The 2nd International Neurotrauma Congress &
the 4th International Roads Safety Congress

Shefa Neuroscience Research Center, Tehran, Iran, 18-20 February, 2015

The Neuroscience Journal of Shefaye Khatam

Volume 2, No. 4, Suppl. 3

Poster Presentation

The Immunological and Neuroimmunological Mechanisms of Traumatic Brain Injury

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Published: ***18 February, 2015***

Abstract

Cortical spreading depression (CSD) are associated with dramatic failure of brain ion homeostasis, efflux of excitatory amino acids from nerve cells, increased energy metabolism and changes in cerebral blood flow. There is strong clinical and experimental evidence to suggest that CSD is involved in the mechanism of migraine, stroke, subarachnoid hemorrhage and traumatic brain injury. Therefore, in the present study, we used the spreading depression model to investigate the effects of repeated spreading depression on peripheral and central adaptive immune responses. Moreover, we studied the effect of repetitive spreading depression on dark neuron density and expression of $GABA_A$ α and β, receptors. The results of the present study demonstrate that repeated spreading depression in rats induced elevated lymphocyte proliferation, IFN-γ, pro and anti-inflammatory cytokines in peripheral and central levels. Brain assays also demonstrated reduced alterations in $GABA_A$ α and β, GAD and HSP70 expression and enhanced the number of dark neurons. The findings could help to explain the interrelatedness of adaptive immunity, peripheral inflammation, and traumatic brain injury.

Keywords: Spreading Depression, Brain Injury, Inflammation, Immune Responses.

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