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Dysregulation of T Cell Differentiation and the IL17A(+)Foxp3(+)Treg Subset in Chronic Hepatitis B Patients with Hepatitis Flare

Le Thi Thuy Hang ¹, Hoang Kim Tu Trinh ¹, Luong Bac An ², Nguyen Thi Tuyet ³, Phan Vinh Tho ⁴, Nguyen Tien Huy ⁵, Pham Thi Le Hoa ¹

Affiliations

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Abstract

The regulatory T (Treg) and T helper 17 (Th17) cells modulate the immune response in chronic hepatitis B virus (HBV) infection by promoting immune tolerance and restricting liver damage or stimulating inflammatory response and rendering hepatocyte injury. These cells act through signaling transcription factors and secreting cytokines. We aimed to observe the percentages of Treg, Th17 cells, and their messenger RNA (mRNA) level of forkhead box protein 3 (Foxp3) and retinoid orphan receptor γ t (ROR γ t) in the chronic hepatitis B (CHB)-infected group and CHB patients with hepatitis flare (HF). We recruited 103 participants, including 88 CHB-infected cases and 15 healthy controls (HCs) in Ho Chi Minh City. CHB cases were enrolled into two groups: HBeAg+ CHB infection (e+CHB); $n = 42$) and HF (including 20 mild HF and 26 severe HF [sHF]). The Foxp3(+)Treg and Th17 cells were

measured by flow cytometry. The mRNA levels of Foxp3 and RORyt were analyzed by real-time polymerase chain reaction. The percentages of Foxp3(+)Treg, of Th17, and the Foxp3(+)Treg/Th17 ratio were significantly higher in the sHF compared to the e+CHBI group. The sHF and e+CHBI groups had significantly higher mRNA levels of Foxp3 and RORyt compared to the HC group. Furthermore, a special subset, interleukin 17A(+)Foxp3(+)Treg cells, were observed with a significantly higher percentage in the sHF compared to the e+CHBI group. This finding revealed the contributions of this new subset on the severe flare cases. Our results explained the diversity of T cells and their subsets in the immune response in CHB. This subset should be further investigated as a specific tool in HBV immune response.

Keywords: Foxp3; IL17A(+)Foxp3(+)Treg; RORyt; Th17; Treg.

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