Effects of glutamine on adhesion molecule expression and leukocyte transmigration in endothelial cells exposed to arsenic

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摘要

Abstract

This study evaluated whether glutamine (GLN) concentration was related to endothelial surface molecule expression and the migration of polymorphonuclear neutrophils (PMNs) through endothelial cells (ECs) stimulated by arsenic. Human umbilical vein endothelial cells (HUVECs) and PMNs were treated with different GLN concentrations (0, 300, 600 and 1000 μ M) for 24 h. After that, we stimulated HUVECs for 3 h with 0.5 μ M arsenic, and PMNs were allowed to transmigrate to ECs for 2 h. HUVEC surface expressions of cell adhesion molecules and integrin (CD11b) and interleukin (IL)-8 receptor expressions on PMNs were measured. The transendothelial migration of PMNs was also analyzed. The results showed that cell adhesion molecule (CAM) and integrin expressions in arsenic groups were higher than in those without arsenic. Among the arsenic groups, the expression of CAMs on ECs and CD11b, and IL-8 receptor on PMNs was lowest with 0 μ M compared with the other GLN concentrations. Vascular CAM-1 on ECs and CD11b on PMN expression were higher with 300 μ M than with 600 and 1000 μ M GLN. IL-8 secretions from ECs and PMNs were higher with 300 μ M than with 600 and 1000 μ M GLN, and this was consistent with the expression of the IL-8 receptor on PMNs. Polymorphonuclear neutrophil transmigration was significantly higher with 300 μ M GLN than with other GLN concentrations. These results suggest that ECs and PMNs were activated after arsenic stimulation. Cell adhesion molecule expressions on ECs and PMNs were suppressed in the absence of GLN. A low GLN concentration comparable to catabolic conditions resulted in higher adhesion molecule expression and greater transendothelial migration of neutrophils. Glutamine administration at levels similar to or higher than physiological concentrations reduced IL-8 and adhesion molecule expression; PMN transmigration was also decreased after stimulation with arsenic.