



ORIGINAL ARTICLE

Chronic actinic dermatitis: A clinical study of 15 cases in northern Taiwan



Tzu-Lin Hsiao, Chia-Yu Chu*

Department of Dermatology, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan

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ABSTRACT

Background: Chronic actinic dermatitis (CAD) is an idiopathic photosensitive dermatosis induced by ultraviolet B (UVB), sometimes ultraviolet A (UVA), and occasionally visible light. Diagnosis is suggested by the clinical findings, typically a chronic eczematous rash on the sun exposed areas, and confirmed by phototesting, which demonstrates the abnormal photosensitivity. The aim of this study was to determine the characteristics of CAD in Taiwanese patients.

Methods: We retrospectively reviewed the clinical and photobiological features of all patients diagnosed as having CAD at our institute from 2002 to 2012.

Results: A total of 15 patients with CAD were identified. The mean age at diagnosis was 58.6 years (range, 28–82 years). All the patients were males. The face, neck, forearms, and dorsal hands were most commonly involved. Eight patients (53.3%) had decreased minimal erythema dose (MED) to both UVB and UVA; six patients (40.0%) had decreased MED to only UVB; one patient (6.7%) had decreased MED to only UVA. All were managed with photoprotection and topical corticosteroids. Four patients received azathioprine (50 mg twice a day to every other day) and one received prednisolone (10 mg per day to every other day).

Conclusion: In Taiwan, CAD affects elderly men more commonly. The most common phototest results were decreased MED to both UVB and UVA, followed by to UVB alone. All patients were managed with photoprotection and topical corticosteroids, and some also required systemic agents, in particular azathioprine.

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Introduction

Chronic actinic dermatitis (CAD), an idiopathic photosensitive dermatosis, is an eczema of the exposed areas induced by UVB, sometimes UVA, and occasionally visible light.¹ The term was originally introduced by Hawk and Magnus in 1979;² persistent light reactivity, actinic reticuloid, photosensitive eczema, and photosensitivity dermatitis are all considered variants of CAD.^{3,4}

Earlier investigations of CAD were mostly from countries with temperate climates. However, the disease appears to have worldwide distribution and affects all skin types. It has also been reported from subtropical or tropical regions, such as Australia,⁵ India,⁶ and Singapore.⁷ As diagnostic phototesting facilities are limited to only a few medical centers in Taiwan, CAD status in

Taiwan has never been studied.⁸ Here, we collected cases of phototesting proven CAD in our institute and reported the clinical features, photobiological characteristics, histological findings, and treatment in a Taiwanese population.

Methods

This was a retrospective analysis of medical records of phototested cases at the National Taiwan University Hospital, Taipei, Taiwan from January 2002 to August 2012. Information concerning their age, gender, clinical manifestations, phototesting results, histopathology, laboratory tests, and treatment was obtained for review. The diagnosis of CAD was based on the following criteria:⁵ (1) a persistent dermatitis in photodistributed areas without a history of exposure to a known topical or systemic photosensitizer; (2) a reduced minimal erythema dose (MED) to UVB or UVA or both on phototesting: MED to UVB ≤ 100 mJ/cm²,⁹ MED to UVA ≤ 15 J/cm²;¹⁰ (3) histology of the lesional skin, when available, showing chronic eczema, with or without cutaneous T cell lymphoma-like changes; and (4) no clinical/laboratory evidence of other

Conflicts of interest: The authors declare that they have no financial or non-financial conflicts of interest related to the subject matter or materials discussed in this article.

* Corresponding author. Department of Dermatology, National Taiwan University Hospital, 7 Chung-Shan South Road, Taipei 100, Taiwan.

E-mail address: chiayu@ntu.edu.tw (C.-Y. Chu).

autoimmune diseases with photosensitivity such as lupus erythematosus or dermatomyositis.

Phototesting was performed using the following light sources: a UV 801 KL (Waldmann, Villingen-Schwenningen, Germany) equipped with four TL 20 W/12 fluorescent tubes (spectral output 285–350 nm, peak 310–315 nm; Philips, Eindhoven, The Netherlands) for UVB and six UV-A Cleo 40 W fluorescent tubes (spectral output 315–400 nm, peak 355–365 nm; Philips) for UVA. The back of each patient was exposed to five graded doses of UVB ranging from 20 mJ/cm² to 100 mJ/cm², with an increment of 20 mJ/cm² and five graded doses of UVA ranging from 3 J/cm² to 15 J/cm², with an increment of 3 J/cm². The MED was defined as the smallest exposure dose required to produce a minimally perceptible erythema with well demarcated borders. Erythema responses were read 24 hours after irradiation; if no response was observed at 24 hours, a second reading at 48 hours would be taken.^{3–9} If needed, a photopatch test was performed according to the guidelines of the International Contact Dermatitis Research Group¹¹ to exclude photosensitive dermatitis or photoallergic contact dermatitis.

