Three Cases of Chronic Actinic Dermatitis Treated with Systemic PUVA Therapy

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Chronic actinic dermatitis is an uncommon disabling eczematous photosensitive eruption. The skin lesion is characterized by persistent eczematous eruptions on sun exposed skin with possible extension into nonexposed areas. The phototest shows decreased minimal erythema doses (MED) to UVB and possibly UVA. Histological features demonstrate chronic eczema with or without the presence of lymphoma like changes. The present cases showed pruritic erythematous patches and plaques on sun exposed areas and showed decreased minimal erythema doses to both UVB and UVA. We herein report three cases of chronic actinic dermatitis treated with systemic PUVA therapy. (Ann Dermatol 9:(3) 197~200, 1997).

Key Words : Chronic actinic dermatitis, Systemic PUVA therapy

Chronic actinic dermatitis (CAD) is an uncommon, sometimes disabling eczematous photosensitive eruption affecting predominantly elderly men[1]. It was proposed by Hawk and Magnus[2] to embrace conditions including persistent light reactions, actinic reticuloid, photosensitive eczema, photosensitive dermatitis and eczematous polymorphous light eruptions. Although there were reports treated by some drugs such as azathioprine, danazol and hydroxychloroquine, or by phototherapy[3-6], this condition is difficult to treat because it is refractory to treatment, and recurs easily.

We herein report three cases of chronic actinic dermatitis improved by systemic PUVA therapy.

CASE REPORTS

Case 1

A 58-year-old man complained of pruritic erythematous patches and plaques on the sun-exposed areas (Fig. 1) that had been present for 5 years. Before the development of the eruptions, he had taken mequitazine for 1 month to treat the seborrheic dermatitis. Phototests for the minimal erythema dose (MED) of UVA and UVB were performed on his back with UV 800 (Waldmann Co., Germany, range: 315-395, peak at 355-365 nm) as the UVA light source, and UV 8001K (Waldmann Co., Germany, range: 280-400, peak at 305-315 nm) as the UVB light source. The phototest showed decreased MED to UVA (3 J/cm^2) and UVB (10 mJ/cm^2) (Fig. 2). A photopatch test with Scandinavian photopatch test series (Chemotechnique Diagnostics Ab, Sweden) and 1% mequitazine in vaseline revealed positive reactions to promethazine and mequitazine. A skin biopsy showed hyperkeratosis, acanthosis and mild perivascular mononuclear cell infiltrate in the upper dermis. He had been treated with hydroxychloroquine and cyclophosphamide for 2 years, but did not show improvement. So we recommended systemic PUVA therapy. After 8 weeks of systemic PUVA therapy, the symptoms including photosensitivity markedly improved (Fig. 3).

Case 2

A 56-year-old man had an 1-year history of pruritic erythematous patches and plaques on the sun-exposed areas. A phototest showed decreased MED

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Fig. 1. Pruritic erythematous patches and plaques on sun-exposed area in Case 1.

Fig. 2. Decreased MED to UVA (3 J/cm2) and UVB (10 mJ/cm^2) in Case 1.

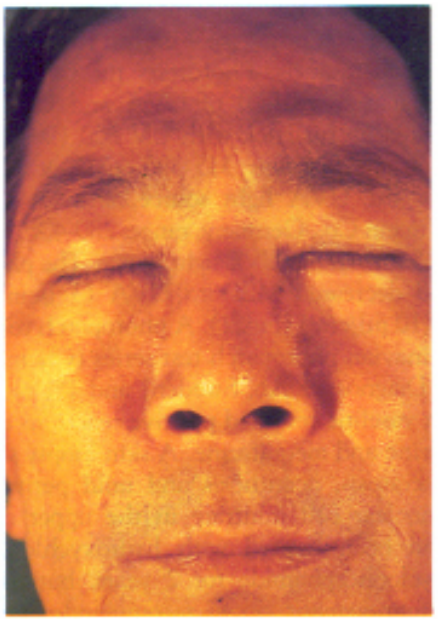

Fig. 3. Markedly improved state of skin lesions after 8 weeks of systemic PUVA therapy in Case 1.

to UVA (3 J/cm^2) and UVB (20 mJ/cm^2). A phototopatch test was negative. Systemic PUVA therapy was given twice weekly for 4 weeks, and there was clinical improvement.

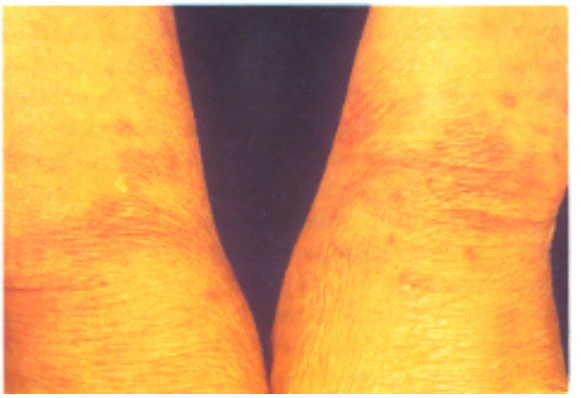

Fig. 4. Multiple erythematous scaly patches and plaques on sun-exposed area in Case 3.

