Hydrogen

Hydrogen is a chemical element with chemical symbol H and atomic number 1. With an atomic weight of 1.00794 u, hydrogen is the lightest element on the periodic table. Its monatomic form (H) is the most abundant chemical substance in the Universe, constituting roughly 75% of all baryonic mass.

Non-remnant stars are mainly composed of hydrogen in the plasma state. The most common isotope of hydrogen, termed protium (name rarely used, symbol 1H), has one proton and no neutrons.

Strong clinical and experimental evidence has shown that hydrogen has potent protective cellular effects in various diseases. However, the effect of hydrogen on ICH remains unclear.

Hydrogen inhalation has been found to confer neuroprotection and anti-oxidative in several brain injury models. Building on these studies, Choi et al., from the Department of Neurosurgery, College of Medicine, Hanyang University, Seoul, Republic of Korea investigated potential neuroprotective effects of hydrogen inhalation in a rat model of intracerebral hemorrhage (ICH), focusing on apoptosis and inflammation.

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 hours 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.

At 6, 24 and 48 hours 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 hours, but not 48 hours, after ICH.

H2 gas administration exerted a neuroprotective effect against early brain injury after ICH through anti-inflammatory, neuroprotective, anti-apoptotic, and antioxidative activity [1)].

The present study investigates whether hydrogen has neuroprotective effects and improves functional outcome in the rat ICH model.

ICH model was generated by injecting 50 µl autologous tail artery blood stereotactically into the right caudate nucleus of Sprague-Dawley rats. Rats were randomly divided into four groups: sham, ICH/vehicle, ICH/hydrogen gas, and ICH/hydrogen-rich saline groups. Hydrogen treatment was performed for 3 days. The evaluation of functional outcome was done before, and at 24 and 72 hours

after ICH. Hemorrhage volume, immunohistochemistry for 8-hydroxy-2'-deoxyguanosine (8-OHdG), and brain water content were evaluated at 72 hours after ICH.

Hydrogen administration reduced the expression of 8-OHdG in the brain, but did not attenuate brain water content or improve functional outcome, regardless of administration route.

Hydrogen administration without surgery has no neuroprotective effect in the blood injection rat ICH model [2].

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2)
Takeuchi S, Nagatani K, Otani N, Wada K, Mori K. Hydrogen does not Exert Neuroprotective Effects or Improve Functional Outcomes in Rats After Intracerebral Hemorrhage. Turk Neurosurg. 2016;26(6):854-859. doi: 10.5137/1019-5149.JTN.14098-15.1. PubMed PMID: 27801926.