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A Clinical Analysis of 11 Patients with Chronic Actinic Dermatitis in Korea

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Abstract

Chronic actinic dermatitis is a rare disease worldwide and also in Korea. However there has been no clinicohistologic and photobiological analysis of chronic actinic dermatitis in Korea. We examined 11 patients who were diagnosed as chronic actinic dermatitis and the results of this study were compared with previous reports. Most patients were elderly men who had erythematous papules or lichenified plaques on sun-exposed areas such as the face, neck, and dorsum of the hands with servere itching sensation. All patients had severe sensitivity to UVB and biopsied specimens showed findings of chronic eczema. Five patients had positive photopatch test materials. The patients were treated with systemic and topical steroid, cyclosporine and antihistamine.

Key Words: Chronic actinic dermatitis, clinical analysis

INTRODUCTION

Chronic actinic dermatitis (CAD) is an uncommon, eczematous photosensitive disease affecting mostly elderly men.[1] It has been defined on the basis of three criteria: 1) clinical; a persistent eczematous eruption on sun-exposed areas with possible extension into nonexposed areas, 2) photobiological; reduction in the minimal erythema dose to UVB irradiation, and possibly longer wavelengths, and 3) histologic; appearance consistent with chronic eczema with or without the presence of lymphoma-like changes.[2] Recently, persistent light reaction, actinic reticuloid, photosensitive eczema, photosensitive dermatitis and eczematous polymorphous light eruption have been considered as unifying terms for chronic actinic dermatitis.[3]

Until now there have been some reported cases of CAD in Korea,[4-8] but no clinicohistologic and photobiological analysis about CAD has been performed.

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In this study, we examined 11 patients who were diagnosed as CAD and the results were compared with previous reports.

MATERIALS AND METHODS

The criteria for patient selection

(1) a persistent eczematous eruption in sun-exposed areas over a 1-year period in the absence of a history of exposure to known photosensitizers;
(2) a decreased minimal erythemal dose (MED) to UVB and/or to UVA;
(3) histologically, epidermal hyperkeratosis or spongiosis and infiltration of lymphocytes or macrophage in dermis.

These criteria particularly exclude solar urticaria, polymorphous light eruption, actinic prurigo, transient light reactivity and photosensitivity associated with atopy.

Examination of patients

A history-taking and skin examination was performed. When possible, biopsy was done and history of exposure to photosensitizers was carefully taken. Phototest and photopatch tests were performed on

all patients.

Light source

Sellas sunlight with UVB filter (Dr. Sellmeier Co., Dusseldorf, Germany) served as the UVA light source, and a UVB fluorescent-lamp (FS 72T 12-UVB HO lamp, Elder Co., Ohio, U.S.A.) was the UVB light source. The output of the UVA and UVB light source was monitored with an IL700 radiometer (International Light Inc., Newburyport, MA, U.S.A.).

Phototest and photopatch test

On day 1, duplicate sets of photoallergen were applied and phototests to UVB and UVA were performed. On day 2, minimal erythemal doses (MED) to UVB and UVA were quantified. The MED was defined as the lowest UV dose that produces perceptible erythema uniformly covering the entire irradiated area. One set of photoallergens was exposed to UVA of 15 J/cm^2. On day 3 and day 5 the irradiated and nonirradiated photopatch test sites were examined.

The photoallergens used were the Scandinavian photopatch series. The responses were graded by a scoring system recommended by the American Academy of Dermatology; 1+: erythema, infiltration, possible papules; 2+: edema or vesicles; 3+: bullae and/or ulcers.

RESULTS

Between 1991 and 1997, 11 patients were examined for chronic actinic dermatitis with phototest. Three of the 11 patients were evaluated for porphyrin and revealed a normal porphyrin profile. Of the 11 patients, 10 were male and 1 was female. All were Korean. The age range was between 31 and 72.

Most patients had erythematous papules or lichenified plaques on sun exposed areas such as the face, neck and dorsum of the hands with itching sensation (Fig. 1). Seven cases were biopsied and all showed findings of chronic eczema such as irregular acanthosis, spongiosis and infiltration of chronic inflammatory cells (Table 1). Of the 11 patients phototested, the range of minimal erythemal dose (MED) to UVB was between 5 and 15 mJ/cm^2 (Fig. 2). Nine of 11 patients were tested to evaluate the MED to UVA. The range of MED to UVA was between 10 and 50 J/cm^2. All had a negative response to provocation for UVA. Five patients had a positive photopatch test response: one had responses to chloropromazine and tetrachlorosalicylanilide (TCSA);

