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Solar urticaria: Clinic, diagnostic, course and therapy management in 27 patients

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Summary

Background and objectives: Solar urticaria is a rare photodermatosis, the diagnosis and therapy of which have not yet been standardized. The aim of this research was to use innovative radiation sources for diagnostics with defined and reproducible emission spectra and doses. A uniform therapy step scheme was to be created.

Patients and methods: In a longitudinal study, 27 patients with solar urticaria were examined over 13 years. With a characteristic anamnesis, the diagnosis was confirmed with phototesting (photoprovocation) from various radiation sources (UVB, UVB-311nm, UVA, UVA-1, green light, red light) and a therapy step scheme was designed consisting of light protection, antihistamines, rush hardening with UVA-1, and administration of omalizumab.

Results: Action spectrum: UVB 44 %, UVA 70 %, UVA-1 89 %, green light 37 % and red light 22 %. Rush hardening with subsequent maintenance therapy was performed on 20 patients, 17 of whom were hereby adequately protected. In three further patients, omalizumab was additionally administered.

Conclusions: Phototesting with UVB, UVB311nm, UVA, UVA-1, and visible light with innovative radiation sources is uniformly possible in every major skin clinic. With the help of the therapy step scheme the patients can be adjusted well. Rush hardening with UVA-1 is a safe method to help the patients during the sunny season. Omalizumab as the last therapy option is effective, but currently only possible in off-label use.

Introduction

Solar urticaria belongs to the idiopathic photodermatoses. It is a rare skin disease triggered by ultraviolet radiation (UVA and/or UVB) and/or visible light [1].

The skin reactions appear within a few minutes of sun exposure. After avoidance of further sun exposure, they resolve without residuals after approximately 30–60 minutes, more rarely after 1–5 hours, depending on the intensity of the stimulus [2–4]. The symptoms start with pronounced pruritus in the area of sun-exposed skin, followed by erythema and wheal formation of varying degree depending on the dosage. More severe cases with headache, vertigo, nausea, bronchospasm, arterial hypertension, and tachycardia even to anaphylactic shock have also been described, especially if large skin areas have been exposed to the provoking light spectrum [3–8]. Duration and intensity of radiation required for provoking a skin reaction differ between individuals.

The exact pathogenesis and etiology of solar urticaria is still unclear. Currently, an immediate-type allergic reaction (type I according to Coombs and Gell) is assumed. According to this hypothesis, incident light is absorbed by a precursor molecule (chromophore) in the dermis resulting in activation of the chromophore and formation of an immunologically active photoallergen. Specific IgE directed against the photoallergen appears on the surface of mast cells of the skin. Attachment of the photoallergen to the FcεRI receptor of IgE results in an antigen-antibody reaction with subsequent mast cell degranulation. The released mediators cause the clinical symptoms of solar urticaria with wheal formation, erythema, and pruritus [1, 9, 10].

The diagnosis of solar urticaria is based on a typical history. It is confirmed by photoprovocation used to identify

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the action spectrum and to determine the minimum urticaria dose (MUD) for therapeutic interventions and further monitoring. Currently, no uniform, generally accepted protocol for photoprovocation exists. In a few, highly specialized centers, monochromators are used, which are very expensive and not available in most dermatology clinics.

Accordingly, it was one aim of this study to perform a standardized photodiagnostic workup with radiation sources available in any clinic with a photo department and covering a wide range of wavelengths. There is no established evidence-based and approved therapy for solar urticaria. The following therapies have been described in the literature based on individual cases or small case series:

While sunscreen products with high sun protection factor are recommended in all approaches, they are usually not sufficient [11–28]. In addition, beta-carotene [11], antihistamines (H1 blockers) [12–15], H2 blockers, for example cimetidine [16], extracorporeal photochemotherapy (photopheresis) [17], plasmapheresis [18–20], cyclosporin A [22], IVIG (intravenous immunoglobulin) therapy [22], and anti-IgE: omalizumab [23–30] have been described in small case series or individual cases.

The following types of phototherapy have been presented most often as effective: systemic PUVA therapy, if necessary in combination with systemic glucocorticoids [31], irradiation with UVA [32–36], irradiation with broadband or narrowband UVB [36–38].

