Dietary arginine enhances adhesion molecule and Th2 cytokine expressions in mice with gut-derived sepsis

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Abstract

This study investigated the effects of arginine (Arg) on cellular adhesion molecules and intracellular Th1/Th2 cytokine expressions in mice with polymicrobial sepsis. Myeloperoxidase activity in organs was also analyzed to identify the extent of tissue injury resulting from neutrophil infiltration. Mice were randomly assigned to a normal group (NC), a control group, or an Arg group. The NC group was fed a standard chow diet. The control group was fed a common semipurified diet, and in the Arg group, part of the casein was replaced by Arg, which provided 2% of the total calories. After 3 weeks, sepsis was induced by cecal ligation and puncture (CLP) in the control and Arg groups. Mice in the experimental groups were sacrificed at 0, 6, 12, and 24 h after CLP, whereas mice in the NC group were sacrificed when the CLP was performed. Blood and organ samples were immediately collected for further analysis. Results showed that compared with the control group, plasma intracellular adhesion molecule-1 levels were significantly higher in the Arg group 12 and 24 h after CLP. Lymphocyte interferon-gamma expression in the Arg groups was significantly lower, whereas interleukin (IL)-4 expression was higher than the control group at various time points after CLP. The expression of lymphocyte CD11a/CD18 was significantly higher in the Arg group 6, 12, and 24 h after CLP than those of the corresponding control group and the NC group. PMN expressions of CD11b/CD18 in the Arg groups were higher than those in the control group at 12 and 24 h after CLP. The Arg group had higher IL-6 levels at 6 and 12 h in the kidney and intestine and 12 h in the lung after CLP. Higher myeloperoxidase activities were observed in the Arg groups at 24 h after CLP than those in the control group in various organs. These findings suggest that pretreatment with an Arg-supplemented diet enhances adhesion molecule and inflammatory cytokine expression during sepsis, which may aggravate the inflammatory reaction and increase neutrophil infiltration into tissues. In addition, Arg supplementation reduced intracellular interferon-gamma and enhanced IL-4 expression. This change may promote the Th2-type response and suppress the cellular immune response in gut-derived sepsis.