عنوان مقاله:

Huperzine A protects against traumatic brain injury through anti-oxidative effects via the Nrf۲-ARE pathway

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خلاصه مقاله:

Objective(s): Traumatic brain injury (TBI) is a prominent health problem worldwide and it may lead to cognitive dysfunction, disability, and even death. To date, there is no effective treatment for TBI. Our previous study showed that Huperzine A (HupA) improved cognitive function in a mouse model of TBI. However, the detailed mechanism of HupA remains unaddressed. In this study, we investigated the possible mechanism of the neuroprotective effect of HupA. Materials and Methods: C۵۷BL/۶ mice were randomly divided into ۳ groups as sham, injured with vehicle treatment, and injured with HupA treatment groups. The Morris water maze task was used to evaluate the impairment of special learning and memory. Brain edema was as-sessed by measuring the wet weight to dry weight ratio. Malondialdehyde (MDA) and glutathione peroxidase (GPx) levels were measured for oxidative stress. Protein expressions of nuclear factor erythroid ۲-related factor ۲ (Nrf۲), heme oxygen-ase-۱(HO-۱), and synaptophysin were detected by Western blot. The brain sections were stained with hematoxylin-eosin (H&E) for histology study.Results: We found that HupA therapy improved histology and cognitive functional outcomes after TBI. HupA reduced brain edema in TBI mice. furthermore, HupA inhibited ox-idative stress. HupA promoted nuclear factor erythroid ۲-related factor ۲ (Nrf۲) nu-clear translocation and activated Nrf۲ after TBI. Conclusion: HupA protects against TBI through antioxidative effects via the Nrf۲-ARE pathway.

کلمات کلیدی:

Huperzine A, Neuroprotection, Nrf۲-ARE, Oxidative stres, Traumatic brain injuries

لینک ثابت مقاله در پایگاه سیویلیکا: