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Systemic inflammation and altered NK and T cell activation drive neuro-psychiatric symptoms manifested by chronic hepatitis C and autoimmune patients

Amare Aregay, Verena Schlapov, Solomon Owusu Sekyere, Julia Hengst, Karin Weisenbörn and **Heiner Wedemeyer**
Hannover Medical School, Germany

Chronic hepatitis infections are generally known to be associated with several neuropsychiatric complications. In particular, more than 60% of patients with chronic HCV complain of having fatigue. In some cases these neuropsychiatric symptoms can lead to severe cognitive dysfunctions affecting quality of life of patients. However, these symptoms persist even after treatment-induced HCV clearance suggesting a role for other mechanisms instead of direct migration of HCV from liver to brain. In this study, we assessed if changes in circulating soluble inflammatory mediators (SIMs) as well as immune cell phenotypes could drive neuropsychiatric symptoms seen in chronic HCV, HBV and autoimmune hepatitis patients. A multivariate analysis was performed upon profiling 50 SIMS from 36 patients with HCV, HBV, autoimmune hepatitis (AIH) and primary biliary cirrhosis (PBC) using LUMINEX-based multiplex bead technology. *Ex-vivo* phenotype of NK-cell, CD3+ T cell, CD8+ T cell, CD4+ T cell and Tregs was also assessed from paired PBMCs from these patients. In addition, psychometric parameters were cross-sectionally assessed using several psychometric scores. Our data indicates that SIMS are significantly up-regulated and NK and T cells are less activated in patients with neuropsychiatric symptoms compared to patients without these symptoms. Interestingly, SIMS and cellular activation markers not only correlate amongst each other but also with neuropsychiatric symptoms. Of note, HCV patients had elevated SIMS compared to autoimmune patients. Indeed, upon principal component analysis (PCA), HCV patients showed a distinct clusterization based on differential concentration of SIMS and this correlated with neuropsychiatric symptoms. Overall, our data suggests that NK and T cells are weakly activated to combat hepatitis infections but induce inflammation leading to fatigue and other neuropsychiatric symptoms seen in HCV, HBV and autoimmune patients. The mechanism by which the elevated SIMs and weakly activated NK and T cells lead to neuropsychiatric symptoms is yet to be understood.

aregay.amare@mh-hannover.de