Case 3

A 79-year-old man who had suffered from chronic obstructive pulmonary disease for 20 years, consulted the dermatology department due to pruritic erythematous patches and plaques on the exposed areas for 1 year (Fig. 4). A phototest revealed decreased MED to UVA (3 J/cm^2) and UVB (20 mJ/cm^2) (Fig. 5), and a photopatch test was negative. He had been treated with hydroxychloroquine for 1 year, but did not show improvement. So he was treated with systemic PUVA

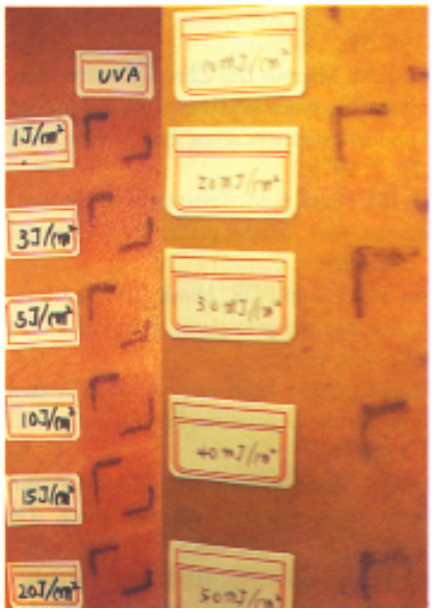

Fig. 5. Decreased MED to UVA (3 J/cm²) and UVB (20 mJ/cm²) in Case 3.

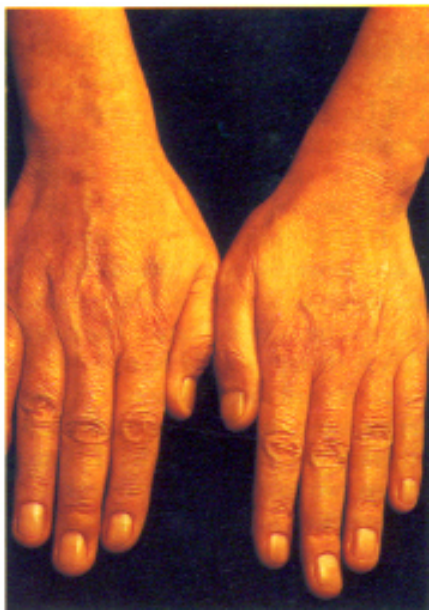

Fig. 6. Markedly improved state of skin lesions after 5 weeks of systemic PUVA therapy in Case 3.

therapy twice weekly for 5 weeks and his skin lesions improved (Fig. 6).

DISCUSSION

Chronic actinic dermatitis (CAD) can be diagnosed by three criteria[7]: 1. clinical; a persistent eczematous eruption on sun-exposed skin 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. All of the present cases fulfilled these criteria.

The exact mechanism of CAD remains unclear. The carrier protein originally bound to photoallergen may become changed during the process of photocontact dermatitis into a neoantigen, thus subsequently stimulating the immune system[8]. Lymphocyte typing studies also suggested a role for chronic antigenic stimulation[9]. Immunohistochemical analysis showed dermal infiltrates of predominantly T lymphocytes, being either CD4+ or CD8+[9]. Also, increase of Langerhans cells may play a role[10]. In addition, some allergic contact or photoallergic contact dermatitis caused by an agent such as musk ambrette, PABA, promethazine, and wood alcohol[9-11] have been reported in patients with CAD.

Case 1 showed positive photopatch test reactions to mequitazine and promethazine, but other cases were negative to this test. However, cases 2 and 3 may be possibly associated with systemic or contact photoallergy. Decreased MED to UVA rendered it necessary to use UVA doses that were much lower than the UVA doses used for photopatch tests in patients with a normal MED to UVA. This might have resulted in false negative photopatch tests. Furthermore, it is conceivable that these patients might have been sensitive to other photoallergens that were not included in the photopatch test series used.

The treatment of CAD usually begins with a complete avoidance of all known topical or systemic photoallergens. The patients are also recommended to avoid sun exposure, by using broad-spectrum sunscreens and covering up with clothing. Conservative therapy with topical steroid preparations is generally of limited benefit[6]. Systemic corticosteroid therapy is usually effective, but recurrence after cessation of therapy is invariable, and side effects of long-term high doses of prednisone are unacceptable[6]. Azathioprine and cy-

closporin have been known to be effective[3,4]. The mechanism of these drugs is a reduction in the number of Langerhans cell and suppressor T cells during treatment. But gastrointestinal, hematologic and hepatotoxic side effects limit its longterm use.

There were several reports of CAD treated with systemic PUVA therapy[5,6,8,12]. Systemic PUVA therapy was generally given two to three times a week, lasting about 2 months. Maintenance treatment may sometimes be indicated. The course of systemic PUVA therapy should be repeated annually for 2 to 7 years until the remission of disease occurs to prevent relapse. In the present cases, systemic PUVA therapy was initiated with 0.1 J/cm^2. Therapy was given two to three times a week, and doses were increased to a rate of 0.5 to 1 J/cm^2 per treatment.

The mechanism of systemic PUVA therapy for CAD is obscure. It may be explained by inhibitory effects on Langerhans cells and T lymphocytes, and increase tolerance by repeated ultraviolet radiation. Some authors commented on triggering or aggravation of CAD by initial systemic PUVA therapy[6,10], but this did not occur in the present cases. Highly sensitive patients may have to be hospitalized initially. To prevent this problem, systemic PUVA therapy initiated with very low doses and followed by gradual increments is needed. Also photosensitivity is suppressed with prednisone simultaneous with PUVA therapy.

We herein report three cases of chronic actinic dermatitis improved by systemic PUVA therapy.

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