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The Effects of Hydrogen-Rich Saline on the Contractile and Structural Changes of Intestine Induced by Ischemia-Reperfusion in Rats

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Background

Hydrogen has been considered as a novel antioxidant that prevents injuries resulted from ischemia-reperfusion (I/R) injury in various tissues. The study was designed to determine the effect of hydrogen-rich saline on the smooth muscle contractile response to KCl, and on epithelial proliferation and apoptosis of intestine subjected to I/R.

Methods

Intestinal I/R injury was induced in Sprague-Dawley rats using bulldog clamps in superior mesenteric artery by 45 min ischemia followed by 1 h reperfusion. Rats were divided randomly into four groups: sham-operated, I/R, I/R plus saline treatment, and I/R plus hydrogen-rich saline treatment groups. Hydrogen-rich saline (>0.6 mM, 6 mL/kg) or saline (6 mL/kg) was administered, respectively, *via* tail vein 30 min prior to reperfusion. Following reperfusion, segments of terminal jejunum were rapidly taken and transferred into isolated organ bath and responses to KCl were recorded. Samples of terminal jejunum were also taken for measuring malondialdehyde and myeloperoxidase. Apoptosis in intestinal epithelium was determined with terminal deoxynucleotidyl transferase-mediated deoxyuridine triphosphate nick-end labeling technique (TUNEL). Expression and distribution of proliferating cell nuclear antigen (PCNA) were detected with immunohistochemistry.

Results

Hydrogen-rich saline treatment significantly attenuated the severity of intestinal I/R injury, with inhibiting of I/R-induced apoptosis, and promoting enterocytes proliferation. Moreover, Hydrogen-rich saline treatment significantly limited the neutrophil infiltration, lipid oxidation, and ameliorated the decreased contractility response to KCl in the intestine subjected to I/R.

Conclusions

These results suggest that hydrogen treatment has a protective effect against intestinal contractile dysfunction and damage induced by intestinal I/R. This protective effect is possibly due to its ability to inhibit I/R-induced oxidative stress, apoptosis, and to promote epithelial cell proliferation.

Key Words: hydrogen, intestinal ischemia/reperfusion, antioxidant, oxidative stress, contractility, apoptosis

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« Previous

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Next »