Antioxidative and Hepatoprotective Effects of Magnolol on Acetaminophen-induced Liver Damage in Rats.

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摘要

Abstract

Acute liver failure (ALF), an often fatal condition characterized by massive hepatocyte necrosis, is frequently caused by drug poisoning, particularly with acetaminophen (N-acetyl-p-aminophenol/APAP). Hepatocyte necrosis is consecutive to glutathione (GSH) depletion and mitochondrial damage caused by reactive oxygen species (ROS) overproduction. Magnolol, one major phenolic constituent of Magnolia officinalis, have been known to exhibit potent antioxidative activity. In this study, the anti-hepatotoxic activity of magnolol on APAP-induced toxicity in the Sprague-Dawley rat liver was examined. After evaluating the changes of several biochemical parameters in serum, the levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), and lactate dehydrogenase (LDH) were elevated by APAP (500 mg/kg) intraperitoneal administration (8 and 24 h) and reduced by treatment with magnolol (0.5 h after APAP administration; 0.01, 0.1, and 1 μg/kg). Histological changes around the hepatic central vein, lipid peroxidation (thiobarbituric acid-reactive substance/TBARS), and GSH depletion in liver tissue induced by APAP were also recovered by magnolol treatment. The data show that oxidative stress followed by lipid peroxidation may play a very important role in the pathogenesis of APAP-induced hepatic injury; treatment with lipid-soluble antioxidant, magnolol, exerts anti-hepatotoxic activity. Our study points out the potential interest of magnolol in the treatment of toxic ALF.

Key words Magnolol - Antioxidant - Acetaminophen - Hepatotoxicity - Lipid peroxidation