PSORIASIS

MILD ELECTRICAL STIMULATION WITH HEAT SHOCK ATTENUATES THE SKIN INFLAMMATION IN MOUSE MODEL OF PSORIASIS

Yu Tsurekawa [(1)] - Misaki Morita [(1)] - Masataka Moriuchi [(1)] - Yoshio Nakano [(1)] - Mariam Piruzyan [(1)] - Sanako Fukami [(1)] - Mary Ann Suico [(1)] - Tsuyoshi Shuto [(1)] - Hirofumi Kai [(1)]

Graduate School Of Pharmaceutical Sciences, Kumamoto University, Department Of Molecular Medicine, Kumamoto, Japan [(1)]

Introduction: Psoriasis is a chronic inflammatory skin disorder. For long-term improvement in the QOL of psoriasis patients, the development of safe and effective, inexpensive therapeutic approach is necessary. Our laboratory has studied the therapeutic effects of optimized combination treatment of mild electrical stimulation (MES, 0.1-ms pulse width, 55-pulse per second) with heat shock (HS, 42˚C) on chronic inflammatory diseases (type 2 diabetes, kidney disease). These effects mostly depend on the activation of signaling molecules. Furthermore, clinical trials showed that MES+HS treatment effectively and safely improved the systemic metabolic disorders in obesity and type 2 diabetic patients. Recently, microarray data analysis showed that MES+HS treatment changes the gene expression profiles in mouse skin. However, the effect of MES+HS treatment on the pathophysiological condition of the skin had not been investigated.

Objective: To investigate the effect of MES+HS treatment on psoriatic skin.

Materials and Methods: We used imiquimod-induced psoriasis-like mouse model by daily application of imiquimod containing cream (15 mg) on mouse ear. Mice were treated with MES+HS for 10 minutes daily.

Results: MES+HS treatment significantly reduced imiquimod-induced ear swelling from 2 days after treatment. The combination treatment of MES and HS had synergistic effect on skin hyperplasia. The expression of keratinocytes proliferation markers such as PCNA and Krt6a were reduced in inflamed-skin. MES+HS treatment also lowered the mRNA expression of inflammatory cytokines (Il1β, Il17A, Il22). The recruitment of CD3-positive T cells was significantly reduced in MES+HS-treated skin. Furthermore, MES+HS treatment lowered the mRNA expression of antimicrobial proteins (Reg3γ, S100A8) which exacerbate the skin inflammation. In IL-17A-stimulated HaCaT cells, the production of antimicrobial proteins (S100A8, S100A9, β-defensin2) was significantly suppressed.

Conclusions: We found that MES+HS treatment improves the pathophysiological

characteristics in imiquimod-induced psoriasis-like mouse model. MES+HS treatment has potential usefulness for therapy of psoriasis.