Association of MBL2 Gene Polymorphisms and Serum Levels of Mannose Binding Lectin (MBL) with Platelet Levels in Patients with HCV

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INTRODUCTION: Hepatitis C virus (HCV) has hepatotropic character and its cytotoxic and/or immune-mediated action cause necroinflammatory lesions in the liver of varying intensity, which can result in HCV chronic disease and liver fibrosis. The mannose binding lectin (MBL), a human collectin encoded by the MBL2 gene, when associated with serine proteases (MASPs), activates the complement and coagulation systems, generating activation of thrombin-like factors, aiding the formation of blood clots by cleaving fibrinogen, factor XIII and platelet activation. The fibrinopeptides A and B, products of this cascade, are chemotactic factors release, attracting phagocytes and serving as a point of adhesion to cells of the immune system. MATERIAL AND METHODS: To determine the serum levels of MBL and the polymorphisms of the promoter (-550 and -221, alleles H/L and X/Y, respectively) and structural region (exon 1, alleles A/O) of MBL2 gene and correlate this data with platelet levels in patients with HCV, were evaluated a total of 56 HCV positive patients, which 29(57.47%) were male, aged between 32 to 75 years, who completed and responded to 48 weeks treatment with IFNa/ribavirin, treated at Osvaldo Cruz University Hospital/UPE and Institute of Liver of Pernambuco. To analyze the polymorphisms in MBL2 gene and quantify of serum levels of MBL, were used the real-time PCR and ELISA techniques, respectively. RESULTS AND DISCUSSION: The results suggest an increase in mean platelet when associated with haplotypes of low production of MBL (HYO/LYO, LYO/ LYO). There was also a relationship between low serum levels of MBL with higher average platelets, suggesting a connection between the complement system and coagulation system in HCV infection. CONCLUSION: Patients with high levels of MBL could show greater activation of the complement and coagulation systems, leading to a more vigorous response against the virus, which could be linked to the consumption of platelets.

Keywords: coagulation, Hepatitis C, MBL, platelets, polymorphism