PSORIASIS

INVOLVEMENT OF AUTOINFLAMMATION IN EARLY PHASES OF PSORIASIS

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Introduction: Psoriasis is a common, immune-mediated, inflammatory disease mainly involving skin and joints, that is genetically determined, strongly influenced by epigenetic mechanisms, and possibly triggered by environmental factors. Although psoriasis is one of the most studied skin disorders, its pathogenesis is extremely complex and still needs to be clarified.
Recently, some authors suggested a bimodal immune activation characterized by an initial neutrophil-mediated 'autoinflammatory' phase and, subsequently, by an adaptive immunity phase where T cells predominate. At early stages, T lymphocytes are rare in lesional skin and there is an IL-1-related cytokine expression profile.

Objectives: The present study is aimed at providing experimental data to support the autoinflammatory nature of the early stages of psoriasis.

Material and methods: Biopsies of both lesional and normal skin were taken from 10 patients with early-phase psoriasis. Each sample was divided into two parts for obtaining both formalin-fixed paraffin-embedded (ffpe) tissue and frozen samples. Ffpe material was studied using immunohistochemistry and confocal laser microscopy.
Lesional and normal frozen samples were analyzed by means of a cytokine array method measuring protein expression of fourteen molecules, including pro-inflammatory cytokines, chemokines and other effectors.

Results: Among the 14 cytokines investigated with cytokine array, IL-1β and TGF-β2 were significantly more expressed in lesional than in normal skin.
Histopathologic and immunohistochemical analyses showed an upper dermal perivascular infiltrate mainly consisting of neutrophils and macrophages, with only scattered T-cells. Mononuclear cells were intensely positive for IL-1β, confirming protein array results.
On confocal laser microscopy, IL-1β co-localized mainly with CD66b and CD68, supporting a prevalent innate immunity profile in early-phases of disease.

Conclusions: The overexpression of IL-1 cytokine family and the predominant innate immunity cell profile in the lesional skin of early-phase psoriasis may support the role of autoinflammation, which pave the way to the involvement of adaptive immunity in late-phase psoriasis.