Discovery of the protective effect of glutamine on acute alcoholic liver injury

Huan Yang[1,2], Wei Guo[1,2], San-Qiang Li[1,2], Bing-Bing Zhang[1,2], Dan Wang[3], Wei-Gang Cheng[3], Shuning Chen[4]

Abstract:

OBJECTIVE: Our research aims to evaluate the important role of glutamine (Gln) in alcohol (ethanol)-induced liver damage.

MATERIALS AND METHODS: In our study, the mice were simultaneously divided into normal group, alcohol group, and Gln+alcohol group. After different treatments, we detected alanine aminotransferase (ALT), aspartate aminotransferase (AST), and liver index. Then, some histopathological examination was used to observe the damage of liver tissue, glycogen, and liver cell apoptosis in mice. In addition, the expression of apoptosis-related proteins Bcl-2, Bax, Caspase3, heat stress protein 70 (HSP70), cytochrome cytochrome P450-2E1, NFκB pathway-related proteins IkB-a, NFκB-p65, and tumor necrosis factor-α were detected in different groups by western blotting. The experiment *in vitro*, we used normal hepatocytes L02, after treatment with alcohol and Gln for 24 h, carried out CCK-8 cell proliferation detection and western blotting to detect the expression of related proteins.

RESULTS: Our results showed that, in serological testing, Gln can significantly reduce the levels of ALT, AST, and liver index in Gln+alcohol group; and in the histopathological examination, Gln can increase the glycogen content and decrease the apoptosis rate in Gln+alcohol group. In addition, the differential expression of IκBα, NFκB-p65, and other factors in Western blotting shows that the NFκB signaling pathway plays important role in acute alcoholic liver injury.

CONCLUSIONS: Our results showed that Gln played an important protective role in alcohol-induced liver injury in mice by regulating glycogen stores, apoptosis, anti-oxidative stress and inhibiting NF-κB signaling pathways in liver cells.

Keywords:

Acute liver injury, alcohol, glutamine, mechanism, protective effect