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Hydrogen-rich saline protects against acute lung injury induced by extensive burn in rat model

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Abstract

Hydrogen has been reported to selectively quench detrimental reactive oxygen species, particularly hydroxyl radical, and to prevent myocardial or hepatic ischemia/reperfusion injury in multiple models. The aim of this study is to investigate whether hydrogen protects against severe burn-induced acute lung injury in rats. Rats were divided into four groups: sham plus normal saline, burn injury plus normal saline, burn injury plus hydrogen-rich saline, and burn injury plus edaravone. Animals were given full-thickness burn wounds (30% TBSA) using boiling water, except the sham group that was treated with room temperature water. The rats in hydrogen group received 5 ml/kg of hydrogen-rich saline, sham and burn controls obtained the same amount of saline, and the edaravone group was treated with 9 mg/kg of edaravone in saline. Lactated Ringer's solution was given at 6 hours postburn. The lungs were harvested 12 hours postburn for laboratory investigations. Severe burns with delayed resuscitation rapidly caused lung edema and impaired oxygenation in rats. These dysfunctions were ameliorated by administration of hydrogen-rich saline or edaravone. When compared with the burn injury plus normal saline group, hydrogen-rich saline or edaravone group significantly attenuated the pulmonary oxidative products, such as malondialdehyde, carbonyl, and 8-hydroxy-2'-deoxyguanosine. Furthermore, administration of hydrogen-rich saline or edaravone dramatically reduced the pulmonary levels of pulmonary inflammation mediators and myeloperoxidase. Intraperitoneal administration of hydrogen-rich saline improves pulmonary function by reducing oxidative stress and inflammatory response in severe burn-induced acute lung injury.

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