
UPREGULATION OF ANTI-HUMAN RIBOSOMAL PROTEIN S6-P240, TOPOISOMERASE II α , CYCLIN D1, BCL-2 AND ANTI-CORNEAL ANTIBODIES IN ACUTE PSORIASIS

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The pathogenesis of psoriasis is complex and includes innate and acquired immunological abnormality and various environmental factors, such as mechanical trauma, infections and emotional stress. However, the real mechanisms in the skin lesion production are still largely unknown. In this study by Dr. Abreu-Verez and her colleagues, the presence of autoantibodies and the altered expression of many epidermal component proteins were examined by direct immunofluorescence and immunohistochemistry, using skin biopsies obtained from active psoriasis skin lesions [1].

Most interestingly, direct immunofluorescence showed multiple immunoglobulin and complement depositions in various areas in the skin; i.e., IgG in the stratum corneum, IgA in the epidermal basement membrane and dermal vessels, IgM in the stratum spinosum and sweat glands, IgE in the stratum corneum, C3 in the stratum corneum and dermal vessels, light chains in the stratum corneum and dermal vessels, and fibrinogen in the basement membrane and dermal vessels.

In addition, the authors also performed immunohistochemistry using specific antibodies various epidermal components. In the epidermis of the patient skin, myeloma oncogene-1 (MUM1), chromogranin, factor XIIIa, p53 and PNL-2 were negative, while surviving, S6-p240, cyclin D1, BCL2, topoisomerase II and Ki-67 were positive.

The results in this study first confirmed the old finding that psoriasis patients show complement activating autoantibodies to stratum corneum, that was reported by the group of Dr. Beutner [2]. In addition, the results in this study also suggested that psoriasis patients may have antibodies of various classes to stratum spinosum, basement membrane, sweat glands and dermal vessels, although the significance of these findings are not known at the present. Moreover, by immunohistochemistry using specific antibodies, the authors showed that expression of some epidermal components in the psoriatic skin may alter.

These changes may contribute to the pathogenesis in pemphigus.

CONFLICT OF INTEREST

The authors state no conflict of interest.

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