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Poster Presentation

Combination Therapies after Traumatic Brain Injury by Bumetanide and Dexamethasone Administration; a Hypothesis Study

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Abstract

Traumatic brain injury (TBI) initiates a cascade of numerous pathophysiological events that evolve over time. Despite the complexity of TBI, research aimed at therapy development has almost exclusively focused on single therapies, all of which have failed in multicenter clinical trials. A variety of brain insults, including traumatic brain injury, encephalitis, stroke, and status epilepticus (SE), have the potential to induce the development of epilepsy, particularly temporal lobe epilepsy (TLE) in humans and rodent models of TLE. The mechanisms underlying this process, which is termed epileptogenesis, are only incompletely understood, but include inflammation, neurodegeneration, blood–brain barrier disruption, alterations in expression and function of diverse receptors and ion channels, and development of hyperexcitability of neurons and neuronal circuits. The goal of the present study was to directly address this hypothesis by treating rats with the NKCC1 inhibitor bumetanide after a TBI. For the present experiments, Wistar rats, weighing 200–230 gr must be used. As previous studies on animal model of TBI treat by co-administration of dose dependent bumetanide and dexamethasone. Predict result: the result may show dexamethasone can inhibition second injury also combination of these two drugs may have reduction and inhibition of neural injury and inflammatory process. Co-administration of dose dependent bumetanide and Dexamethasone may have therapeutic role in traumatic brain injury.

Keywords: Traumatic Brain Injury, Bumetanide, Dexamethasone.

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