

Low-dose Ultraviolet A1 Phototherapy for Treating Pityriasis Rosea

Sang Hee Lim, M.D., Sang Min Kim, M.D., Byung Ho Oh, M.D., Jong Hyun Ko, M.D.,
Yang Won Lee, M.D., Yong Beom Choe, M.D., Kyu Joong Ahn, M.D.

Department of Dermatology, Konkuk University School of Medicine, Seoul, Korea

Background: UVA1 phototherapy has recently demonstrated high levels of efficacy and tolerability for treating a variety of inflammatory and neoplastic skin diseases. **Objective:** The purpose of the present study was to assess the clinical efficacy of UVA1 (340~400 nm) phototherapy for treating pityriasis rosea and to assess the course of the disease after treatment. **Methods:** Fifteen patients with extensive pityriasis rosea were treated with low-dose UVA1 phototherapy (starting at 10~20 J/cm² and then it was increased to 30 J/cm²). The treatments were given 2~3 times a week until complete clearance of lesions was achieved or until there was partial improvement without further amelioration, in spite of 5 additional treatments. The rate of clearing was monitored by estimating the pityriasis rosea severity (PRSS) score and the pruritus score. **Results:** The extent of disease (PRSS) in all 15 patients lessened during the study (30.1 ± 3.6 vs. 2.0 ± 1.6 , respectively, $p < 0.05$). The overall reduction of the PRSS showed a significant improvement after the second or third treatment. The pruritus of 12 of 15 patients lessened during the treatment period, and it was unchanged in the remaining 3 patients. The mean previous duration of disease was 11.2 ± 4.9 days and this did not interfere with the successful outcome of UVA1 phototherapy. **Conclusion:** This study shows that UVA1 phototherapy is a useful, well-tolerated treatment option for patients suffering from pityriasis rosea with extensive eruptions and considerable pruritus. (Ann Dermatol 21(3) 230~236, 2009)

-Keywords-

Pityriasis rosea, Pityriasis rosea severity score, UVA1

Received July 9, 2008, Accepted for publication November 7, 2008

Reprint request to: Yong Beom Choe, M.D., Department of Dermatology, Konkuk University School of Medicine, 4-12, Hwayang-dong, Gwangjin-gu, Seoul 143-729, Korea. Tel: 82-2-2030-5170, Fax: 82-2-2030-5179, E-mail: cyb@kuh.ac.kr

INTRODUCTION

Pityriasis rosea (PR) is an acute, self-limited papulosquamous disorder that begins with the appearance of an initial plaque most often on the trunk, and this is followed in about a week or two by the development of an analogous spotty rash and it usually persists for 4~7 weeks. The exact etiology of the disease is still unknown, although active infection with both human herpes viruses 6 and 7 is thought to play a role in PR¹⁻³. No specific therapy is available⁴ and in many cases none is needed; however, some patients have an extensive eruption and considerable pruritus⁵⁻⁷. For patients with severe pruritus, experts have recommended treatment with zinc oxide, calamine lotion, topical steroids, oral antihistamines and even oral steroids⁷. Ultraviolet radiation, through artificial sources or intentional exposure to natural sunlight, has been recommended to decrease the duration of the rash and the intensity of itching in patients with pityriasis rosea⁸⁻¹¹.

Ultraviolet (UV) A1 (340~400 nm) phototherapy has recently demonstrated high levels of efficacy and tolerability for treating a variety of inflammatory and neoplastic skin diseases that are characterized by epithelial and dermal infiltrates rich in T lymphocytes¹². The mechanism of action is largely unknown, but the therapeutic activity of UVA1 light could be at least partly related to the UVA1's photophysical properties and photobiologic effects. Approximately 10% to 40% of UVA1 applied to the skin can penetrate the epidermis and so target the CD4+ and CD8+ mononuclear cells infiltrating the epidermis, as well those in the deep dermal compartments¹³. UVA1 phototherapy has been conducted for the treatment of atopic dermatitis, systemic lupus erythematosus (SLE), vitiligo, localized scleroderma and PLE, and it has been used for a variety of other indications

for which it may be effective¹⁴⁻³⁵.

