

Correspondence

Solar urticaria: long-term rash hardening by inhibition-spectrum narrow-band ultraviolet B0

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Idiopathic solar urticaria (SU) constitutes a severe and potentially life-threatening photos induced disease, and usually represents a therapeutic challenge that may not be manageable by antihistamines or sun protection only.¹ Each case of SU presents with sensitivity to individual ultraviolet (UV) light spectra, which trigger urticarial lesions. We present the case of a patient with SU with sensitivity to both visible light and UVA1, who was treated effectively with a narrow-band (NB)-UVB regimen.

A 25-year-old man with Fitzpatrick skin type II presented with a 1-month history of severe generalized SU for months before admission. After a session of sunbathing, he had experienced dizziness, nausea and grade II anaphylaxis-like symptoms. Urticarial lesions had also been provoked indoors by glass-filtered sunlight. Sunscreens with high levels of UVA and UVB protection, oral antihistamines, and steroids had little effect.

Laboratory investigations showed normal results for serum tryptase, porphyrin levels in faeces and for blood cell count, including numbers of erythrocytes and mast cells, routine serum and urine analysis, and levels of C-reactive protein, antinuclear antibodies, double-stranded DNA antibodies, and total serum IgE.

Physical urticaria to heat or cold was excluded by challenge testing. Phototesting produced an immediate urticarial response to both visible light (from a slide projector at 380 W and 300 mm distance for 15 min) and UVA1 [minimal urticarial dose (MUD) = 7 J/cm²; Dermalight; Dr Hönle AG, Martinsried, Germany], which extended beyond the margins of the test area without generalization (Fig. 1a,b).

A treatment method known as action-spectrum hardening using the causative wavelengths has been shown to be effective in UVA-induced SU, but the treatment is time-consuming, risks SU exacerbation, and induces only short-term tolerance that requires

maintenance treatment at close intervals.^{2,3} In addition, our patient reacted to a very broad spectrum of light, making it difficult to define the best wavelength for hardening. Therefore, we used an alternative treatment, starting the patient on total body irradiation with an inhibitory NB-UVB spectrum, which was tolerated well without causing urticarial lesions.

The initiating NB-UVB dose used was 0.03 J/cm², and corresponded to one-third of the dose usually used for phototherapy in patients with skin type II. For subsequent treatments, the NB-UVB dose was increased by approximately 30%. The patient received a total dose of 1.49 J/cm², given as 10 irradiations of 0.32 J/cm² on successive weekdays (Monday to Friday), resulting in a clinical reduction and delay in the urticarial response to UVA1 (MUD = 20 J/cm²). After an additional 10 NB-UVB applications with a dose escalation to 0.52 J/cm² (cumulative dose 7.02 J/cm²), the physiological responses to UVA1 and visible light had completely normalized (Fig. 1c). Neither extensive UVA1 nor visible light exposure after this point could provoke further SU reactions, and UVA1 application only induced immediate pigment darkening at a minimal tanning dose of 20 J/cm². To sustain the achieved hardening effect, the patient was treated with a further 10 irradiations at a final maintenance dose of 0.55 J/cm², reaching a cumulative NB-UVB dose of 12.52 J/cm². During this time, the patient increased his exposure to natural sunlight as well, without experiencing any adverse skin or systemic reactions. The NB-UVB therapy was stopped, and the patient experienced no SU relapse despite continued sunbathing during the summer. The remission lasted for several months, but the SU relapsed the following summer.

This case illustrates the clinical and therapeutic challenge of SU. For diagnosis, it is necessary to assign the causative action spectrum to either UVA, UVB or visible light by phototesting. If sunscreens or systemic therapy with antihistamines are insufficient to control the SU, phototherapy may be considered. Three alternative regimens are possible.

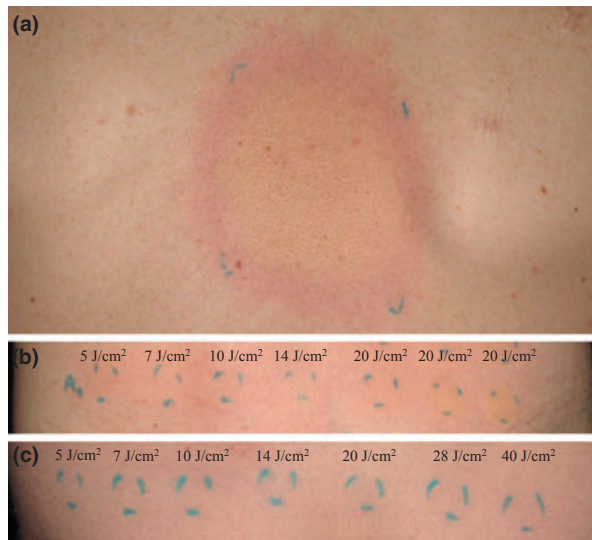


Figure 1 Patient with solar urticaria. Phototesting showed an immediate urticarial response after exposure to (a) visible light for 15 min (from a slide projector at 380 W and 300 mm distance) and (b) ultraviolet (UV)A1 [minimal urticarial dose (MUD) = 7 J/cm²]. The incremental increase in the diagnostic UVA1 irradiation test was stopped at a dose of 20 J/cm² because of the severe urticarial response. (c) Treatment with inhibitory narrowband UVB (311 nm) suppressed the urticarial response to UVA1 (maximum test exposure dose 40 J/cm²).

1 Systemic psoralen UVA (PUVA) therapy. PUVA has been used successfully to control SU.⁴ It is effective but requires special equipment and expertise, and is associated with risk of photodamage. Therefore, we do not consider it as first-line phototherapy for SU.

2 Action-spectrum hardening, using the causative wavelength to induce tolerance. However, this may require long-term maintenance therapy, and the therapeutic remissions may last only a few days and be followed by SU rebound. In our case, the patient's reaction to a broad spectrum from UVA to visible light made it difficult to define the wavelength for hardening.

3 Treatment with an inhibitory UV spectrum. In this technique, the therapeutic wavelength differs from the causative action spectra; accordingly, NB-UVB may be used for the treatment of SU induced by UVA1 or visible light, whereas SU triggered by UVB or UVA2

might be best treated with UVA1. The inhibition spectrum may be identified by phototesting or by increasing exposure to a known nonurticarial wavelength. MUD testing in our patient showed that UVB was well tolerated, therefore, he was exposed to increasing doses of NB-UVB, which proved to be effective. This therapeutic effect has been reported in case reports⁵ and in a larger cohort of patients with SU provoked by UVA and visible light.⁶ The protection may be sustained by UVA-selective sunblocks that protect from the SU action spectrum but allow continuous exposure to the inhibitory UVB wavelengths.

In conclusion, idiopathic SU is a disease with individual action and inhibition spectra that may be identified by phototesting. Use of inhibition spectra to treat SU treatments has several advantages, which include a low total number of necessary irradiations, prolonged remission and the widespread availability of NB-UVB devices. Thus, NB-UVB therapy should be considered the first-line treatment in cases of SU sensitive to UVA or visible light, as illustrated by our case.

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Conflict of interest: none declared.

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