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Saturated hydrogen saline attenuates endotoxin-induced acute liver dysfunction in rats

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Abstract

To determine the effect of saturated hydrogen saline on lipopolysaccharide (LPS)-induced acute liver dysfunction, rats were divided into control, LPS, and LPS plus saturated hydrogen saline (LPS+H(2)) groups. Treatment with saturated hydrogen saline prolonged the median survival time and reduced liver dysfunction. Moreover, saturated hydrogen saline significantly reduced pathological alterations in liver tissues, the number of ballooned hepatocytes, serum tumor necrosis factor (TNF)-alpha and interleukin (IL)-6 levels, and myeloperoxidase (MPO) and malondialdehyde (MDA) levels in liver tissues ($P<0.05$). Cell apoptosis was detected in liver tissues after LPS treatment, and attenuated by saturated hydrogen saline treatment. Saturated hydrogen saline also decreased phosphorylated extracellular signal-regulated kinase (p-ERK), phosphorylated Jun kinase (p-JNK), nuclear factor-kappa B (NF-kappaB), and second mitochondria-derived activator of caspase (Smac) levels, and increased p38 activation ($P<0.05$). Thus, saturated hydrogen saline may attenuate LPS-induced acute liver dysfunction in rats, possibly by reducing inflammation and cell apoptosis. Mitogen-activated protein kinase (MAPK), NF-kappaB, and Smac may contribute to saturated hydrogen saline-mediated liver protection.

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