Results

Patient characteristics

A total of 15 patients with CAD were identified. The mean age at diagnosis was 58.6 years (range, 28–82 years). Most patients (>85%) had CAD after age 40 years. All the patients were males. Among the 15 patients, none of them were Fitzpatrick's skin phototype I, II, or VI. Three patients were phototype III, 10 patients were phototype IV, and two patients were phototype V. All 15 patients were Han Chinese. The interval between disease onset and diagnosis of CAD confirmed by phototesting ranged from 1 month to > 10 years. Demographic data are presented in Table 1 and Figure 1.

Clinical manifestations

The distribution of the eruptions, as shown in Figure 2, was characteristically located on the sun-exposed areas. The face (93%) was the most commonly affected, followed by the dorsal hands (80%), arms (73%), and neck (60%; Figure 3). The morphology of the lesions included papules, patches, and plaques. Most were described as scaly or lichenified lesions. Itching was the most common symptom.

Phototesting results

Eight patients (53.3%) had decreased MED to both UVB and UVA; six patients (40.0%) had decreased MED to only UVB; one patient (6.7%) had decreased MED to only UVA (Figure 4).

Histopathology

Four patients had skin biopsies taken from areas of existing dermatitis. The pathology reports showed superficial dermatitis, chronic dermatitis, hypersensitivity reaction compatible with allergic contact dermatitis, and chronic cheilitis, respectively.

Management

All patients were advised to ensure sun protection and were treated with topical corticosteroids. Most required medium to high potency topical corticosteroids for disease control initially. One patient received topical pimecrolimus for facial rash. Four patients received oral azathioprine treatment: the doses ranged from 50 mg twice a day to every other day, and the duration ranged from 2 weeks to > 2 years. Two of the patients were lost to follow-up 2–3 weeks after the use of the medication. Azathioprine was discontinued after 3 weeks in one patient due to nausea and vomiting. Another patient received prednisolone 10 mg/day for 24 days and then 10 mg every other day for 16 weeks with remarkable initial improvement but the patient was lost to follow-up thereafter.

Discussion

The diagnosis of CAD is suggested by the clinical findings, most commonly a chronic eczematous rash on the sun exposed areas, and corroborated by phototesting, which characterizes the action spectrum and degree of photosensitivity. When necessary, histology of the lesional skin is used to exclude other disorders. In this study, we retrospectively reviewed the clinical features, phototest results, and treatment of CAD in a Taiwanese population. Our study showed that CAD commonly affected elderly men, with a remarkable male predominance and a mean age at diagnosis of 58.6 years, in line with previous reports.^{3,5,7,12,13} The lack of female patients in this study might be due to the small sample size. There has been evidence that CAD may occur in young patients with atopic dermatitis;¹⁴ however, the patient in our study who presented the disease at a relatively young age (Patient 2, age 28 years) did not

Table 1 Clinical and pathological data of the 15 cases.

No.	Age	Sex	Int	Occupation	Histopathology	MED A	MED B	Treatment
1	51	M	4 y	Construction	NA	↓	↓	C, F, BV, H, P
2	28	M	2 y	NA	NA	—	↓	D
3	75	M	1 y	NA	NA	—	↓	F
4	50	M	2 mo	Postman	Superficial dermatitis	—	↓	D, H
5	59	M	1 mo	NA	NA	↓	↓	C, F, H
6	59	M	3 y	Salesman	Chronic cheilitis	—	↓	AZT 50 mg bid to qod for > 2 y
7	71	M	10 y	NA	NA	↓	↓	AZT 50 mg bid for 2 wk → LFU
8	40	M	3 y	Clerk	NA	—	↓	C
9	45	M	1 y	Construction	NA	↓	↓	AZT 50 mg qd for 3 wk, DC due to nausea and vomiting → C
10	50	M	1 y	Salesman	NA	↓	↓	C, F
11 ^a	61	M	3 y	Carpenter	Hypersensitivity reaction	—	↓	C, D, H, BD
12	69	M	5 y	Farmer	NA	↓	↓	AZT 50 mg qd for 2 wk → LFU
13	74	M	10 y	NA	Chronic dermatitis	↓	—	Prednisolone 10 mg qd for 24 d → 10 mg qod for 16 wk → LFU
14	66	M	1 mo	Businessman	NA	↓	↓	C, F, H
15	82	M	1 y	NA	NA	↓	↓	C, D, F, BD

AZT = azathioprine; BD = betamethasone dipropionate; bid = twice a day; BV = betamethasone valerate; C = clobetasol; D = desoximetasone; F = fluticasone; qd = each day; qod = every other day; H = hydrocortisone; Int = interval between disease onset and diagnosis of CAD; LFU = loss of follow-up; MED A = MED to UVA; MED B = MED to UVB; NA = not applicable; P = pimecrolimus.

^a Patient 11 has been reported previously.⁸

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