

Magnesium Ameliorates Lipopolysaccharide-Induced Liver Injury in Mice

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Abstract : Lipopolysaccharide (LPS) endotoxin, a component of the outer membrane of Gram-negative bacteria, is involved in the pathogenesis of sepsis. LPS administration induces systemic inflammation that mimics many of the initial clinical features of sepsis and has deleterious effects on several organs including the liver and eventually leading to septic shock and death. The present study aimed to investigate the protective effect of magnesium, a well-known cofactor in many enzymatic reactions and a critical component of the antioxidant system, on hepatic damage associated with LPS induced- endotoxemia in mice. Mg (20 and 40 mg/kg, po) was administered for 7 consecutive days. Systemic inflammation was induced one hour after the last dose of Mg by a single dose of LPS (2 mg/kg, ip) and three hours thereafter plasma was separated, animals were sacrificed and their livers were isolated. LPS-treated mice suffered from hepatic dysfunction revealed by histological observation, elevation in plasma transaminases activities, C-reactive protein content and caspase-3, a critical marker of apoptosis. Liver inflammation was evident by elevation in liver cytokines contents (TNF- α and IL-10) and myeloperoxidase (MPO) activity. Additionally, oxidative stress was manifested by increased liver lipoperoxidation, glutathione depletion, elevated total nitrate/nitrite (NOx) content and glutathione peroxidase (GPx) activity. Pretreatment with Mg largely mitigated these alternations through its anti-inflammatory and antioxidant potentials. Mg, therefore, could be regarded as an effective strategy for prevention of liver damage associated with septicemia.

Keywords : LPS, liver damage, magnesium, septicemia

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