**MCP-1 is related with functional connectivity and phospho-Tau protein in amnestic Mild Cognitive Impairment**

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**Introduction:** Emerging evidence suggests that inflammatory events precede the clinical development of Alzheimer’s Disease (AD), as cytokine dysregulation has been observed also in patients with amnestic Mild Cognitive Impairment (aMCI) (1). Upregulation of a number of chemokines, including monocyte chemotactic protein-1 (MCP-1), is associated with pathological changes. It has been shown that human monocytes expressing MCP-1 may contribute to the maturation of senile plaques and phosphorylation of protein Tau (2). Inflammatory processes also play a role in pathological AD cascade, but their relationship with changes in functional connectivity (FC) of Default Mode Network (DMN) is still unknown.

**Materials and Methods:** 227 individuals (117 aMCI and 110 mild AD) underwent MRI at 3T to evaluate DMN FC as well as cerebrospinal fluid (CSF) analysis of amyloid-beta, phospho-Tau (p-Tau), total-Tau and MCP-1. A DMN mask was used as a template to extract each patients FC value of the DMN subregions. We used BD CBA Human MCP-1 Flex Set kit to quantify MCP-1. We aimed to verify if MCP-1 levels were associated to DMN FC and AD CSF biomarkers.

**Results:** In the aMCI group, MCP-1 was related with DMN left temporal FC (p = 0.032, R = 0.507); right hipocampal FC (p = 0.033, R = 0.503) and p-Tau (p = 0.023, R = 0.354), corrected to age. We did not find significant correlations in patients with mild AD.

**Discussion:** This study showed that MCP-1 in our aMCI patients was related with increased FC of DMN and with p-Tau. These findings suggest that inflammation seen in the early phase of the disease is associated with subtle connectivity changes and with a marker of neurodegeneration.

**Conclusion:** MCP-1 is related to different pathophysiological aspects of the predementia phase of AD (aMCI). Further studies are needed to evaluate MCP-1 reliability as an AD biomarker.

**References:**

(1)Xia MQ and Hyman BT. J Neurovirol 5(1):32-4, 1999; (2)Petersen RC, *et al.*, Arch Neurol 56(3):303-8 1999.