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Cytokines associated with antiretroviral induced hepatotoxicity in people infected with the human immunodeficiency virus type 1 in Northwest region of Cameroon

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Statement of the Problem: Cytokines provide one of the most targeted factors to investigate alterations of viral kinetics. The complexity of HAART induced hepatotoxicity has been extensively studied. However, the role of the cytokine in HAART induced hepatotoxicity has not been elucidated. This study for the first time assessed the cytokine profiles in patients with HAART induced hepatotoxicity.

Methodology: Blood samples were collected from a total of 68 HIV-1 drug naïve patients initiating HAART. Biochemical analysis of serum ALT and AST for hepatotoxicity was done based on enzymatic method at baseline, D30 and D180 of therapy. In addition, cytokines profiling were measured using Cytometric Bead Array with the Human Th1/Th2/Th17 CBA kit. Data were analyzed using GraphPad Prism 6 and SSPS.

Findings: Out of 68 patients recruited into the study with median (age range) 35.8 (18-61) years, significant mean ALT and AST increases with increase in treatment duration ($p<0.001$). Similarly, the prevalence of hepatotoxicity increased significantly ($P=0.000$) and was found to be 0(0.0%), 34(50.0%) and 47(69.1%) at day D0, D30 and D180, respectively. Cytokine IFN-γ, IL-17A, IL-10 and IL-2 levels decreased with increase in treatment duration. Comparison between treatment duration showed no statistical difference ($p>0.05$) with TNF-α, IFN-γ and IL-2 and a significant difference ($p<0.05$) was observed with IL-10, IL-6 and IL-17A (Fig. 1). TNF-α, IL-17A IL-6, were higher in hepatotoxicity patients compared to those without hepatotoxicity at D30 (Fig. 2) and D180 (Fig. 3) with a significant difference ($p<0.05$) in IL 17-A and IL 6.

Conclusion & Significance: The prevalence of hepatotoxicity increased with increase in treatment duration. Cytokines IL- 6 and IL-17A play a significant role in the pathophysiology of hepatotoxicity. As such they might be used as indicators of a global inflammatory state in hepatotoxicity and markers of disease progression in HAART induced hepatotoxicity.

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