DELAYED HYPERSENSITIVITY IN PSORIASIS

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Summary

Twenty two adult male patients of psoriasis and 100 normal volunteers were skin tested with DNCB, mumps skin antigen, candidin, coccidiodin, PPD, croton oil and histamine phosphate. Except for decreased sensitization seen with DNCB, the response of psoriatics to skin testing was comparable with the normals.

Lesions in psoriasis are characterized by accumulation of glycogen, increased cell proliferation and reduced terminal differentiation[1]. Drugs capable of inhibiting such cellular proliferation have been used as therapeutic agents[2]. Although such drugs may interfere with the development of immune response, the effects of psoriasis on the dermal immune response are not yet known. This report deals with the investigations carried out to study the delayed hypersensitivity in psoriasis.

Materials and Methods

Patients selection: Twenty two adult patients attending the Psoriasis Clinic of the Institute and 100 normal volunteers were enrolled for the study. Only those patients who had not been treated with any immunosuppressive drugs in any form and were free from any other disease were accepted. None of the patients had psoriatic arthropathy or erythroderma.

Skin testing: All the patients and volunteers were tested against a battery of antigens which were: dinitrochlorobenzene (DNCB, K and K Labs., Plain View NY), mumps skin antigen (Eli Lilly and Company, Indianapolis, IN), candida albicans (Dermatophytin 1:100, Hollister-Steir Laboratories, Spokane, WA), coccidiodin 1:100 (Cutters Laboratory, Berkeley CA) and purified protein derivative (PPD, State Serum Institute, Copenhagen). Nonspecific irritants, croton oil (Sigma Chemical Company, St. Louis, MO) and histamine phosphate were also used.

(i) Sensitization to DNCB. A sensitizing dose of 2000 ug DNCB in 0.1 ml acetone was applied, in a steel ring of 1 cm diameter, on the volar surface of right forearm. Simultaneously, 50 ug of DNCB in 0.1 ml acetone was also applied on the volar surface of the left arm to elucidate any prior sensitization. In addition, 0.1 ml of 10% solution of croton oil in acetone was applied on the left arm near the DNCB spot. Acetone was allowed to evaporate and spots were covered with

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gauze and surgical tapes for 24 hours when response to croton oil was noted. If the subjects did not show any reaction to DNCB even after fourteen days an additional challenging dose of 0.1 ml (50 ug/ml) of DNCB was applied on the left arm and the response noted after additional one week. Reactions were graded 0 to + + + + according to the method of Catalona et al[8].

(ii) Tests with intradermal antigens. Each subject was injected intradermally with 0.1 ml of each of various antigens listed above, with a disposable tuberculin 26 gauge needle syringe. Concentration of PPD injected to each individual was 1 TU in 0.1 ml. Skin reaction to all the recall antigens were evaluated after 72 hours. Induration of 5 mm or more was considered to be positive reaction to all the antigens except PPD when induration of 10 mm or more was considered as positive.

(iii) Histamine sensitivity: One drop of 1:100 solution of histamine phosphate was placed on the volar aspect of the arm and a prick made with a pricking needle (keeping the length of needle fixed) through the drop of the solution. A flare extending at least twice the size of the wheal was taken as positive (normal response). Wheal without flare or flare alone was taken as negative.

(iv) Irritant reaction with croton oil: 0.1 ml of 10 percent croton oil was applied on a circular area in a metal ring with a diameter of 0.6 cm. when dry it was covered with a non-porous patch kept in position for 24 hours with adhesive tape. Only erythema after 24 hours was labelled as negative, but marked erythema with small vesicles was considered a positive reaction.

Results

Table I shows the response to different antigens.

TABLE 1

Skin Reactivity in Psoriatics

Antigen tested	Patients No. reactive/ No. tested	%	Controls No. reactive/ No. tested	%
DNCB	17/22	77	20/20	100
Mumps	5/22	23	9/30	30
Candida albicans	6/22	27	12/30	40
PPD	14/22	64	72/100	72
Coccidiodin	0/22	0	0/100	0
Croton oil	20/22	91	28/30	93
Histamine	22/22	100	30/30	100

DNCB response: None of the patients or the normal volunteers were found to be presensitized to DNCB. 100% of the normal volunteers could be sensitized but only 77% (17 out of 22) ($p < 0.05$) patients responded to this antigen. However, non-specific skin reactivity to croton oil was similar in the two groups (93% in normal control and 91% in the patients). All the subjects responded to histamine.

Response to recall antigens. None of the subjects presented positive response to coccidiodin. 23% (5/22) patients responded to mumps antigens while 30% (9/30) of the normal subjects were positive to this antigen. 64% of patients and 72% of the normal volunteers gave positive response to PPD. 40% of normal volunteers and 27% of the patients were positive to candida. Although the intensity of response to various antigens was similar in the two groups, response to candida was very severe in the normal volunteers who invariably developed granulomas.

Discussion

Skin sensitivity to various antigens is a good indicator of the systemic immune response if the dermal chemistry has not been adversely effected by the pathological state. Skin irritants such as histamine and croton oil have been used to assess non-immunological response[4]. In the

present study, as the skin reactivity to these irritants in the patients with psoriasis is similar to that of the control subjects, response to antigens can be taken as a measure of their systemic immune status. Although response of patients to different antigens was depressed, this depression was more pronounced to the virgin antigen, DNCB, than to that of the various recall antigens. This may be due to the possibility that the immunologic memory to early antigenic exposures persists for long period of time. Positive reaction to PPD in a high per centage of subjects is usual as BCG vaccination is administered in childhood. It is to be noted that none of the subjects in this study showed positive response to coccidiodin and this may be due to an extremely low coccidiodal infection in our population.

Significance of our data in the pathogenesis of psoriasis remains unclear but a change in *in vitro* function of the peripheral lymphocytes of psoriatic patients has been reported[5,6]. Our studies showing a depression in delayed hypersensitivity of the skin may, therefore, be a result of systemic dysfunction of "specific lymphocytes" in psoriasis.

References

1. Van Scot EJ: Tissue compartments of the skin lesion of psoriasis. J Invest Dermatol, 59: 4, 1972.
2. Kojima A, Sugimoto, M and Endo, H: Epidermal protein metabolism directed towards keratinisation by hydrocortisone in the chick embryonic skin growing in a chemically defined medicine. Dev Biol, 48: 173, 1976.
3. Catalona WJ, Taylor PJ, Ralson AS et al: A method for dinitrochlorobenzene content sensitization. New Eng J Med 286: 399, 1972.
4. Kumar M and Axelrod, AE: Cellular antibody synthesis in vitamin B6 deficient rats. J Nutrn, 96: 53, 1968.
5. Rimbaud P, Meynadier J and Guilhou JJ et al: Anti IgG activity on peripheral blood lymphocytes in psoriasis, Arch Dermatol 108:371, 1973.
6. Guilhou JJ, Clot J, Meynadier J et al: Immunological aspects of psoriasis I. Immuno-globulins and anti IgG factors. Brit J Dermatol 94: 501, 1976.