PSORIASIS

INVOLVEMENT OF HIGH MOBILITY GROUP BOX-1 (HMGB1) IN IMIQUIMOD-INDUCED PSORIASIS-LIKE MICE MODEL

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In the previous work, we have indicated that HMGB1, a pro-inflammatory cytokine, is closely associated with the pathogenesis of psoriasis. To further clarify the role of HMGB1 in the pathogenesis of psoriasis, we investigated the direct function of HMGB1 application and HMGB1 blockade in imiquimod (IMQ)-induced psoriatic mouse model in this study. Mice were treated with imiquimod (IMQ) to induce psoriasis-like inflammation, and consecutively injected with recombinant HMGB1 or PBS intradermally. Abundant cytoplasmic expression of HMGB1 was observed in lesional skin from IMQ-treated skin. The injection of HMGB1 into the IMQ-treated skin further aggravated the psoriasis-like disease, enhanced the infiltration of CD3+T cells, MPO+neutrophils, and CD11c+DCs, increased the number of γδ T-Cells, and up-regulated the mRNA expression of IL-6, TNF-α, IFN-γ and IL-17 compared with the PBS injection. Finally, by using anti-HMGB1 monoclonal antibody (mAb) or HMGB1 inhibitor Glycyrrhizin, we indicated that HMGB1 blockade reduced the number of γδ T-Cells, suppressed the mRNA expression of IL-6, TNF-α, IFN-γ and IL-17, and moderated clinical and histological evolvement in the IMQ-treated skin. Our data suggest that HMGB1 may act as a proinflammatory cytokine, contribute to the development of IMQ-induced psoriasis like inflammation. HMGB1 blockade might bring new direction for the suppression of psoriasis.

Key words: high mobility group box-1, glycyrrhizin, imiquimod, psoriasis, inflammation