Glutamine reduces the expression of leukocyte integrins LFA-1 and Mac-1 in mice exposed to arsenic

Man-Hui Pai, Yi-Wen Chien, Ya-Hui Tsai, Ya-Mei Hu and Sung-Ling Yeh

Abstract

Chronic arsenic exposure results in an increased oxidative stress and inflammation in the body. Glutamine (GLN) is an amino acid considered to have immunomodulatory effects and attenuate the inflammatory reaction. This study was designed to examine the effect of GLN supplementation on inflammatory-related leukocyte integrin expression and in vitro splenocyte cytokine production in mice exposed to arsenic. Mice were assigned to the control and experimental groups. The control group drank deionized water, whereas the experimental group drank deionized water containing 50 ppm of sodium arsenite. Each control and experimental group was further divided into 2 subgroups and fed diets for 5 weeks. One subgroup was fed a semipurified diet, whereas the other subgroup was fed a diet where part of the casein was replaced with GLN, which provided 25% of the total amino acid nitrogen. The results showed that plasma GLN levels of mice in the arsenic group were significantly lower than those in the control groups. Glutamine supplementation reversed the depletion of plasma GLN in the arsenic group. β2 intergins, including leukocyte function–associated antigen-1 and macrophage antigen-1 expressed by leukocytes, were significantly higher in the arsenic group than the control groups. Glutamine supplementation reduced leukocyte integrin expression in mice exposed to arsenic. There were no differences in interleukin 4, interleukin 6, interferon γ, and tumor necrosis factor α production between the 2 arsenic groups when splenocytes were stimulated with mitogen. These results suggest that arsenic exposure results in depletion of plasma GLN and higher leukocyte integrin expression. Glutamine supplementation normalized the plasma GLN levels and reduced leukocyte leukocyte function–associated antigen-1 and macrophage antigen-1 expression. However, cytokine modulation may not be responsible for reducing leukocyte integrin expression in mice exposed to arsenic.