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Hydrogen-rich saline prevents neointima formation after carotid balloon injury by suppressing ROS and the TNF-α/NF-κB pathway

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Abstract

Background: Reactive oxygen species (ROS) play a pivotal role in neointima hyperplasia after balloon injury. Molecular hydrogen has emerged as a novel antioxidant and has been proven effective in treating many diseases.

Objectives: We aimed to determine the mechanism by which hydrogen affects neointima formation.

Methods: We assessed the influence of a hydrogen-rich saline solution (HRSS) by daily injection in rats. Rats were euthanized to evaluate the neointima. ROS, malondialdehyde (MDA) and superoxide dismutase (SOD) and reduced glutathione (GSH), were detected in the injured artery. Macrophage infiltration and the production of inflammatory factors (i.e., IL-6, TNF-α and NF-κB) were also observed. The in vitro effects of hydrogen on vascular smooth muscle cell (VSMC) proliferation were also measured.

Results: HRSS decreased the neointima area significantly. The neointima/media ratio was also reduced by HRSS. There was a decline in the number of PCNA-positive cells in the intima treated with HRSS. Meanwhile, HRSS ameliorated the ROS and MDA levels and increased SOD, reduced GSH levels in the injured carotid. In addition, the levels of inflammatory factors, such as IL-6, TNF-α and NF-κB p65, were attenuated by HRSS. In vitro studies also confirmed the anti-proliferative capability of the hydrogen solution and ROS generation in VSMCs induced by PDGF-BB.

Conclusion: HRSS may have a protective role in the prevention of neointima hyperplasia and restenosis after angioplasty. HRSS may partially exert its role by neutralizing the local ROS and suppressing the TNF-α/NF-κB pathway.

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