J Surg Res. 2018 Aug;228:238-246. doi: 10.1016/j.jss.2018.03.024. Epub 2018 Apr 11.

Hydrogen-rich water attenuates oxidative stress in rats with traumatic brain injury via Nrf2 pathway

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PMID: 29907217 DOI: 10.1016/j.jss.2018.03.024

Abstract

Background: Several studies have recently found that oxidative stress plays a pivotal role in the pathogenesis of traumatic brain injury (TBI) and may represent a target in TBI treatment. Hydrogen-rich water was recently shown to exert neuroprotective effects in various neurological diseases through its antioxidant properties. However, the mechanisms underlying its effects in TBI are not clearly understood. The purpose of our study was to evaluate the neuroprotective role of hydrogen-rich water in rats with TBI and to elucidate the possible mechanisms underlying its effects.

Materials and methods: The TBI model was constructed according to the modified Feeney weight-drop method. In part 1 of the experiment, we measured oxidative stress levels by observing the changes in catalase (CAT), glutathione peroxidase (GPx), and malondialdehyde (MDA) expressions. We also evaluated nuclear factor erythroid 2-related factor 2 (Nrf2) levels to determine the role of the protein in the neuroprotective effects against TBI. In part 2, we verified the neuroprotective effects of hydrogen-rich water in TBI and observed its effects on Nrf2. All the experimental rats were divided into sham group, TBI group, and TBI + hydrogen-rich water-treated (TBI + HW) group. We randomly chose 20 rats from each group and recorded their 7-d survival rates. Modified neurological severity scores were recorded from an additional six rats per group, which were then sacrificed 24 h after testing. Spectrophotometry was used to measure GPx, CAT, and MDA levels, whereas western blotting, reverse transcription polymerase chain reaction, and immunohistochemistry were used to measure the expression of Nrf2 and downstream factors like heme oxygenase 1 (HO-1) and NAD(P)H quinone oxidoreductase 1 (NQO1).

Results: GPx and CAT activity was significantly decreased, and MDA content was increased in the TBI group compared with the sham group at 6 h after TBI. MDA content peaked at 24 h after TBI. Nrf2 nucleoprotein levels were upregulated in the TBI group compared with the sham group and peaked at 24 h after TBI; however, no significant changes in Nrf2 mRNA levels were noted after TBI. Hydrogen-rich water administration significantly increased 7-d survival rates, reduced neurologic deficits, and lowered intracellular oxidative stress levels. Moreover, hydrogen-rich water caused Nrf2 to enter the cell nucleus, which resulted in increases in the expression of downstream factors such as HO-1 and NQO1.

Conclusions: Our results indicate that hydrogen-rich water has neuroprotective effects against TBI by reducing oxidative stress and activating the Nrf2 pathway.

Keywords: Hydrogen-rich water; Nrf2 pathway; Oxidative stress; Traumatic brain injury.

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