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Hydrogen-Rich Saline Attenuates Acute Hepatic Injury in Acute Necrotizing Pancreatitis by Inhibiting Inflammation and Apoptosis, Involving JNK and p38 Mitogen-Activated Protein Kinase–dependent Reactive Oxygen Species

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Abstract

Objectives

The objective of this study was to study the role of hydrogen-rich saline (HRS) on acute hepatic injury (AHI) in acute necrotizing pancreatitis (ANP).

Methods

Rats were used for this study and an ANP model was induced by injecting 5% sodium taurocholate into the biliary-pancreatic duct. Experiments were performed in 3 groups: sham, ANP, and ANP + HRS (HRS). Animals were killed at 3, 12, and 24 hours after operation, and then blood and tissue samples were harvested. Various physiological, histological, and cellular and molecular parameters were analyzed.

Results

Analyses of serum, lipase, alanine transaminase, and aspartate aminotransferase indicated that ANPinduced AHI model was established successfully and HRS attenuated hepatic dysfunction. Hepatic superoxide dismutase and malondialdehyde levels showed HRS against oxidative stress. Cellular and molecular analyses including p-p38, p-JNK, p-ERK, and caspase-3, caspase-9, NF- κ B, and TNF- α in hepatic tissues revealed that HRS attenuated ANP-induced AHI by inhibiting apoptosis and phosphorylation of JNK and p38, as well as NF- κ B activation.

Conclusions

Hydrogen-rich saline plays a protective role in ANP-induced AHI through inhibiting inflammation and apoptosis, involving JNK and p38 MAPK-dependent reactive oxygen species.

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