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Successful Treatment of Solar Urticaria with UVA1 Hardening in Three Patients

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Summary Statement

Solar urticaria (SU) is an uncommon photodermatosis that presents with sensitivity to ultraviolet (UV) or visible light which triggers urticarial lesions. Treatment of SU is a challenge and usually involves antihistamines and photoprotection as a first line treatment. Phototherapy regimens are recommended if these first line treatments fail to control the disease. Induction of tolerance by phototherapy (UV hardening) has been commonly described as a treatment modality. UVA1 has deeper penetration to the dermis than UVA or NB-UVB; its use in SU has not been previously reported. We present 3 patients with SU who were successfully treated with UVA1.

Key Words: solar urticaria, phototesting, UV hardening, UVA1

Introduction

Solar urticaria (SU) is an uncommon photodermatosis that presents with sensitivity to ultraviolet (UV) or visible light which triggers urticarial lesions. It is an immunoglobulin E (IGE)-mediated disease that is caused by specific wavelengths of light; a patient's action spectra can usually be determined through phototesting.¹

Treatment of SU is a challenge; it usually involves antihistamines and photoprotection as a first line treatment. Phototherapy regimens are recommended if these first line treatments fail to control the disease. Induction of tolerance by phototherapy (UV hardening) has been commonly described as a treatment modality; UVA is the most frequently reported light source utilized, although narrowband UVB (NB-UVB) has also been used with success.^{2,3} UVA1 (340-400 nm), being a long spectrum UVA, has deeper penetration to the dermis than UVA or NB-UVB. We present 3 patients with SU who have undergone successful hardening phototherapy with UVA1.

Case Series

Patient 1

A 49-year-old African American female presented with a 2-year history of SU with the development of urticaria after sun exposure of greater than 15 minutes. Sunscreen, antihistamines, and hydroxychloroquine had little effect. Phototesting with UVA from PUVA booth (National Biological Corp, Twinsburg, Ohio), UVB from a broadband UVB wall unit (Daavlin Distributing Company, Bryan, Ohio), and visible light from a slide projector (Kodak, Rochester, NY) with a water filter failed to induce urticaria immediately after the exposure. At 24 hours, minimal erythema dose (MED) to UVA was normal at greater than 18 J/cm², UVB was normal for Fitzpatrick skin phototype (SPT) V at 96 mJ/cm². All sites exposed to up to 60 min of visible light did not show any erythema at 24 hrs. Although phototesting results were negative, the diagnosis of SU was made based on characteristic clinical presentations. ANA and serum porphyrin tests were negative. The patient had previously completed broadband UVA hardening with no improvement in symptoms. Hardening with UVA1 was initiated at 20 J/cm² with increases of 10 J/cm² up a maximum of 50 J/cm² twice weekly over 6 weeks. With the improvement of her symptoms, treatments were decreased to once weekly during the winter months and maintained at twice weekly for the summer months. Attempts to discontinue treatments resulted in flares. She has since been stable on the maintenance UVA1 regimen as described above for the past 5 years.

Patient 2

A 39-year-old Caucasian female, SPT II, presented with an 8-year history of SU. Phototesting yielded negative minimal urticarial dose (MUD) for UVA, UVB, and VL. At 24 hours, MED of UVA was greater than 18 J/cm², UVB was 72 mJ/cm², and VL was negative; these were all normal values. ANA and serum porphyrin tests were negative. The diagnosis

of SU was made based on the characteristic clinical presentation. The patient had previously failed sunscreen, antihistamines, topical corticosteroids, hardening with broadband UVA with two different protocols, one course of intravenous immunoglobulin (IVIG) (5 daily infusions for a total of 2 grams/kg), and methotrexate. A UVA1 hardening therapy regimen was initiated at 3 times per week starting at 2.5 J/cm² with 2 J/cm² incremental increases to reach 10 J/cm², then 5 J/cm² increases to reach a maximum of 20 J/cm² over a 6-week period with significant improvement in her symptoms. When 20 J/cm² was reached, maintenance therapy was continued twice per week at 20 J/cm² for three weeks and then decreased to once weekly with minimal flaring. Treatment was able to be ceased during the winter months and resumed in the spring with good control of her symptoms.

Patient 3

A 26-year-old Caucasian female with SPT II presented with a two-year history of SU. She exhibited urticarial eruptions after short durations of sun exposure; sunscreen, topical corticosteroids, and antihistamines failed to control her symptoms. Phototesting was resulted in a brisk positive reaction immediately with wheal and flare at MUD of 6 J/cm² for UVA (Figure 1a), 6 mJ/cm² for UVB (Figure 1b), and a normal result for visible light. MED to UVA and UVB, and response to visible light, assessed at 24 hrs, were all normal. ANA was negative, and serum porphyrin was not performed. UVA1 hardening therapy was initiated at 2.5 J/cm² three times per week with increases of 2 J/cm² per session to a maximum goal of 20 J/cm². She was also started concomitantly on prednisone 40 mg daily. She tolerated the treatments until the dose of 16 J/cm² was reached, at which time she was noted to be forming linear urticarial streaks on her legs, developed dizziness, and noticed that her throat started to swell with each ensuing session. Of note, the patient had self-discontinued the prednisone

when she discovered that she was pregnant around this time. The patient shortly thereafter moved out of state and was lost to follow-up.

Discussion

The prevalence of SU is uncommon with one study finding that of all their 280 patients who were diagnosed with photodermatoses over a 7 year period, only 2% of African American patients and 8% of Caucasian patients had SU.⁴ Another study from Singapore found that of 270 patients diagnosed with photodermatoses over a 10-year period, only 7% had SU.⁵ Because of its uncommon presentation, most treatment guidelines are drawn from case reports or small case series.

Action-spectrum hardening involves utilizing the causative wavelength to induce tolerance, while treatment with an inhibitory UV spectrum involves using a therapeutic wavelength that differs from the wavelength causing the SU.⁶ Skin exposed repeatedly to sunlight or artificial light sources every few days builds up tolerance to subsequent irradiation.⁷ This effect may be due to exhaustion of chemical mediators by repeated exposures.⁸ Phototherapy with UVA, PUVA, broadband UVB, and narrowband UVB have all been used to induce a hardening effect.⁶ Systemic psoralen UVA (PUVA) photochemotherapy has been used to control SU with long-lasting results.⁹ Several other studies have reported patients who were sensitive to visible light and UVA and were successfully treated with narrow-band (NB)-UVB.^{2,6} These treatments require numerous sessions and have prolonged duration until tolerance is achieved. Leenutaphong et al proposed that hardening with phototherapy is due to the persistent binding of photoallergens to IgE binding sites after irradiation, thus preventing histamine release.¹⁰ Recently, omalizumab, a humanized anti-IgE antibody, has been successfully used in many cases of SU.^{11,12}

To our knowledge, this is the first case series to report successful hardening of SU with UVA1. Further investigation is needed to elucidate the best protocol for this part of the spectrum and identify which patients are the ideal candidates for hardening with UVA1. Given the limited number of effective treatments for this highly symptomatic disease, hardening with UVA1 can be a valuable option in the management of solar urticaria.

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Figure Legends

Figure 1a and 1b. Patient 3 phototesting sites immediately after the completion of exposure.

(a) UVA site, and (b) UVB site. Note the marked wheal and flare reaction extending to the covered area during testing.



