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Increased serum levels of inflammatory markers in patients with multiple sclerosis

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Multiple sclerosis (MS) is a common, autoimmune inflammatory disorder of the central nervous system (CNS) characterized by demyelination and succeeding axonal degeneration. The remission and recurrent relapsing disease affects young adults especially women. The cause of MS remains unclear; however, its pathogenesis involves a complex mechanism in the immune system, genetic and environmental factors. Inflammation of CNS plays a major role in the pathogenesis of MS. The trigger of the inflammation is unknown, but several studies have suggested that genetic, environmental, or infectious agents may influence development of the disease. Procalcitonin (PCT) is known as an important marker for sepsis. Moreover, PCT levels increased in patients with autoimmune disorders reflects systemic infection. According to recent reports the higher concentration of CRP is associated with MS. The proinflammatory cytokines such as interleukin-1 (IL-1), IL-6, tumor necrosis factor-α (TNF-α), interferons, macrophage migration inhibitory factor, HMGB1 (high mobility group B1) contribute to inflammation and neuronal damage via increased secretion of reactive oxygen species, including nitric oxide in MS. The higher levels of interferon-γ (IFN-γ), TNF-α, interleukins (ILs)-1β, 2, 4, 10, 13 have been shown in patients with MS. The glycoprotein YKL-40 (chitinase 3-like 1) synthesized by macrophages, neutrophil granulocytes, chondrocytes, synovial cells, bone cells, vascular smooth muscle cells, hepatocytes, mammary epithelial cells and other tissues. Although the function of YKL-40 is not fully understood, it is induced in inflamed tissues during inflammatory process. In addition, YKL-40 reflects neuroinflammation in acute and chronic neurological diseases such as MS, ALS, Alzheimer's disease and different stages of brain infarction. The primary aim of this study was evaluation of the role of inflammation, specifically of PCT, YKL-40 and some cytokines in subjects with MS.

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Influence of the innate immune system in a model of cortical inflammation as an approach to the progressive forms of multiple sclerosis

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Multiple Sclerosis (MS) is a neurodegenerative disease characterized by repeated inflammatory events, demyelination and axonal damage, along with loss of function. MS exhibited different forms: relapsing remitting (RRMS), primary and secondary progressive (PPMS, SPMS). Even though neuroinflammation is hallmark in every form of the disease, immunomoduladory treatments are beneficial in the early stages of MS, but ineffective in PPMS and SPMS. Recently, cortical lesions were described in PPMS and SPMS patients, which contribute to physical disability and cognitive impairment that characterize the progressive forms. The pathogenesis of cortical lesions is still unknown. Therefore, the cortical microenvironment could influence the degree of inflammation, tissue damage and the repair of the lesions. We developed a model of chronic and focal inflammatory triggered by the long term expression of one inflammatory cytokine, interleukin-1beta (IL-1b). Additionally, regional differences to the long term expression of IL-1b, were found between striatum and Substantia nigra. The aim of this work is to study the effect of the chronic expression of IL-1 in the cortex of adult rats and the effect of peripheral pro-inflammatory stimulus on these lesions. We used an adenovector expressing humanIL-1b (AdIL-1b) or beta-galactosidase (Adbgal) to induce chronic expression in the cortex. We performed behavioral, histological, immunohistochemical and molecular analysis. The long term expression of IL-1 in the cortex induces inflammation characterized by neutrophil recruitment and edema, neurodegeneration, and astro and microglia activation. The inflammation peaked at 15-21 days post injection and the lesion is restored by 30 days after injection. These results are correlated with a worse performance in the behavioral test of novel object recognition of IL-1 injected animals at the peak of inflammation (15-21 days), which is improved as far as the lesion is recovered (30 days). Additionally, systemic peripheral stimulation exacerbates the cortical lesion, with an increased inflammatory and glial response. In conclusion, we developed an experimental model of cortical inflammation and cognitive deterioration. The details of the pathophysiology of the progressive MS need to be better understood. A simple animal model allows the analysis of individual components of MS pathology, which could be use as targets for designing specific progressive MS treatments.

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