GENE MS POLYMORPHISM A2756G AND CORONARY ARTERY DISEASE RISK IN PATIENTS FROM PERNAMBUCO

Will B. Pita[1], Karina A. Silva[1]; Érica R.F. Siqueira, Vânia D´Almeida[4], Maria S.M. Cavalcanti[2], Cláudio A. Sampaio[3], Maria T.C. Muniz[1,2]

1.Centro de Oncohematologia Pediátrica/HUOC/UPE 2. Departamento de Ciências Fisiológicas-ICB/UPE, 3. Departamento de Bioquímica – UNIFESP 4.Departamento de Psicobiologia – UNIFESP

The Coronary Artery Disease (CAD) is the most representative cause of death in western. It has a multifatorial etiology presenting genetic, environmental, atherogenic and thrombogenic causes. Homocystein is considered an independent risk factor to atherosclerosis. Hyperhomocysteinemia may be caused by inherited genetic disorders, such as Methionine Synthase (MS) homozygotic fault, besides B12 vitamin metabolic disturbs. MS is responsible to remethylation of homocystein to methionine. An adenine to guanine substitution in the 2756 position of MS gene determines the switching of the aspartic acid to the glicine, compromising the enzymatic activity. The aim of this study was verify the association of MS polymorphism with the development of CAD. The genotyping was performed by PCR-RFLP in 46 patients and 28 controls. The genotyping distribution was in accord Hardy-Weinberg equilibrium, in both groups, to the analyzed gene, showing no statistically differences between them ($_{MS}$ c^2= 4,49; p= 0,10). The *odds ratio* indicates no alterations in the risk of CAD to the analyzed genotypes yet (OR = 1,98, p = 0,26;). Our data suggest that MS A2756G polymorphism is not associated to DAC susceptibility.

Key words: CAD, polymorphism, MS, homocystein