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Neuroprotective effects of hydrogen inhalation in an experimental rat intracerebral hemorrhage model

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Abstract

Objective: Hydrogen inhalation has been found to be neuroprotective and anti-oxidative in several brain injury models. Building on these studies, we investigated potential neuroprotective effects of hydrogen inhalation in a rat model of intracerebral hemorrhage (ICH), focusing on apoptosis and inflammation.

Methods: Forty-five 8-week-old male Sprague-Dawley rats were randomly divided into three groups (n = 15 per each group): a sham group, ICH group, and ICH + hydrogen group. Induction of ICH was performed via injection of 0.23 U of bacterial collagenase type IV into the left striatum. Hydrogen was administered via spontaneous inhalation. Mortality and neurologic deficits were investigated at 6, 24, and 48 h after ICH. To investigate the antioxidative activity of hydrogen gas, the expression of malondialdehyde was measured. Real-time polymerase chain reaction analyses of TNF-a, IL-1b, BDNF, and caspase-3 expression were used to detect anti-inflammatory and anti-apoptotic effects. Neuroprotective effect was evaluated by immunohistochemical and TUNEL staining.

Result: At 6, 24 and 48 h post-intracerebral hemorrhage, animals showed brain edema and neurologic deficits, accompanied by up-regulation of TNF-a, IL-b, BDNF, and caspase-3, which is indicative of neuroinflammation, neuroprotection, and apoptosis. Hydrogen treatment significantly reduced the level of oxidative stress, neuroinflammation, neuronal damage, and apoptosis-related genes. This was accompanied by increased neurogenesis and expression of growth factor-related genes at <24 h, but not 48 h, after ICH.

Conclusion: H_2 gas administration exerted a neuroprotective effect against early brain injury after ICH through anti-inflammatory, neuroprotective, anti-apoptotic, and antioxidative activity.

Keywords: Apoptosis; Hydrogen; Intracerebral hemorrhage; Key words; Neuroprotection; Oxidative stress; Rat.

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