. It was built on single-base primer extension with the MALDI-TOF MS detection possessing high sensitivity and specificity. The SNPs were genotyped through Taqman assay. Hardy Weinberg test was applied. The 10 SNPs were selected as genetic markers for this study (rs579459, rs1561198, rs2954029, rs1122608, rs17114036, rs9515203, rs10947789, rs7173743, rs2895811, rs2075650). Results: Mean age of the patient was 52 ± 11 years. Blood pressure and positive family history was found a significant risk factor for CAD. None of the selected SNPs showed significant association with coronary artery disease in our population ($p>0.05$). Conclusion: rs579459, rs1561198, rs2954029, rs1122608, rs17114036, rs9515203, rs10947789, rs7173743, rs2895811, rs2075650 are not significant genetic markers for CAD in our population.

Keywords : CAD, genetic markers, loci, risk factors
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