Based on the existing literature and our own experience, we designed an escalation therapy scheme and applied it to our patients (Figure 1).

Patient collective/Material and Methods

In the present longitudinal study, we examined 27 patients with solar urticaria presenting at our dermatology department during 2002–2015.

After a detailed medical history, all patients were treated by photoprovocations of areas not exposed to the sun (back and buttocks). These were performed with the Saalmann Multitester for incremental doses of UVA/UVB light (Saalmann GmbH Medizintechnik, Herford, Germany), the UVA-1 metal-halide radiator (Sellas medizinische Geräte GmbH, Ennepetal, Germany), the UVB-311 radiator (Waldmann Medizintechnik, Villingen-Schwenningen, Germany), the PhotoCure lamp for red light (CURElight, CE 0470, Version:1.8, PhotoCure ASA, Oslo, Norway), and the Saalmann PDT-FDAP complex (Saalmann GmbH Medizintechnik, Herford, Germany) for green light. The Saalmann Multitester has an attachment for incremental doses of UVA and UVB light. Lead rubber templates were used as cover for the other phototests with UVA-1, UVB-311, red light and green light (Figure 2).

The aim of the phototesting was to confirm the diagnosis, classify the action spectrum, and determine the minimum urticaria dose (MUD) which is the threshold dose of the particular light source at which a wheal formation is triggered.

The PhotoCure lamp emits red light with a spectrum from 570–670 nm. At a distance of 15 cm to the skin, the measured intensity was 90 mW/cm^2. Given that irradiation duration and intensity as well as dose can be reproduced exactly, irradiation can be repeated at any time under identical conditions.

The Saalmann PDT-FDAP complex emits green light with a spectrum from 543–550 nm. At a distance of 25 cm (between light source and skin area to be irradiated), the measured irradiation intensity was 12 mW/cm^2.

Depending on disease severity, we performed an escalation therapy according to the following step scheme: first sunscreen, then oral administration of antihistamines up to the highest dose; in case of non-response, first a UV rush

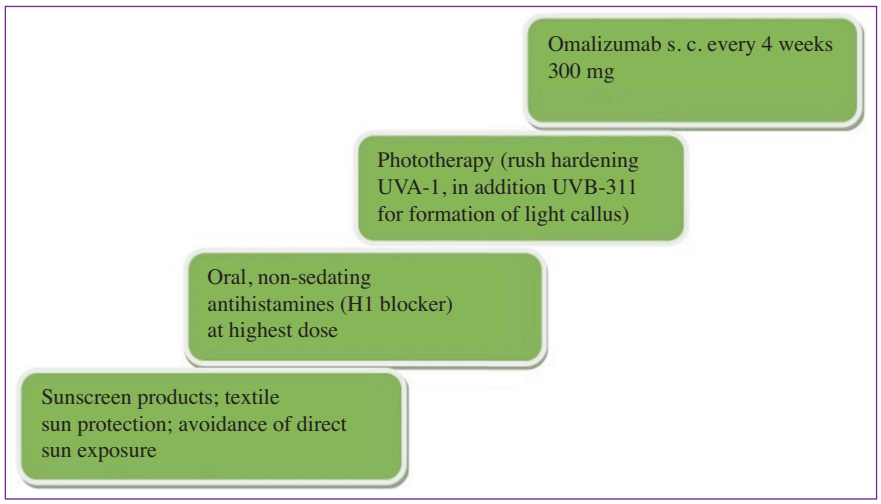

Figure 1 Four-step escalation therapy.

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Figure 2 Photoprovocation on the back of a solar urticaria patient. Saalmann PDT-FDAP complex (left site: green light) and PhotoCure Lamp (right site: red light).

hardening, followed by maintenance therapy. If there was still no response, this was followed by an additional systemic therapy with omalizumab (Figure 1). The respective escalation steps were initiated if the previous interventions showed insufficient efficacy.

In 18 patients, we used the UVA-1 metal-halide radiator (high-intensity device: blue filter, UVA edge filter, infrared-absorbing glass; type 24000, Sellas medizinische Geräte GmbH, Dr. Sellmeier, Ennepetal, Germany) with a UVB content of 0 %, thus excluding the risk of solar dermatitis (emission spectrum 340–420 nm). For practical and organizational reasons, the remaining two patients received rush hardening with a conventional UVA device (801 K, emission spectrum 315–400 nm, Waldmann Medizintechnik, Villingen-Schwenningen, Germany).

