



Impact of ursolic acid on chronic ethanol-induced oxidative stress in the rat heart

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Abstract:

Oxidative stress plays an important role as a mediator of myocardial damage produced by ethanol. This work was designed to investigate the effect of ursolic acid (UA), a reported radical scavenger and antioxidant, on oxidative stress in the heart of chronically ethanol-administered rats. Chronic ethanol administration (7.9 g/kg/day for 60 days) caused tissue damage that was manifested by the elevation of serum lactate dehydrogenase (LDH) and creatine phosphokinase (CPK). It also induced oxidative stress in the heart by increasing the lipid peroxidation process and by decreasing the antioxidant capacity of the heart. After the induction of toxicity (i.e. initial 30 days), treatment groups received UA (10, 20 and 40 mg/kg/day) along with ethanol for another 30 days. Coadministration of UA effectively (20 mg/kg/day) restored the activities of marker enzymes. It also controlled the oxidative stress by decreasing lipid peroxidation products (manifested by decreased lipid peroxidation products such as thiobarbituric acid reactive substances – TBARS, lipid hydroperoxides – LOOH and conjugated dienes – CD), increasing the activities of free radical scavenging enzymes (superoxide dismutase – SOD, catalase – CAT, glutathione peroxidase – GPx and glutathione S-transferase – GSH) and increasing the levels of non-enzymic antioxidants such as reduced glutathione, ascorbic acid and α -tocopherol. These findings demonstrate that UA acts as a protective agent against ethanol-induced abnormalities in the heart by reducing the lipid peroxidation process and by enhancing the antioxidant capacity.

Key words:

ursolic acid, triterpenoid, ethanol, heart, oxidative stress, antioxidants
