Impact of IL-10, MTP, SOD2 and APOE gene polymorphisms on the severity of liver fibrosis induced by HCV genotype 4

Amr Ali Abass Ahmed Hemeda ,Amal Ahmed Mohamed, Ramy Karam Aziz, Mohamed Salaheldin-Abdelhakeem

Abstract

Complications of hepatitis C virus (HCV) chronic infection cause ~400,000 deaths worldwide annually. One complication, liver fibrosis, is influenced by host genetic factors. Genes influencing fibrosis include immune, metabolic, oxidative stress, and viral entry genes, such as interleukin 10 (IL10), microsomal triglyceride-transfer protein (MTP), superoxide dismutase-2 (SOD2), and apolipoprotein E (APOE)-encoding genes, respectively. Thus, correlating variations in these genes with HCV-induced fibrosis represents an attractive biomarker for the prognosis of fibrosis severity in chronically infected patients. Here, we aimed to test whether polymorphisms in IL10, MTP, SOD2, and APOE genes correlated with the severity of fibrosis induced by HCV genotype 4 (HCV-gt4) in a cohort of chronically infected Egyptian patients. Our results demonstrate a significant association between the severity of fibrosis and specific SNPs in IL-10, SOD2, and ApoE-encoding genes. Haplotype-combination analysis for IL10, MTP, SOD2, and APOE showed statistically significant associations between specific haplotype combinations and fibrosis severity. Identifying biomarkers correlating with the severity of HCV-gt4-induced fibrosis would significantly impact precision prophylaxis and treatment of patients at risk

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