Once the minimum urticaria dose (MUD) had been determined by the photoprovocations mentioned above, we started irradiation with approximately 50 % of the MUD using a quadrant system (UVA or UVA-1) (Figure 3). The time interval of one hour between two irradiations recommended by Beissert et al. [33] was shortened to 15 minutes to achieve a more dynamic hardening. This shortening of the interval was tolerated well by the patients.

If no or only minor skin reactions were observed after 15 minutes, we continued with irradiation per quadrant with the same dose until all quadrants had been treated once. Then, the dose was increased by 30 %. In case of erythema, we first irradiated with the same dose in the next quadrant, once the erythema had subsided. Only if no further erythema could be induced was the dose was increased by 30 %. If an urticarial reaction was observed, we only irradiated with the same dose in the next quadrant once the urticarial reaction had subsided. When all body parts were irradiated with the same dose and urticarial reactions were still present, the dose

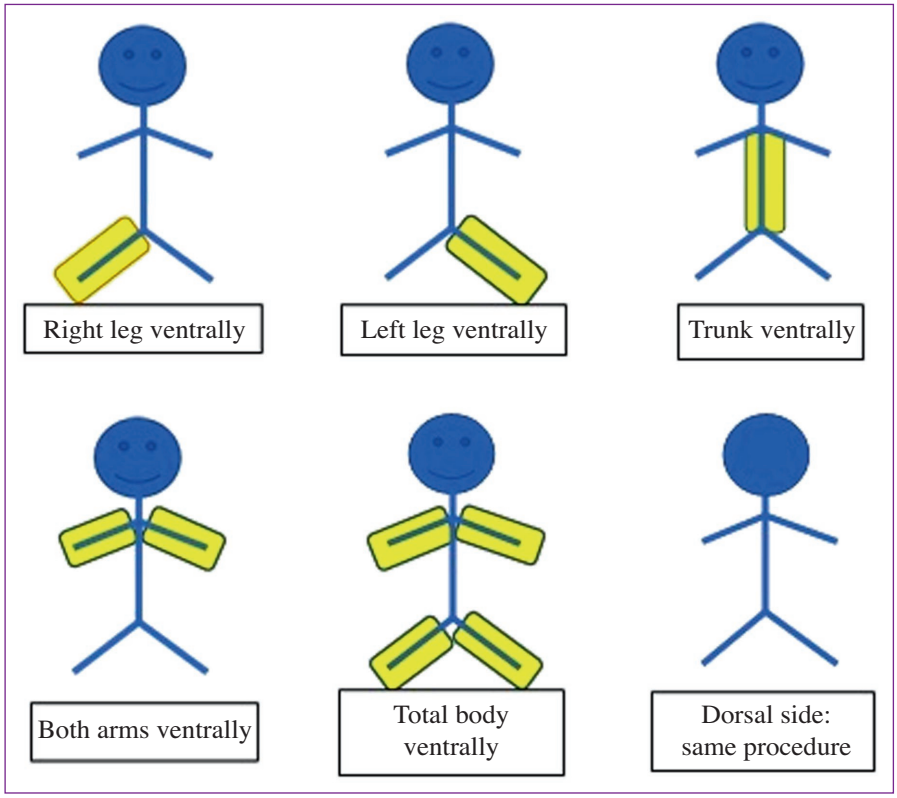

Figure 3 Rush hardening (scheme).

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was reduced by 30 % and the process was continued as mentioned above. Face and neck were also irradiated. If an overnight interval lay between the irradiations, we started with the last well-tolerated dose from the previous day.

In 16 patients, additional irradiations with UVB-311 (type UV 7001, Waldmann Medizintechnik) were performed to boost the protective effect. At first, these irradiations were performed every day. After achieving remission, the frequency was reduced to three times and, if possible, two times per week. The chosen doses corresponded to the recommendations of the currently valid guidelines for phototherapy with UVB light [39, 40].

