Abstract

Multiple sclerosis (MS) is an inflammatory autoimmune disease which is presented by environmental factors and genetic predisposition, increasingly affecting a large number of people worldwide. CNS inflammation is a local tissue response to stimulants and is characterized by induction of cytokines, chemokines and vascular permeability. Our goal in this study is to understand the micro-environment and immunopathogenesis in neurobiology of MS. A group of cytokines such as Interleukin-1 (IL-1), play an important role in MS pathogenesis. In spite of the fact that IL-1β is a pro-inflammatory cytokine and a mediator responding to inflammation of the nerves, but its role in chronic pathophysiologic conditions isn’t well known. IL-1β is mainly secreted from endothelial, T cells, fibroblasts, astrocytes and microglial, meanwhile it is secreted from monocytes, B and T cells as autoimmune mediator in MS patients. So, increment in serum, CSF and CNS lesions level of IL-1β titration in patients is of importance compared with healthy ones. IL-1β is like a sword of two edges, it is secreted from gelial cells in the hypothalamus and activates the neurons, reducing plaque volume. On the other hand, its neuronal excessive expression in MS patients causes severe clinical symptoms, apoptosis of the neurons and the loss of axons and myelin sheath. According to failure in several therapeutics based strategy, MS progression has been remained as a dilemma, leading clinical researchers look for novel agents for target therapy in MS immunoopathogenesis and microenvironment.

Keywords: IL-1β, Target Therapy, Multiple Sclerosis, Neuroinflammation

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