Therefore, we evaluated the use of low-dose (usually around 10~30 J/cm²) UVA1 therapy as a possible therapeutic approach to treat pityriasis rosea. In the present study, we report the clinical results obtained with using UVA1 therapy for treating 15 patients affected by pityriasis rosea.

MATERIALS AND METHODS

Patients

Fifteen patients (5 women and 10 men) with extensive pityriasis rosea participated in the study. At entry to the study, the average duration of generalized eruption was 11.2 days (mean: 4 to 21 days). The patients younger than 18 years and the pregnant women were excluded. The patients' mean age was 30.6 years (range: 19~50). For the patients with typical clinical manifestations, the diagnosis was based on clinical criteria; biopsies were done on 6 patients to confirm the diagnosis. All the patients had no history of photosensitivity, skin malignancy, abnormal reactions to sunlight or immunosuppression, and they were not taking potentially phototoxic or immunosuppressive medication. The patients were asked not to expose themselves to ambient sunlight during the study. No other treatment had been given for at least 3 months prior to the start of UVA1 radiation therapy. Two patients had type II skin, eight patients had type III skin and five patients had type IV skin (Table 1).

Radiation source and dosimetry

The UVA1 irradiation equipment consisted of a Waldmann

7001 K cabin fitted with Waldmann TL10R low-pressure lamps (Waldmann GmbH, Schwenningen, Germany), which generate UVA1 wavelengths in the 340~400 nm range with a peak emission at 370 nm (Fig. 1). The irradiance was determined by an intrinsic UV meter and it was found to be approximately 80 mW/cm² at skin level.

Irradiation protocol

The patients were treated with low-dose UVA1 phototherapy (starting at 10~20 J/cm² and it was then increased to 30 J/cm²). The therapy regimen and the number of treatment were determined by the skin conditions (Table 1). Dose increments of 20% at each visit were applied unless erythema developed, and if it did, the dose increments were reduced to 10%. The patients received a variable number of total exposures (from 3 to 10) to the whole body, and these were given 2~3 times a week. Treatments were continued until complete clearance of lesions was achieved or until partial improvement without further amelioration was accomplished, in spite of 5 additional treatments. Additional therapy was not allowed except for the use of emollients.

Clinical grading the severity of pityriasis rosea

The severity of the disease was determined according to the Pityriasis Rosea Severity Score (PRSS)³⁶. Two areas were assessed for determining the PRSS: (1) the head and trunk (t) and (2) the upper and lower extremities (e). The extent of the disease was first assessed with a 0 to 3 scale (0=absence of lesions, 1=1 to 9 lesions, 2=10 to 19 lesions, 3=≥20 lesions). To evaluate the severity of the

Table 1. Characteristics of the patients with pityriasis rosea treated with UVA1 phototherapy and the treatment regimen

Patient no.	Gender	Age (y)	Fitzpatrick skin type	Disease duration (day)	Starting dose (J/cm ²)	Maximum dose (J/cm ²)	Total no. of treatments	Cumulative UVA1 dose (J/cm ²)	Adverse effect
1	F	32	III	5	10	30	6	120	—
2	M	26	III	7	20	30	5	110	—
3	M	26	II	15	10	20	6	110	Erythema
4	M	26	IV	11	10	30	8	160	—
5	M	19	III	13	20	30	9	180	—
6	M	43	III	12	10	30	7	150	Erythema
7	F	27	IV	5	10	10	3	30	PMLE
8	F	46	II	21	10	20	10	210	—
9	M	25	III	15	20	30	6	130	—
10	M	30	IV	13	20	30	6	120	Pruritus
11	F	24	III	4	20	30	7	150	—
12	F	19	III	6	10	30	8	170	Burning sensation
13	M	32	IV	14	10	30	5	100	—
14	M	50	IV	16	10	30	7	130	—
15	M	34	III	11	10	30	5	110	—
Mean ± SD		30.6 ± 9.3		11.2 ± 4.9	13.3 ± 4.9	27.3 ± 5.9	6.5 ± 1.8	132.0 ± 41.8	

lesions, three target symptoms termed erythema (E), infiltration (I) and scale (S) were assessed according to a scale of 0 to 3, in which 0 means a complete lack of cutaneous involvement and 3 represents the most severe possible involvement. To calculate the PRSS, the sum of the severity rating for these three main changes was multiplied with the numeric value (N) of the extent of the disease.

