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Differential serum levels of ubiquitin C-terminal hydrolase-L1 between patients with or without white matter lesions

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Ubiquitin carboxy-terminal hydrolase-L1 (UCH-L1) has been established as a reliable and potential biomarker of neuronal damage. There is not much information about the effects of white matter lesions (WMLs) on serum UCH-L1 levels in white matter disease patients. This study was aimed to assess whether serum UCH-L1 levels are a reliable marker of brain damage in patients with WMLs. Serum levels of UCH-L1 were assessed by sandwich enzyme-linked immunosorbent assay (ELISA) in 74 patients with type 2 diabetes mellitus, depression, or vascular disease. MRI was performed by a neuro-radiologist blinded to clinical data. Of these 74 patient cases, 26 showed periventricular WMLs, 22 showed subcortical WMLs, and 26 displayed no well-defined WMLs (controls). Serum UCH-L1 levels were significantly different between the two groups ($p<0.05$). Further subgroup analysis proved that serum UCH-L1 levels in participants with subcortical WMLs were significantly increased when compared with controls ($p<0.001$), but there was no significant differences between controls and patients with periventricular WMLs ($p>0.05$). These findings suggest that serum UCH-L1 levels may serve as a novel biomarker for neuronal damage from WMLs, especially subcortical WMLs.

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The potential of neurogenesis induction by immune cells in patients with multiple sclerosis

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Background: Neural stem cells (NSCs) are abundantly present in MS lesions but they fail to mature into active neurons and oligodendrocytes. It was suggested that the immune system act in repairing damaged CNS tissue. We studied the production of pro-neurogenic factors: noggin, follistatin, differential screening-selected gene aberrative in neuroblastoma – DAN, leukemia inhibitory factor- LIF and anti-neurogenic factors: bone morphgenic proteins (BMPs), epidermal growth factor – EGF by immune cells and in the serum and cerebral spinal fluid (CSF) of RR-MS patients.

Methods: Supernatants of cultured PBMCs, serum and CSF from untreated RR-MS patients and matched health controls (HC) were tested for the factors by ELISA. The levels of the factors were also examined in separated T cells and monocytes using MACS. Immune regulation of these factor was studied by co-culturing PBMCs with various stimulatory conditions and pro and anti-inflammatory cytokines.

Results: PBMCs of RR-MS patients secreted significantly lower levels of noggin and follistatin (0.38 ng/ml± 0.14 and 12.26±2.28 pg/ml, respectively) *vs.* HC (1.69 ng/ml±0.51, 30.6 ±4.34 pg/ml, respectively, $p=0.03$ and $p=0.001$, respectively).

Discussion: Immune mediated neuro-regeneration is probably defective in MS patients. Our data suggests that immune cells in RR-MS patients may directly impair neurogenesis/ oligodendrogenesis by creating a non-supportive milieu for these processes, especially by T cells. A different expression profiles exist for different BMP antagonists: while noggin and follistatin were secreted by immune cells DAN was not secreted by immune cells and predominantly exists in the CNS. Elevated levels of DAN, in the CSF of RR-MS patients, suggest that a mechanisms intended to overcome the anti- neurogenic immunological milieu may exist in the CNS. The insufficient regeneration of neurons and oligodendrocytes in MS may be related to a defective immune mediated neurogenesis.

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