

Original Article

IFN- γ , IL-17, IL-22⁺ CD4⁺ subset in patients with hepatitis C virus and correlation with clinical factor

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Abstract: Background: CD4⁺ T cell responses in HCV infection have a crucial role in the immunopathology of hepatitis C virus (HCV) infection. Our aim was to investigate the frequency of Th1, Th17, and Th22 cells in HCV-infected patients and elucidate their role in the progression of the disease. Methods: Twenty-six HCV-infected patients and 26 healthy individuals were recruited. Peripheral blood mononuclear cells (PBMCs) were stained to separate CD4, IFN- γ , IL-17, and IL-22 producing cells using flow cytometry. Results: Results showed that the mean expression of IL-22 in CD4⁺ T cells was significantly lower in HCV-infected patients compared to healthy controls. About correlation with clinical factor and T subsets, a negative correlation between the frequency of CD4⁺ IFN- γ ⁺ cells and Thyroxine level (T4) was observed in the patients. The data showed a positive link between thyroid-stimulating hormone (TSH), cholesterol levels, and the frequency of Th17 cells. In addition, a positive correlation was seen between serum creatinine level with both Th1 and Th17. Ultimately, it was found that there was a positive link between viral burden and IL-17⁺ IL-22⁺ cells and a negative correlation between viral load and pure Th22. Conclusions: Our findings indicate that Th22 cells may play a part in the immunopathology of HCV and show the associations between Thelper subsets and the clinical signs of the disease.

Keywords: Hepatitis C, Th1, Th17, Th22, inflammation

Introduction

Hepatitis C is a liver infection that arises from the hepatitis C virus (HCV) categorized as a hepatotropic and non-cytopathic virus that is transmitted through contaminated blood products, sexual activity, and intravenous drug use [1, 2]. HCV infection remains a major global health problem, with around 58 million people of the world population being infected with it, while there is no vaccine for clinical use yet [3]. HCV infection is the prime cause of acute hepatitis, consequently resulting in chronic liver disease, which is characterized by fibrosis and cirrhosis and may ultimately lead to hepatocellular carcinoma (HCC) [4, 5]. In the chronic course

of HCV infection, persistent virus replication in the hepatocytes may promote the stimulation of inflammatory responses leading to progressive fibrosis and damage to the liver [6].

It has been established that during HCV infection, a wide variety of cytokines contribute to viral clearance but also tissue injury [7]. The pro-inflammatory cytokines are fundamental prerequisites for initiating the inflammatory cascades and sustaining the chronic infection, which eventually results in hepatocellular liver injury [8]. Besides, chronic and inflammatory courses of HCV infection might cause an imbalance between the expression of pro-inflammatory and anti-inflammatory cytokines and che-

the viral load, genotypes, the extent of liver fibrosis and damage, and duration of inflammatory responses. Notwithstanding these limitations, our results provide new insights into the role of different helper subsets and their cytokine profiles in the pathology of HCV infection, which would be a fruitful area for further research.

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Disclosure of conflict of interest

None.

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