The formula can be written as: $PRSS = N_t (E_t + I_t + S_t) + N_e (E_e + I_e + S_e)$.

The subscript "t" indicates one side of the trunk and the head, and the subscript "e" indicates one side of the extremities. The pruritic symptoms were also assessed with a 0 to 3 scale as follows: 0=absence of pruritus; 1=mild (if it occurred only intermittently and it did not interfere with work or rest), 2=moderate (if it was present for much of the day, but at a more tolerable level) and 3=severe (if it interfered with daytime activities or sleep).

Evaluation of therapeutic efficacy

Before treatment was begun, the distribution and the severity of lesions were assessed and scored as we previously described. During the study, the severity scores (PRSS) were assessed when the patients were treated. The patients were then observed at 1-week intervals until the disease remitted. A patient's condition was defined as clearing if he or she had a PRSS score of 2 or less.

Statistical analysis

The data was analyzed by using paired-t tests. Significance was defined as *p* values <0.05.

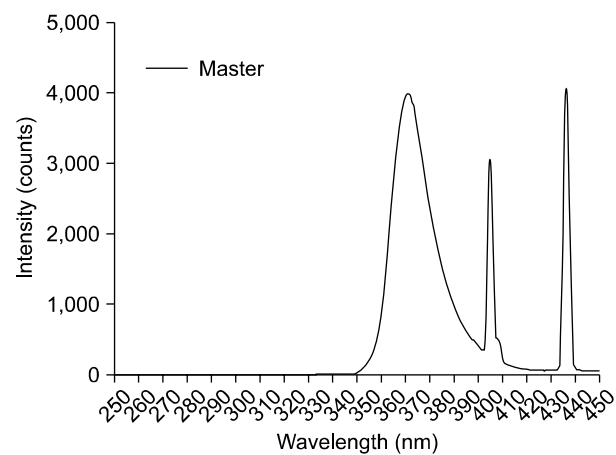


Fig. 1. Spectral emission of the Waldmann TL10R low-pressure lamps (Waldmann GmbH, Schwenningen, Germany), as measured with 2000 USB Fiber Optic Spectrophotometer (Ocean Optics Inc., USA). The Waldmann TL10R low-pressure lamps generate UVA1 wavelengths in the 340~400 nm range with a peak emission at 370 nm.

RESULTS

The mean±SD cumulative UVA1 dose was 132.0±41.8 J/cm², and the mean±SD total number of treatments was 6.5±1.8 (Table 1). The exposures were well tolerated. No significant side-effects were noted during the course of treatment. All the patients required regular use of emollients because of mild skin dryness.

Extent of the disease

The extent of disease (PRSS) in all 15 patients lessened during the study (30.1±3.6 vs. 2.0±1.6, respectively, *p* <0.05) (Table 2). The lesions exposed to UVA1 radiation completely disappeared in 12 of the 15 patients with pityriasis rosea (Fig. 2, 3). The remaining 3 patients showed significant improvement with persistence of less than 25% of the lesions.

Evaluation of the severity score (PRSS) demonstrated at all time points during treatment a great reduction of the disease by the UVA1 irradiation. The overall reduction of the PRSS showed a significant improvement after the second or third treatment (Fig. 4). No relapse was observed over the 3 months of the follow-up period.