Results

Overall, 18 female and 9 male patients aged 8 to 62 years were examined and evaluated. The average age was 41 years at initial diagnosis and 35 years at initial manifestation. Wheals developed within a few seconds to 20 minutes after sun exposure, even behind window glass and under thin clothing. After termination of sun exposure, wheals persisted for 20 minutes to a few hours. In the 27 patients, initial manifestation of the disease had occurred 4 weeks to 34 years previously.

In 24 patients, we analyzed the time between first manifestation and first presentation with diagnosis (Figure 4). For nine patients it was below one year, for 10 patients 1–5 years, for three patients 6–10 years, and for two patients 30 and 34 years, respectively. For three patients, no information was available. It thus took 4.8 years on average from initial manifestation to initial diagnosis.

The 27 patients received incremental doses of UVA and UVB light as well as photoprovocations with the Saalmann PDT-FDAP complex (green light), the PhotoCure lamp (red light), and the UVA-1 metal-halide radiator on areas usually not exposed to the sun, such as buttocks and/or back. The minimum urticaria dose (MUD) was at a typical, very low level in all patients. For UVA at 0.4–6.3 J/cm², for UVA-1 at 1–10 J/cm², for UVB at 17–40 J/cm², for red light at 1–75 J/cm², and for green light at 0.18–5.04 J/cm².

89 % of the patients showed an action spectrum with UVA-1, 70 % with UVA, 44 % with UVB, 37 % with green light, and 22 % with red light (Figure 5). In all patients, the action spectrum covered several ranges: 48 % of the patients were simultaneously sensitive to UVA and UVA-1 light sources, 30 % to UVB, UVA, UVA-1, and visible light, 11 % to UVB, UVA, and UVA-1, 7 % to UVA, UVA-1, and visible light, and 4 % to UVB and visible light.

The therapeutic step scheme that we designed for escalation therapy was used on 23 patients: three patients achieved good results with only antihistamines and UV-protection. Four patients did not consult us again. Twenty patients were treated with rush hardening by UVA-1 or UVA radiation. 16 of these 20 patients were treated additionally with UVB-311 irradiation to boost the protective efficacy. In three of these 16 patients, this protection was insufficient. Therefore, they additionally received omalizumab 300 mg s. c. every four weeks in off-label use.

Two of these three patients discontinued phototherapy and remained in remission. One patient required a combination of antihistamines, phototherapy, and omalizumab for adequate protection. She was the most light-sensitive patient of our collective.

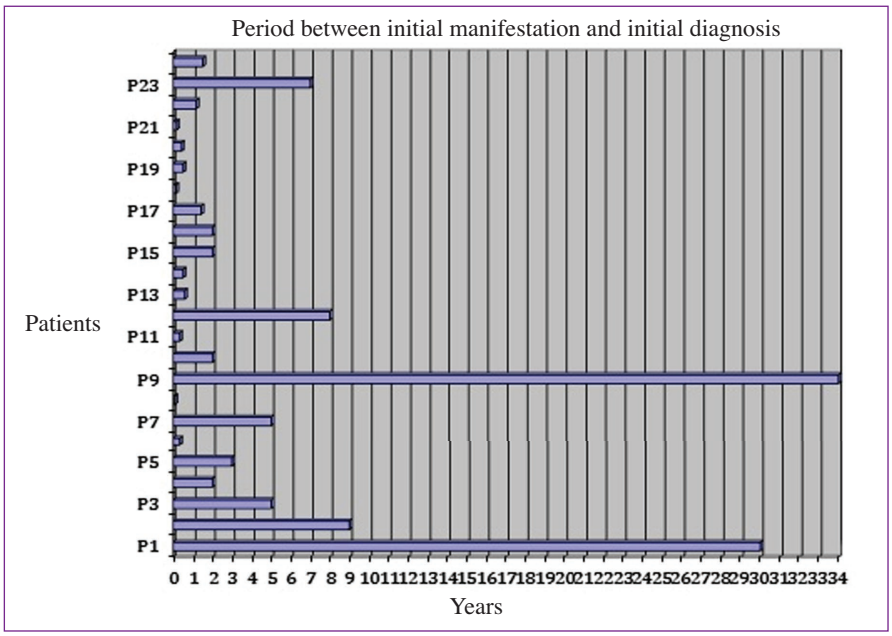

Figure 4 Period between initial manifestation and initial presentation with diagnosis.

