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Postconditioning with inhaled hydrogen promotes survival of retinal ganglion cells in a rat model of retinal ischemia/reperfusion injury

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Abstract

Retinal ischemia/reperfusion (I/R) injury plays a crucial role in the pathophysiology of various ocular diseases. Intraperitoneal injection or ocular instillation with hydrogen (H2)-rich saline was recently shown to be neuroprotective in the retina due to its anti-oxidative and anti-inflammatory effects. Our study aims to explore whether postconditioning with inhaled H2 can protect retinal ganglion cells (RGCs) in a rat model of retinal I/R injury. Retinal I/R injury was performed on the right eyes of rats and was followed by inhalation of 67% H2 mixed with 33% oxygen immediately after ischemia for 1h daily for one week. RGC density was counted using haematoxylin and eosin (HE) staining and retrograde labeling with cholera toxin beta (CTB). Visual function was assessed using flash visual evoked potentials (FVEP) and pupillary light reflex (PLR). Potential biomarkers of retinal oxidative stress and inflammatory responses were measured, including the expression of 4-Hydroxynonenalv (4-HNE), interleukin-1 beta (IL1-β) and tumor necrosis factor alpha (TNF-α). HE and CTB tracing showed that the survival rate of RGCs in the H2-treated group was significantly higher than the rate in the I/R group. Rats with H2 inhalation showed better visual function in assessments of FVEP and PLR. Moreover, H2 treatment significantly decreased the number of 4-HNE-stained cells in the ganglion cell layer and inhibited the retinal overexpression of IL1-β and TNF-α that was induced by retinal I/R injury. Our results demonstrate that postconditioning with inhaled high-dose H2 appears to confer neuroprotection against retinal I/R injury via anti-oxidative, anti-inflammatory and anti-apoptosis pathways.

Keywords: Hydrogen; Inflammatory; Ischemia/reperfusion injury; Neuroprotection; Oxidative stress; Postconditioning.

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