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Poster Presentation

Neuronal Death Following Posttraumatic Excitability and Seizure

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Abstract

Seizure may occur after mild traumatic brain injury (TBI), and the severity of TBI can be considered the most crucial factor for an increased risk of recurring seizures as well as for the development of posttraumatic epilepsy. However, the effect of seizures in epileptogenesis after mild TBI cannot yet be accurately confirmed. This study was designed to determine whether mild TBI increases seizure susceptibility as well as to investigate the histopathological and molecular consequences of seizure occurrence after mild TBI. Using a novel method of TBI induction, seizures were induced by sub-convulsive doses of pentylenetetrazole (PTZ) fifteen days after induction of focal mild TBI. Behavioral assessments were performed for one hour after PTZ injection and histopathological as well as molecular evaluations were performed. A significantly higher score and longer duration of seizure attacks were observed in the TBI+PTZ group compared to sham and TBI groups. A higher number of apoptotic cells was observed in the TBI+PTZ group compared to sham and TBI rats. Molecular investigations revealed higher levels of Bax/Bcl2 ratio, Caspase 3, and NF-κB in the TBI+PTZ group compared to the other animal groups. Our data indicated that seizure occurrence following mild TBI aggravates cell injury and death via activation of neuroinflammatory processes and may increase the risk of posttraumatic epilepsy.

Keywords: Epileptogenesis, Hippocampus, Posttraumatic seizure, Neuronal death, Neuroinflammation.

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