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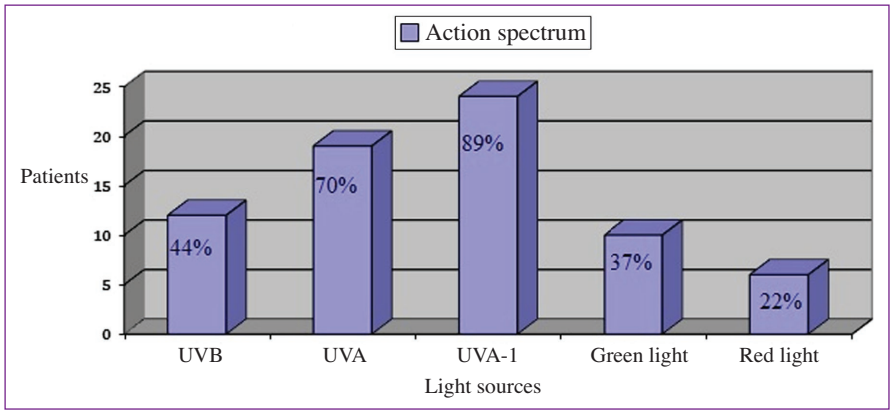

Figure 5 Action spectrum of the 27 patients.

19 of the 20 patients were hospitalized for treatment initiation. The duration of in-patient rush hardening ranged from 2–18 days. Twelve patients (63 %) were hospitalized for up to five days. All patients tolerated the shortened intervals between irradiations, down to 15 minutes compared to the original description [33], very well. After successful rush hardening, the patients received maintenance therapy during the sunny period from April to October. Maintenance therapy involved daily irradiation for one week, which was subsequently reduced to three times per week. If sufficiently effective, an attempt was made to reduce the irradiation frequency to two times per week.

The indicator for a successful therapy is the increase of the minimum urticaria dose (MUD) or the final tolerated dose. All 20 patients treated with rush hardening showed a marked increase of MUD or tolerated dose after therapy (Figure 6).

With the exception of patients 2 and 3, who were treated with UVA instead of UVA-1, the MUDs or tolerated doses of all patients ranged from 10 to 90 J/cm² UVA-1 after therapy. In six patients, the tolerated dose after therapy ranged from 60 to 90 J/cm², in ten patients from 30 to 50 J/cm², in two patients from 10 to 20 J/cm², and in two patients (P2 and P3) it was below 10 J/cm² (Figure 6). Accordingly, all patients reported that they were able to experience the sunny months significantly better and almost without restrictions.

Discussion

Solar urticaria is a rare photodermatosis causing tremendous psychological strain with major restrictions of the quality of life in affected patients. In case of anaphylactic shock, solar urticaria can be life-threatening.

