Esam Elshimi et al., J Liver Disease Transplant 2017, 6:3
DOI: 10.4172/2325-9612-C1-009

Natural history of chronic hepatitis C development and progression as a consequence of iron and HFE or TfR1 mutations

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Introduction & Aim: Heavy iron overload is toxic to virtually all cells and tissues. There is growing evidence that only modest amounts of iron in the liver may serve as a co-morbid factor to increase the severity and/or rate of progression of liver disease. Aim of this study is to explore the role of iron, HFE mutations, and polymorphisms of the *TfR1* gene in the progression of chronic hepatitis C infection and possible therapeutic implications of iron overload on interferon therapy of patients with chronic hepatitis C.

Methods: From 3rd October 2012 to 6th January 2016, we studied 300 consecutive patients with chronic hepatitis C, correlating clinical, laboratory, histopathological, and genetic data. Frequencies of genetic variations were compared with healthy controls.

Results: HFE mutations were more common in patients than controls (25% vs. 11.7%, P=0.00006), and the C282Y mutation were more common in patients than controls (38.0% vs. 48.0%, P=0.02). Patients carrying C282Y had higher mean hepatic iron concentrations (P=0.02). Hepatic fibrosis was correlated with hepatic iron concentration (P=0.03). HFE and TfR1 polymorphisms bore detectable relation to disease severity and to response to interferon therapy.

Conclusions: Hepatic iron and HFE andTfR1mutations are co-morbid factors that increase progression of chronic hepatitis C and decrease the response to interferon therapy.

Biography

Esam Elshimi is working at Menoufia University, Egypt. He is the recipient of numerous awards for his research works in related fields. His research interests reflect in his wide range of publications in various national and international journals.

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