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Cytokines and apoptosis genes polymorphisms influence the outcome of hepatitis C virus infection

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Introduction

Chronic hepatitis C virus (HCV) infection persists despite the presence of specific humoral and cellular immune [1-2] responses and the factors leading to viral clearance or persistence are poorly understood [3-6].

Aim

To investigate the possibility of a significant relationship between outcome of HCV infection and cytokines/apoptosis genes polymorphisms.

Materials and methods

The polymorphisms of genes: IL-1 (-889 IL-1α, -511 and +3954 IL-1β, IL-1-Ra), IL-18 (-137 and -607), IL-12p40 (-1188) and Apo1/Fas (-670) were determined by PCR-RFLP, PCR-SSP and PCR-VNTR; in 100 Tunisian hemodialysed patients infected by the HCV and 100 healthy blood donors. The patients were classified in two groups: G1 included 76 active chronic hepatitis patients (positive RNA-HCV) and G2: 24 hemodialysed having eliminated spontaneously virus (negative RNA-HCV).

Results

The univariate analyze of the genotypes and alleles frequencies of the cytokines polymorphisms studied do not reveal any positive or negative association statistically significant with the outcome of the HCV infection. Nevertheless, the frequency of genotype association [-37GC/-607CA] IL-18 is statistically higher among G2 patients (41,7%) compared to that at G1 hemodialysed (15,8%) (p=0,008, OR: 0.26, 95%CI: [0.10-0.73]). We found also, a significant increased frequency of AA genotype of Apo1/Fas gene in G2 patients (41,6%) than in G1 (17,5%) (p=0,026, OR=3,49, 95% CI [1,13-10,69]). Adjustment for known covariates factors (age, gender and genotypes) confirmed these univariate findings and revealed that the genotype association GC-CA of (-137and-607) IL-18 gene and AA genotype of Apo1/Fas gene were associated to the clearance of HCV (p=0.041 and p=0.017 respectively).

Conclusion

In Tunisian hemodialysed patients, spontaneous clearance of HCV seems to be associated with the two genotypes GC-CA of (-137and-607) IL-18 polymorphism and the AA genotype of ApoI /Fas gene.

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