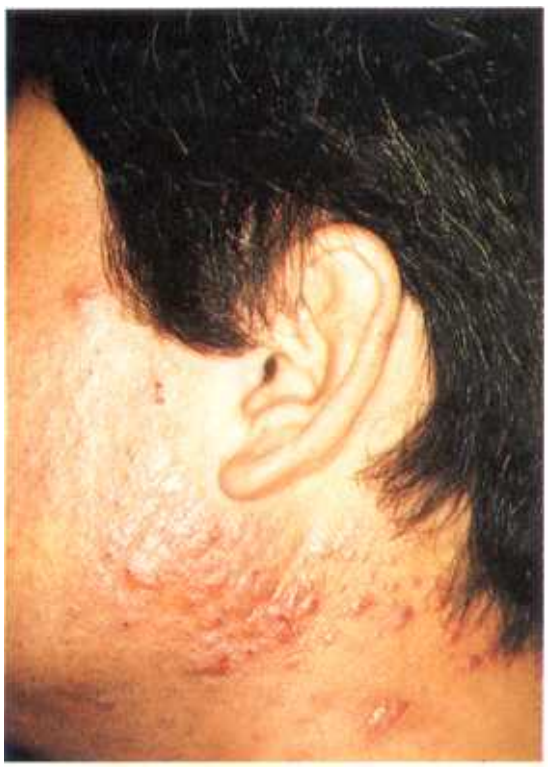

Fig. 1. Erythematous papules or lichenified plaques on sun-exposed areas.

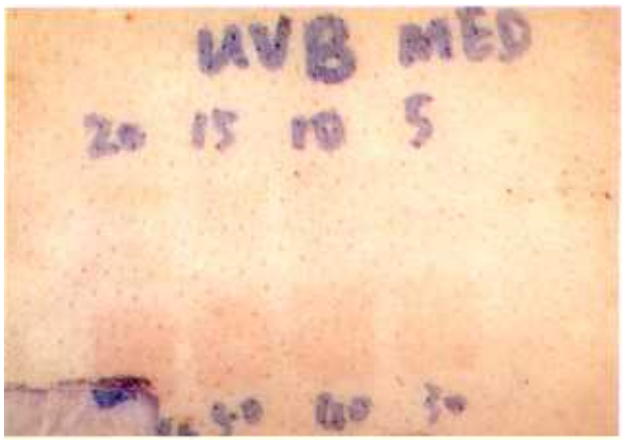

Fig. 2. The ranges of minimal erythemal dose (MED) to UVB was 15 mJ/cm^2.

Table 1. Summary of Clinical and Histologic Findings

No.	Sex	Age	Duration (yrs)	Histologic finding	Occupation
1	M	50	3	Not done	Keeper
2	F	50	20	Not done	Housewife
3	M	42	1	C/W* chronic eczema	Farmer
4	M	51	12	Not done	Accountant
5	M	42	1	Chronic eczema	Chemical co. worker
6	M	72	10	C/W chronic eczema	Public official
7	M	50	4	C/W chronic eczema	Carpenter
8	M	31	3	C/W chronic eczema	Pig breeder
9	M	56	9	C/W chronic eczema	Accountant
10	M	61	7	C/W chronic eczema	Fish farmer
11	M	47	7	Not done	Craftsman

* Consistent with.

Table 2. Summary of Photobiologic Results

No.	MED UVA (J/cm^2)	MED UVB (mJ/cm^2)	Photopatch test	Provocation with UVA
1	<20	<20	Negative	Negative
2	Uncheck	15	Chloropromazine tetrachlorosalicylanide	Negative
3	Uncheck	12	Negative	Negative
4	<10	<5	Trichlorocarbanilide promethazine chloropromazine	Negative
5	50	<5	Negative	Negative
6	<10	<5	Negative	Negative
7	<10	15	6-methylcoumarinebalsam of Peru	Negative
8	<20	<10	Negative	Negative
9	<10	<5	Trichlorocarbanilide promethazinechloropromazine	Negative
10	40	<10	Negative	Negative
11	<10	5	Promethazine	Negative

the second and third had responses to trichlorocarbanilide, promethazin and chloropromazine; the fourth had a response to 6-methylcoumarine (MC) and balsam of Peru; and the fifth had a response to promethazin hydrochloride (Table 2).

DISCUSSION

In 1962, Wilkinson first described one of the diseases induced by UV which affected men who developed a photoallergy after using soap containing tetrachlorosalicylanilide (TCSA).[9] He called this phenomenon "persistent light reaction" because the photosensitivity persisted even for years after contact with the photoallergens was eliminated. And some cases that had the same clinical picture, not photoallergy but photosensitivity, were reported. Later, these conditions were called "chronic photosensitivity dermatitis" or "photosensitive eczema".[10,11] Eventually, because all these conditions had the same clinical picture, the term chronic actinic dermatitis was proposed.

