Neuroinflammation in Multiple Sclerosis

Gelareh Vakilzadeh\textsuperscript{1,2*}

\textsuperscript{1}Shefa Neuroscience Research Center, Khatam Alanbia Hospital, Tehran, Iran
\textsuperscript{2}Neuroscience Department, Tehran University of Medical Sciences, Tehran, Iran

Published: 11 April, 2017

Abstract

Multiple sclerosis (MS) is a complex disease which is correlated with increasing inflammatory factors, demyelination and axonal loss. In this auto-immune disease, Neuroinflammation is mediated by different types of T cells with macrophage/microglial activation and B cells involvement that interact in a collaborative manner. Focal inflammation is the main cause for the onset of relapses and could be presented in different regions of the central nervous system (CNS), but as the disease advances to progressive form, immune-inflammatory and oxidative stress pathways, which lead to axonal damage, play major role in the development of the disease. Neuroinflammation in MS can be studied for many approaches. For instance, a recent study have reported new classification of MS lesions including active, mixed active/inactive and inactive lesions for better comparison of tissue pathogenesis based on the inflammatory activity, demyelinating areas and duration of the disease. Moreover, it has been recently shown that the presence of leptomeningeal inflammation could be an important hallmark mainly for diagnosis of the progressive form of MS. Neuroinflammation could also be found in deep gray matter with pathological and clinical relevance. Finally, CNS injury in MS is associated with inflammatory reactions with further axonal degeneration. Therefore, control of inflammation with anti-inflammatory therapies must be taken into account as one of the main purposes of MS treatment parallel with other immunomodulatory and immunosuppressive treatments.

Keywords: Inflammation, Demyelination, Multiple sclerosis, Axonal degeneration

*Corresponding Author: Gelareh Vakilzadeh
Email: gelareh.vakilzadeh@gmail.com