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# REDUCTION IN OKT6 POSITIVE EPIDERMAL LANGERHANS CELLS IN PSORIASIS

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Langerhans cells (LC) were defined by indirect immunoperoxidase using $OKT_6$ monoclonal antibody in the skin lesions of 10 untreated psoriasis patients. The distribution and the numbers of OKT6+epidermal LC in the lesions was low in comparison to that found in the normal skin. In some of the lesions, T6+LC could not be seen in the epidermis. This observation suggests that the reduction in LC may be a contributing factor in the pathogenesis of psoriasis.

**Key words :** Langerhans cells, $OKT_6$ monoclonal antibody, Psoriasis.

Psoriasis, a disorder of as yet unknown aetiology, is characterized by increased epidermopoiesis. Along with epidermal changes, dermal changes are seen in the form of mild to moderate inflammatory cell infiltrate, considerable capillary dilatation and tortuosity in the elongated dermal papillae. The dermal infiltrate is mainly mononuclear but a few polymorphonuclear leucocytes and eosinophils are also seen. A lot of information is available on the possible immunological defect in psoriasis.[1] For example, there is a decrease in T cell numbers with possible increase of immature T cells in the blood of patients with psoriasis. A decrease of suppressor cells in the blood has also been reported.[2] Currently, monoclonal antibodies are considered to be one of the valuable tools for delineating the cell types in tissues. These monoclonal antibodies have been used for characterizing the dermal infiltrates in psoriasis.[3]

Langerhans cells (LC) present in the epidermis have been shown to participate in delayed hypersensitivity reactions especially in allergic contact dermatitis in experimental animals.[4-6] These cells bear receptors for Fc component of IgG and $C_3$ component of complement, express Ia like antigens and contain high concentrations of ATPase enzymes.[7] LC can be defined by a specific T6 marker. This marker does not show any cross reaction with morphologically similar dendritic macrophages.[8,16] Scant information is available on the role of LC in psoriasis. We have recently used the monoclonal antibody OKT6 (defining $T_6$ antigen) to study LC in leprosy lesions.[9] In the present communication, the status of LC in psoriasis has been assessed using the OKT6 monoclonal antibody.

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## Materials and Methods

**Skin biopsies :** The patients with chronic plaque type psoriasis of more than 3 months duration were selected from the out patient clinic. Apart from the skin lesions of psoriasis, the patients were otherwise healthy. A typical skin lesion was biopsied. One half of the biopsy was fixed in buffered formalin and processed for conventional paraffin embedded blocks for histopathological analysis. The other half was collected in isopentane and frozen at -20°C for cryostat sections. Skin from normal individuals was used as controls. However, it was not possible to obtain skin from uninvolved sites of the same patients.

**Reagents :** Monoclonal antibody OKT6 (defining cortical thymocytes and LC) was obtained from Orthopharmaceutical corporation, U.S.A. as culture supernatent; Sheep anti-mouse Ig $F(ab)_2$ from New England Nuclear,

Boston, U.S.A.; and 3 : $3^1$ Diaminobenzidine tetrahydrochloride from Sigma Chemical Company, U.S.A.

**Immunoperoxidase :** 5-6 $\mu$m thick cryostat sections were cut and fixed in cold acetone-chloroform mixture (1:1 ratio) for 10 minutes. The sections were then dried, layered with normal rabbit serum and incubated at room temperature for 30 minutes. They were then washed in 0.85% saline and incubated further with 1: 20 dilution of the monoclonal antibody at room temperature for 45 minutes. Sections layered with phosphate buffered saline served as controls. Subsequently, the sections were washed for 10 minutes in 0.85% saline. They were then incubated with 1 : 80 dilution of peroxidase conjugated sheep anti-mouse Ig $F(ab)_2$ for 30 minutes, and wahsed in 0.85% saline for 15 minutes. The peroxidase reaction was performed using 3:$3^1$-diaminobenzidinetetrahydrochloride, hydrogen peroxide and characterized by brown membrane staining.[17] The number of $OKT_6$+ epidermal LC per high power field were quantitated.

**Results**

Only untreated patients of psoriasis were included in this study. All the biopsies showed histopathological features typical of psoriasis. Preliminary experiments had been carried out on the cryostat section of normal skin to assess the optimal dilution of the monoclonal antibody and the conditions required for staining. It was found that 1 : 20 dilution of the antibody was optimal. The OKT6 monoclonal antibody used was culture supernatant and contains very low concentrations of mouse globulin.

It is evident from table I, that the distribution and the number of OKT6+epidermal LC in the psoriatic lesions was very low in comparison to normal skin. A mean of 4.2 cells/high power field was seen in the lesions in comparison to 33 cells/high power field in the normal skin ($p < 0.001$). In some of the lesions, the T6+LC could not be detected in the epidermis.

**Table I.** Number of LCs in the skin lesions of untreated patients with psoriasis and controls.

| | Number of cases | Number of $OKT_6$+ cells/high power field Mean$\pm$SE |
|---|---|---|
| Normal skin (controls) | 5 | 33$\pm$1.4 |
| Psoriasis | 10 | 4.2$\pm$1.5 |

**Comments**

Currently, it appears that the basic defect in psoriasis is immunogical. Studies have shown imbalances in various subsets of lymphocytes,[2] participation of T lymphocytes in the pathogenesis of psoriasis;[3] autoimmune pathology has been implicated.[10] LCs are thought to play some role in psoriasis.[11] The present study was undertaken to assess the distribution of LC in the lesions of psoriasis with $OKT_6$ monoclonal antibody. The staining with this antibody delineates the morphology of LCs better than the conventional histochemical (ATP ase) method and can thus be useful and specific in the quantitation of LC.[11] Our earlier observations have shown that there is no significant difference in LCs count whether it is expressed per high power field or per 100 keratinocytes.[9]

The distribution and the number of OKT6+ epidermal LC in the lesion was markedly reduced in comparison to the normal skin as already reported recently.[11]

LC have been shown to be involved in the proliferation and keratinization of the epidermis.[12] The extent of epidermal cell proliferation was inversely related to the number of LC.[13] A similar relationship has been observed between the degree of parakeratosis and LC density.[14,15] So, lower number of LC in the psoriatic lesion may lead to a high degree of epidermal cell proliferation and parakeratosis, thus contributing to the pathology of the disease. Further, it has recently been shown that there is an increase of T helper cells with concomitant reduction of T suppressor cells in the skin of

psoriasis lesions.[3] It is possible that a reduction of LCs results in poor interaction of these antigen presenting cells with underlying T cells and this may possibly be the mechanism of initiation of psoriasis. Admittedly it is not possible to assign any definite cause or effect relationship to LCs in psoriasis from the present study.

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