In our study, all patients had skin lesions on sun-exposed areas such as the face, V-neck zone, lower extremities and dorsum of the hands with a mild-to-severe itching sensation. The lesions were erythematous papules or plaque and some lesions were lichenified. The duration of disease was between 1 year and 20 years. The symptoms developed in spring and persisted in winter, becoming aggravated after sun exposure. Most patients were over 40 and male. This was similar to previous reports, in which most patients were elderly and male.[1]

He severity of chronic actinic dermatitis in a patient largely depends on professional and recreat-

ional activities, Tas well as UV intensity.[12-14] But the susceptibility of individuals also plays a role. Many of our patients had occupations working outside, such as farmers, and fish-farmers, and even those who worked indoors, but whose hobbies were outside, such as golf and tennis.

Phototests revealed that almost all patients had severe sensitivity to UVB. The range of MED to UVB was between 5 and 15 mJ/cm^2, which was very low compared to MED to UVB of 40−90 mJ/cm^2.[15] Five patients showed low MED to UVA which is less than 10 J/cm^2 (normal MED: 70 J/cm^2).[16] Five patients showed positive reactions to several photopatch test materials, but all showed a negative response to provocation tests which were performed to rule out polymorphous light eruption. For provocation, 50 J/cm^2 of UVA was irradiated 3 times on a patient's back and reading was done 1 day, 2 days and 3 days after irradiation. In photocontact allergy, the photopatch tests revealed positive responses and phototests were normal. One patient showed a positive reaction to chloropromazine hydrochloride which was contained in some drugs and to tetrachlorosalicylanilide which is an antiseptic of soap. Two patients showed positive reactions to chloropromazine, tetrachlorosalicylanilide and promethazine, which were contained in fluorescence dye. One patient showed a positive response to 6-MC, also known as balsam of Peru, which was contained in fragrances. However, the clinical symptom and treatment response were not different between the patients with positive photopatch test and the patients with a negative result. The results of phototests were most important in the diagnosis of CAD, and for the differential diagnosis of airborne dermatitis. In our study, many cases had a positive response to some photopatch allergens. In these cases, photoallergic dermatitis may have an important role in initiating and maintaining the disease. Frain-Bell et al. found that many patients in CAD had allergic contact dermatitis to the oleoresin of plants[17] and Addo et al. also reported allergic contact dermatitis to some fragrance materials.[18] Perhaps some contact allergens or photoallergens affect the provocation of chronic actinic dermatitis. Lim et al. proposed that the natural history of CAD is that photoallergic dermatitis evolves into persistent light reaction and then into actinic reticuloid.[19]

The pathogenesis of chronic actinic dermatitis is unknown, but several hypotheses have been proposed. First, photosensitizing drugs such as benozaprofen, quinidine and thiazide diuretics may cause chronic actinic dermatitis.[19-22] Among our patients, there were none who had taken these kinds of drugs. Autosensitization to skin proteins may be concerned. Kynurenic acid has been proposed as an endogenous photosensitizer.[23-26] Another explanation may be the cytopathologic effects of UVA on fibroblast.[27] Difficulties in neutralizing oxygen radicals could account for the cellular photosensitivities, and allergic factors or irritative factors may be involved.

The symptoms were relieved by the use of systemic or topical steroid and antihistamines. In 5 severe cases, systemic cyclosporine was used. Cyclosporine 100−200 mg a day for 4 to 8 weeks showed marked improvement of itching sensation and skin lesions and avoidance of sun-exposure and use of sunscreen were advised. According to Fotiades et al, sunscreens and their ingredients such as PABA (4-aminobenzoic acid) elicited the highest percentages of positive photopatch reactions, followed by fragrances and antimicrobial agents.[28] Therefore, patients should restrict the use of topical medication and skin care products to reduce the risk of sensitization. Protection from sun exposure with clothing is also effective. Partial effects of whole body application of mechlorethamine have also been reported.[29] Oral administration of 2.5 mg/kg of azathioprine daily can be used. In some reports, danazol 600 mg daily or cyclosporine, between 2.5 and 6.0 mg/kg daily, has been proposed.[30-32] Finally, PUVA therapy is one of the most effective treatments. The inhibitory effect on Langerhans cells and T lymphocytes can be attributed to clinical improvement. Also, it can be used in combination with oral or topical steroid.[33]

In conclusion, most patients in our study were elderly men who had erythematous papules or lichenified plaques on sun-exposed areas. All patients showed severe sensitivity to UVB and some showed sensitivity to UVA, while biopsied specimens showed spongiosis, acanthosis and infiltrations of inflammatory cells. For treatment, topical or oral steroid, cyclosporine and antihistamine were used.

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