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Potential effects of sCD40L, CD36, IL-23 and arginase-1 molecules on the pathogenesis of brucellosis

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Brucellosis is a systemic infectious disease that can be transmitted from animals to humans, ranging from mild to severe clinical pictures. In our study, we aimed to reveal the roles of sCD40L, CD36, IL-23 and arginase-1 (ARG1) molecules in the pathogenesis of brucellosis. sCD40L binds and activates CD40 on antigen-presenting cells, thereby promoting the secretion of pro-inflammatory cytokines and nitric oxide (NO) synthesis. CD36 promotes phagocytosis and apoptosis, can participate in pro-inflammatory responses. IL-23 is a pro-inflammatory cytokine and contributes to the maintenance and pathogenicity of the Th17 cells. ARG1 mediates down-regulation of NO synthesis and suppression of T cell immune responses. 30 acute, 30 chronic patients diagnosed with brucellosis and 20 healthy controls were included in our study. In addition, analyzes have been associated with a predisposition to bone joint involvement (osteoarticular). Standard ELISA procedures were performed for all molecules evaluated in our study. The values of sCD40L, CD36, IL-23 and ARG1 molecules were found to be lower in the acute and chronic patient groups compared to the healthy control group, but there was no statistically significant difference for ARG1. There was no significant difference between acute and chronic groups. In our study, we found that serum levels of sCD40L, CD36, IL-23 and ARG1 decreased in patient groups compared to healthy controls. According to this result, it can be thought that the expression of these molecules is suppressed in brucellosis infection. We believe that researching suppression mechanisms can contribute to the treatment of the disease.

Keywords: Bacterial infections, effector molecules, immune response tracing, infectious disease