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Poster Presentation

The Neuroprotective Effect of Chloroquine in Animal Model of Traumatic Brain Injury

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Abstract

Traumatic brain injury (TBI) is one of the leading causes of morbidity and mortality in young adults and children, and is a leading public health problem worldwide. In TBI, neurological impairment is caused by immediate brain tissue disruption (primary injury) and postinjury cellular and molecular events (secondary injury) that exacerbate the primary neurological insult. However, the destructive molecular events that follow TBI evolve over several days, and therefore there is a window of opportunity during which therapeutic strategies may improve outcome. The antimalarial drug, chloroquine (CQ), has been reported as an autophagy inhibitor in a variety of disorders, including Alzheimer's disease and brain ischemia. To the best of our knowledge, no studies to date have examined the potential for CQ to provide neuroprotection in animal models of traumatic brain injury (TBI). Chloroquine (CQ) has long been used in the treatment and prevention of malaria, and less commonly has been employed in the treatment of autoimmune diseases, due to its immunosuppressive properties. In summary, this study demonstrated that neuronal autophagy was inhibited by postinjury treatment of CQ in a rat model of TBI. Furthermore, CQ attenuates secondary brain edema and improves cognitive functioning. These findings emphasize that CQ administered immediately following injury, could be neuroprotective against the damaging consequences of TBI, and we anticipate that this study has shed light on the potential use of CQ as a neuroprotective agent in the treatment of cerebral injuries.

Keywords: Chloroquine, TBI, Neuroprotective, Rat

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