Glutathione Inhibited the Glutamate Receptor and Apoptosis-related Protein Following Traumatic Brain Injury in Animal Model

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Abstract

Traumatic brain injury (TBI) remains a major cause of death and disability. Oxidative stress is an important element of the injury cascade following TBI. Progressive compromise of antioxidant defenses and free radical-mediated lipid peroxidation is one of the major mechanisms of secondary TBI. NR2B is a glutamate receptor and its activation caused by TBI increasing neuronal cell death, along with caspase-3 as a hall mark of apoptosis. Glutathione is a potent free radical scavenger that might prevent secondary TBI damage and inhibited apoptosis. In this study, we aimed to demonstrate the effect of glutathione on inhibition of brain oxidative damage in TBI in animal model. Using TBI animal model, the expression of mRNA glutamate receptor gene and apoptosis-related protein was examined by PCR and immunohistochemistry. In this study, the expressions of mRNA NR2B in placebo group and groups with glutathione administration at 0, 3, and 6 hours after TBI were 328.14, 229.90, 178.50 and 136.14, respectively (p 80%); as expected, glutathione administered in 0, 3, and 6 hours groups had lower strong positive results of 50%, 16.7% and 16.7%, respectively (p = 0.025). This study showed that glutathione administration in TBI animal model decreased NR2B gene- and caspase-3 protein-expression that lead to inhibition of brain cell death. Our results suggest that glutathione, as a potent free radical scavenger, has a neuroprotective effect against oxidative damage and cell death induced by TBI in our animal model. [**MKB.** 2010;42(4S):29S].

Key words: Apoptosis, caspase-3, glutamate receptor, glutathion, traumatic brain injury