Pruritus

The pruritus in 12 of 15 patients lessened during the treatment period, and it was unchanged in the remaining

Table 2. Clinical responses of the patients with pityriasis rosea that was treated with UVA1 phototherapy

Patient No.	Initial PRSS*	Follow-up PRSS	Pruritus score
1	32	2	2→1
2	36	1	2→2
3	28	2	1→0
4	30	6	3→1
5	36	1	2→1
6	32	5	1→1
7	30	Discontinued	Discontinued
8	35	2	3→2
9	30	1	2→1
10	30	2	1→0
11	26	0	1→1
12	28	1	1→0
13	24	2	2→1
14	26	1	1→0
15	28	2	2→0
Mean±SD	30.1±3.6	2.0±1.6	

*Pityriasis rosea severity score (PRSS) = $N_t (E_t + I_t + S_t) + N_e (E_e + I_e + S_e)$. N_t , E_t , I_t and S_t refer to N, E, I and S of one side of the trunk and the head; N_e , E_e , I_e and S_e refer to N, E, I and S of one side of the extremities. The initial PRSS was assessed before treatment began. The follow up PRSS was assessed after UVA1 treatment.

3 patients (Table 2). Yet the overall reduction of the pruritus score showed no significant difference at any time point of treatment.

Relation of the duration of disease to the decrease of the severity score

In our study, the mean duration of disease was 11.2 ± 4.9 day (Table 1). Four of the five patients who had pityriasis rosea for 1 week or less at the onset of therapy noted a greater improvement with UVA1 phototherapy. On the other hand, six of the ten patients who had their disease for more than 1 week showed an improvement with

UVA1 phototherapy. Given the results, it was speculated that the previous duration of disease did not affect the success of phototherapy for producing objective improvement in the severity score (PRSS) and there was no statistically difference between the patients with a longer duration of disease before treatment and the patients with a shorter duration of disease before treatment ($p > 0.1$).

Adverse effects

All patients experienced varying degrees of tanning. Except for one patient who developed PMLE and for whom treatment was discontinued, no other patients



Fig. 2. Clinical features of pityriasis rosea before (A) and after (B) UVA1 phototherapy. The patient was subsequently treated with UVA1 5 times, commencing at 10 J/cm^2 and increasing to a maximum 30 J/cm^2 , and the cumulative dose was 110 J/cm^2 .

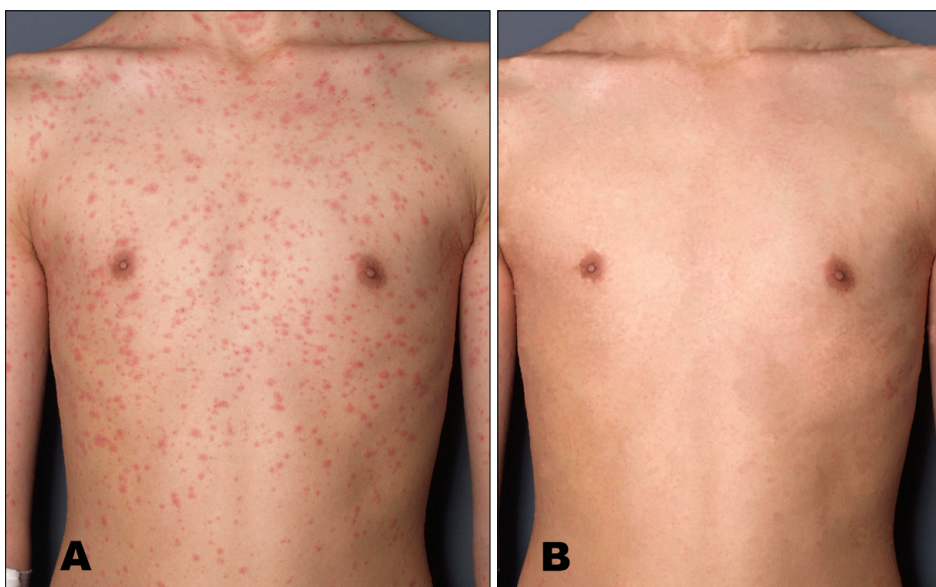


Fig. 3. Clinical features of pityriasis rosea before (A) and after (B) UVA1 phototherapy. The patient was subsequently treated with UVA1 8 times, commencing at 10 J/cm^2 and increasing to a maximum 30 J/cm^2 , and the cumulative dose was 160 J/cm^2 .

