Hydrogen-rich saline improves memory function in a rat model of amyloid-beta-induced Alzheimer's disease by reduction of oxidative stress.

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Abstract
This study is to examine if hydrogen-rich saline reduced amyloid beta (Abeta) induced neural inflammation, and learning and memory deficits in a rat model. S-D male rats (n=84, 280-330g) were divided into three groups, sham-operated, Abeta1-42 injected and Abeta1-42 plus hydrogen-rich saline-treated animals. Hydrogen-rich saline (5ml/kg, i.p., daily) was injected for 14 days after intracerebroventricular injection of Abeta1-42. The levels of MDA, IL-6 and TNF-alpha were assessed by biochemical and ELISA analysis. Morris Water Maze and open field task were used to assess the memory dysfunction and motor dysfunction, respectively. LTP were used to detect the electrophysiology changes, HNE and GFAP immunohistochemistry were used to assess the oxidative stress and glial cell activation. After Abeta1-42 injection, the levels of MDA, IL-6, and TNF-alpha were increased in brain tissues and hydrogen-rich saline treatment suppressed MDA, IL-6, and TNF-alpha concentration. Hydrogen-rich saline treatment improved Morris Water Maze and enhanced LTP in hippocampus blocked by Abeta1-42. Furthermore, hydrogen-rich saline treatment also decreased the immunoreactivity of HNE and GFAP in hippocampus induced by Abeta1-42. In conclusion, hydrogen-rich saline prevented Abeta-induced neuroinflammation and oxidative stress, which may contribute to the improvement of memory dysfunction in this rat model.

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