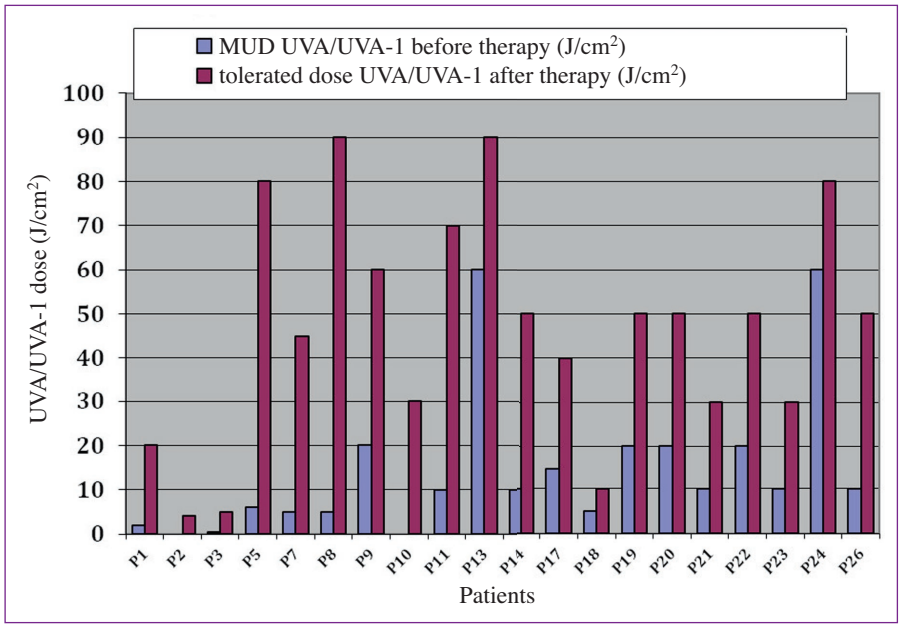

Figure 6 MUD (minimum wheal/urticaria dose) UVA/UVA-1 before therapy and tolerated dose of UVA/UVA-1 after therapy (MUD UVA only for P2 und P3).

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The age of manifestation of solar urticaria varies, with increased occurrence in young adulthood [3–8, 41–44]. In our patient group, the age of manifestation also varied greatly (8–62 years). In patients 1 to 26, the median age of initial manifestation was 35 years. This is in good agreement with major case series from several countries published in recent years [3–8, 41–44]. Similar to our study, the gender distribution in the recently published major case series showed that female patients are predominantly affected: Beattie et al. [4] 61 vs. 26, Du-Than et al. [5] 43 vs. 18, Haylett et al. [6] 100 vs. 45, Perez-Ferriola et al. [7] 141 vs. 83.

In our study, nine patients were male (33 %) and 18 patients were female (67 %). Only in the studies of Uetsu et al. [43] and Monfrecola et al. [3] was there a majority of male patients.

In our case series, the time between initial manifestation and final diagnosis ranged from a few weeks to 34 years in 24 patients. While some patients consulted their physician only at a late stage due to tolerable symptoms, in the majority of cases the diagnosis was made very late due to the rarity of the disease.

Diagnosis of solar urticaria is based on the typical medical history and subsequent light tests. By means of irradiation devices emitting UVA, UVA-1, UVB, red or green light, it was possible to confirm the diagnosis in all patients, classify the action spectrum, and determine the minimum urticaria dose (MUD). This is important for further monitoring.

To date, measurements with visible light for classifying the action spectrum of the respective solar urticaria patient were usually performed with slide projectors (for example, Kodak Carousel, Philips) without standardized measurements and specifications on spectrum, irradiation intensity, and dosage. On the other hand, monochromators are very expensive instruments, and given the low number of patients, their purchase is out of the question for most dermatology clinics. In this study, defined red-light and green-light devices originally developed for photodynamic therapy (PDT) were used for photoprovocation with visible light.

Given the precise knowledge of emission spectra and irradiation intensities of the utilized radiation sources, the test results are exactly reproducible. Accordingly, it is possible to compare data between patients as well as disease courses of individual patients. To date, no comparable diagnostic technology with the mentioned devices has been described.

We determined the provoking action spectrum in each patient. As described in the literature, it varied between individuals. In summary, UVA-1 was the most common provoking action spectrum, followed by UVA, UVB, green light, and finally red light. All patients were sensitive to several wavelength ranges.

Botto and Warshaw [41] compared six different case studies with solar urticaria patients from different nations (2 x Scotland, Italy, Japan, Belgium, Singapore). This study also showed varying action spectra, albeit with different distribution. In their study, visible light predominated in most cases (110 patients [65 %], in our study 33 %), followed by UVA (49 patients [26 %], in our study 70 %), and finally UVB (9 patients [5 %], in our study 44 %). We do not know the reason for this large difference in the distribution of action spectra between the study of Botto and Warshaw and the results from our patient collective.

Horio [41] hypothesized that different characteristics of the chromophores affect the various action spectra. Ethnical and geographical differences might also play a role. Common to all major case series is the broad distribution of action spectra from UVB to visible light, in each case with different weighting of the dominant spectra [3–8, 40–43].

While previous therapeutic approaches for light hardening with PUVA [31, 36] or UVA therapy [32–35] were often successful, inducing tolerance was very time consuming.

In 2000, Beissert and Ständer [33] described the rush hardening procedure with UVA-1 irradiation for initiation of phototherapy.

