Effect of cardamonin on hepatic ischemia reperfusion induced in rats: Role of nitric oxide.

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Abstract

Ischemia reperfusion (I/R) injury is a cellular damage in a hypoxic organ following the restoration of oxygen delivery. It may occur during organ transplantation, trauma and hepatectomies. Nitric oxide (NO) effects during hepatic I/R are complicated. The iNOS-derived NO has a deleterious effect, whereas eNOS-derived NO has a protective effect in liver I/R. Cardamonin (CDN) is an anti-inflammatory molecule and a novel iNOS inhibitor, and Nɣ-Nitro-L-arginine (L-NNA) is a NOS inhibitor. L-Arginine is a precursor of NOS. This study was designed to investigate the possible protective effects of CDN on hepatic I/R and the role of NO. Wistar rats were randomly divided into 5 groups (Sham, I/R, CDN, L-NNA and L-arginine). Liver ischemia was induced for 45 min then reperfusion was allowed for 1 h. L-Arginine and CDN ameliorated the deleterious effects of I/R through reducing the oxidative stress and hepatocyte degeneration. Both molecules decreased the elevated inflammatory cytokines and increased the antiapoptotic marker, Bcl2. Both agents increased NO and eNOS expression and decreased iNOS expression. In conclusion, increased NO/eNOS and suppression of iNOS expression have protective effects on I/R injury. While inhibition of eNOS and reduction of NO have deleterious effects on I/R injury. For the first time, we demonstrated that cardamonin improved functional and structural abnormalities of the liver following I/R by improving oxidative stress and inflammation and increasing the availability of NO produced by eNOS. Treatment with cardamonin could be a promising strategy in patients with hepatic I/R injury in different clinical situations.

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