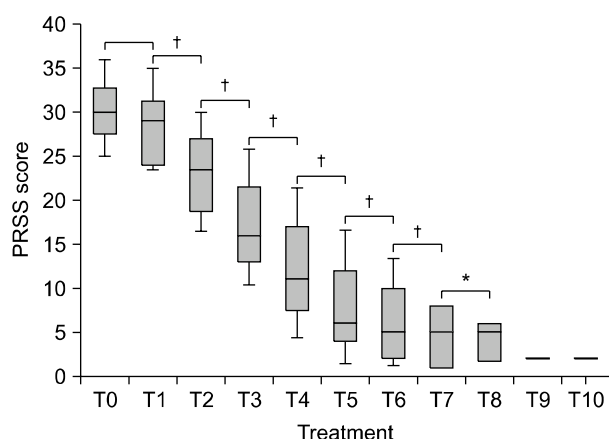


Fig. 4. Pityriasis rosea severity score (PRSS) during UVA1 phototherapy. The data is shown as means \pm SDs. The asterisk indicates a statistically significant difference between the base-line values and the measured values. $n=14$, $*p<0.05$; $^{\dagger}p<0.01$, paired-t test (vs. T0).

developed side effects that warranted discontinuation of UVA1. Adverse effects were found in 5 of patients (Table 1). The adverse effects included erythema (2), pruritus (1), a burning sensation (1) and PMLE (1). But these adverse effects were all improved within 1 month after completing the therapeutic course.

DISCUSSION

Low-wavelength UVA1 (340~400 nm) phototherapy is currently available in only a few dermatology departments. A light source releasing UVA1 was developed in the early 1980s and it was sometimes used as a diagnostic tool for provocation of polymorphic light eruption and for photopatch testing, and as experimental therapy for vitiligo and acne¹². After demonstrating the photophysical properties and photobiologic effects of UVA1, it began to be more widely used for treating various skin diseases. A UVA1 light source delivers more of the deeper penetrating UVA1 wavelengths than the broadband UVA source that's used for PUVA therapy and UVB, so that UVA1 therapy could be expected to have a greater effect on the inflammatory infiltrate in the dermis. Approximately 10% to 40% of UVA1 applied to the skin can penetrate the epidermis and target the CD4+ and CD8+ mononuclear cells infiltrating the epidermis, as well as those CD4+ and CD8+ mononuclear cells in the deep dermal compartments¹³. The possible mechanisms of action include an induction of T-cell apoptosis, reduction of the number of Langerhans cells and mast cells in the dermis, down-regulation of the interferon- γ expression in lesional skin and induction of collagenase and matrix metalloproteinase³⁷.

Using these characteristics and mechanisms, UVA1 phototherapy is now recognized as one of the first-line treatments for several inflammatory dermatoses such as atopic dermatitis, morphea, systemic sclerosis, extragenital lichen sclerosus, chronic sclerodermic graft-versus-host disease, systemic lupus erythematosus, granuloma annulare, sarcoidosis, keloids, idiopathic follicular mucinosis, pityriasis lichenoides and cutaneous T-cell lymphoma¹⁴⁻³⁵. There have been a few reports of successfully treating pityriasis rosea using UV phototherapy³⁶, but there are currently no reports on using UVA1. In this study, we attempt to use low-dose UVA1 phototherapy for pityriasis rosea patients with extensive inflammation and severe itching sensations. As the etiology of pityriasis rosea is still unknown, we can only speculate on the therapeutic effects of UVA1 phototherapy on the disease process. A modern understanding of immunology has also suggested a viral agent or other infectious agents as the cause of PR⁶. At the time of presentation, there is an increased incidence of local mononuclear cells in the deeper perivascular and superficial dermis. The immunohistologic data shows perivascular aggregates of predominantly active CD4+ T-helper cells in the superficial dermis. There is also an increase in Langerhans cells, which may point to an infectious source because of their antigen-processing capability³⁸. The increased number of Langerhans cells and the higher ratio of T-helper to T-suppressor/cytotoxic cells correlate with the stage and severity of the disease³⁹. Several experimental findings indicate that CD4+ T-helper cells are important targets for UVA1. UVA1 can trigger both immediate and delayed pathways for the apoptosis of CD4+ cells, and successful UVA1 phototherapy of atopic dermatitis is associated with the downregulation of the in situ expression of CD4+ cell-derived cytokines, as well as a reduction of CD4+ cells⁴⁰. Therefore, we can hypothesize that UVA1 has a high level of efficacy and tolerability for PR, which is characterized by dermal infiltrates rich in T lymphocytes.

