RELATIONSHIP BETWEEN MBL2 POLYMORPHISM AND THE MOLECULAR MASS PATTERN OF CIRCULATING MANNOSE-BINDING LECTIN IN SERA OF HCV INFECTED PATIENTS

Cavalcanti, I.T.[1,2], Albuquerque, D. A.[1,2,3], Moura, P.M.[3] ; Beltrão, E.I.C.[1,2].

1 - Depto. Bioquímica, CCB, UFPE, Brazil; 2 - Laboratório de Imunopatologia Keizo Asami, LIKA – UFPE, Brazil; 3 - Dept. Patologia, ICB - UPE, Brazil.

Mannose-binding lectin (MBL) is a C-type lectin that activates the complement system and its function seems to depend to the degree of oligomerization of final molecule. A deficiency in MBL due to mutations at exon 1 of the human MBL2 gene causes low levels of protein and vulnerability to infection. We studied sera from HCV patients of known MBL genotype and assessed the MBL using mannan immobilized to nitrocellulose membrane and through elution by mannose. The observed patterns using a reducing electrophoresis and immunoblot with a rabbit polyclonal antibody revealed a range of high-molecular-mass form thru lower-molecular-mass form related to the genotypes. Wild type (A/A) showed bands of 32, 55, 66, 95 and 180 KDa; a homozygous mutation (00) showed predominantly bands of 55 e 66 and a heterozygote (A/0) showed an intermediary pattern tending to lower-molecular-mass. An apparent MBL deficiency does not in fact represents deficiency in MBL molecules but rather the presence of circulating oligomeric MBL with impaired function.

Key words: MBL, oligomers, molecular mass

Supported by: CNPq