In our study, low-dose UVA1 irradiation was introduced as an innovative therapeutic modality for patients suffering with pityriasis rosea, and excellent results were achieved for 15 patients with pityriasis rosea. The overall reduction of PRSS showed a significant improvement (30.1 ± 3.6 vs. 2.0 ± 1.6 , respectively, $p<0.05$) and the pruritus score of 12 of 15 patients lessened during the treatment period. Even though all the patients experienced varying degrees of tanning during the therapeutic course, the tans were mild. Only one patient developed pruritic papules during treatment that were consistent with PMLE. However, no other patients developed side effects that warranted discontinuation of UVA1. The adverse effects included

erythema, pruritus, a burning sensation and PMLE in 5 patients. But these adverse effects were all improved within 1 month after completing the therapeutic course. Therefore, the present study demonstrates that the application of low-dose UVA1 therapy in patients with PR seems to be an effective, safe and well-tolerated treatment option for PR.

UVA1 may be most beneficial in the early stages of the disease, as we found that the patient with the shortest disease duration had the greatest response. This may be because late-stage disease has less of an inflammatory infiltrate and UVA1 may be acting via T-cell apoptosis and hence, regulation of pro-inflammatory cytokines. In the previous reports, the duration of disease appeared to be related to the success of phototherapy for producing clinical improvement¹¹. Yet in the present study, we could not demonstrate any relation between the duration of disease and the success of phototherapy for producing both clinical and pruritic improvement (PRSS).

The rash of pityriasis rosea typically lasts about 5 weeks and it resolves by 8 weeks, so no specific treatment exists for pityriasis rosea⁵. However, proper treatment is required for the cases with extensive inflammation and severe itching. Topical steroid creams or lotions, dapsone and streptomycin can be used when severe inflammation occurs⁷. Although the exact mechanism of UV phototherapy in this disease is unknown, there have been reports that lesions do not occur in the body areas where UV radiation is utilized. Therefore, UV radiation, though an artificial source or by intentional exposure to natural sunlight, has been recommended to decrease the duration of rash and the intensity of itching in patients with pityriasis⁸⁻¹¹. More specifically, a 1995 study that used the same unilateral approach for 17 patients found initial improvement in the appearance of the lesions, but no change in the itching or the overall patient status (the severity of lesions or itching) on follow-up of two and four weeks after the two-week treatment course³⁶. Arndt et al.¹¹ reported that for 50% of patients, the itching and the presence of a rash decreased when UVB treatment was utilized. In cases where half of the body was exposed to UVA and the remaining half of the body was exposed to UVB, UVB was more effective on the skin lesions and no difference existed with respect to decreasing the itching sensations. The duration of the disease before phototherapy does not affect the outcome. Broadband UVB therapy for PR patients may substantially decrease the severity of the disease, and sometimes it leads to total recovery.

In this study, the post-therapeutic clinical results were satisfactory for both the patients and the physicians,

including a significant reduction of the PRSS. This study was valuable in that UVA1 phototherapy was introduced as an innovative therapeutic modality for patients suffering with pityriasis rosea, and any previous studies on this have been lacking.

As adverse effects of low dose UVA1, hyperpigmentation, erythema, pruritus, a burning sensation, PMLE and recrudescence of herpes simplex infection have been previously reported. But for the patients undergoing phototherapy, they had mild symptoms and the decrease of itching sensation was relatively fast. When considering this, the results of this research can be applied to the treatment of pityriasis rosea patients who have severe symptoms.

In conclusion, the excellent results of the present study should prompt further controlled clinical trials to compare low-dose UVA1 phototherapy with well-established treatment options such as NB-UVB, medium-dose, high-dose UVA1 phototherapy and PUVA. In addition, prospective studies with long-term follow-up are needed to assess the carcinogenic potential of this treatment.

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