In our case series, we used an escalation therapy consisting of 4 steps for 20 patients. Application of sunscreens had already been attempted in these patients with insufficient efficacy. In accordance with current guidelines on treatment of chronic spontaneous urticaria, four patients received antihistamines (cetirizine dihydrochloride 10 mg or desloratadine 5 mg) at the highest dose of 4 x/day in addition to phototherapy.

In the present study, we used the UVA-1 metal-halide radiator to perform the rush hardening procedure (only in 2 patients was a conventional UVA device used). In 19 patients, we were able to achieve a tolerable dose of 10–80 J/cm² UVA-1 and 4–5 J/cm² UVA (in 2 patients), respectively, within 2 to 18 days (14 patients within 7 days, 5 patients between 11 and 18 days) under controlled inpatient conditions. One patient received treatment in an outpatient clinic only, to accommodate her needs.

The patients showed transient reactions in the individual quadrants consisting of pruritus, erythema, and/or wheals that were, however, well tolerated. Interestingly, two patients undergoing the rush hardening procedure with UVA instead of UVA-1 had to be hospitalized significantly longer (11 and 18 days) than other patients. Compared to UVA, treatment with UVA-1 was more effective. However, due to the low number of patients treated with UVA (n = 2), this finding cannot be generalized. Furthermore, these patients in particular exhibited very pronounced photosensitivity with low MUD.

The effectiveness of rush hardening with consecutive maintenance therapy led to a significant improvement in symptoms in all but three patients. Consequently, they

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experienced markedly fewer, if any, restrictions in their leisure time and at work. This therapeutic success is also reflected in the MUD before and after therapy (Figure 6). In all patients, continuation of radiation therapy in an outpatient setting was required.

In accordance with the currently valid guidelines [39], we performed additional UVB-311 irradiations of the entire integument approximately once daily for four weeks to boost the efficacy in 16 patients. Once remission was achieved, the frequency was reduced first to three times per week and later to two times per week. One reason for the enhanced protective effect is the induction of a light callous caused by stimulation of the mitotic activity including hyperproliferation of the epidermis and thickening of the horny layer [37]. Moreover, UVB-311 nm irradiation has immunomodulatory effects and results in tanning of the skin due to enhanced melanin synthesis, thus reducing the effect of light on dermal mast cells. The additional protective effect of UVB-311 nm is also known from other photodermatoses triggered by UVA and has also been described in aquagenic pruritus [36–38, 40].

Phototherapy was insufficient in three patients. These patients therefore received additional treatment with the anti-immunoglobulin E omalizumab (300 mg s. c. every four weeks).

This therapy was administered during the sunny months from April to October and terminated in late autumn (November). For practical reasons, two of the three patients discontinued phototherapy. They were sufficiently protected by omalizumab without phototherapy. In the most light-sensitive patient, the combination with phototherapy had to be continued to maintain adequate protection.

Omalizumab is a recombinant human monoclonal antibody against IgE approved for refractory, chronic spontaneous urticaria. It is currently not approved for solar urticaria. Binding to free IgE occurs at the same site involved in interaction with its receptor (Fc&RI) on mast cells, thereby reducing free IgE [24, 45]. An explanation for the effectiveness of omalizumab in solar urticaria might be that in solar urticaria the incident light activates the chromophore to generate an effective photoallergen, which in turn binds to IgE at the Fc&RI of mast cells causing the release of mediators that result in clinical symptoms. Güzelbey et al. [24], Brüning et al. [25], as well as Baliu-Piqué and Aguilera Peiró [26] illustrate in a small number of cases the successful therapy of solar urticaria with omalizumab (150 mg, 300 mg, or 400 mg s. c. every 4 weeks) in highly therapy-resistant patients. In contrast, Duchini [24] (omalizumab 150 mg for 4 months) and Müller [27] (omalizumab 150 mg for 3 months) have reported on the ineffectiveness of omalizumab in a small number of patients.

While solar urticaria is a chronic disease, only very few publications on long-term monitoring exist [44, 46]. Spontaneous resolution is possible and likely, although the time frame until complete resolution of the disease is very heterogeneous and unpredictable. Beattie et al. [4] described long-term monitoring of up to ten years in 87 patients. The majority of these patients still had symptoms after ten years. Similarly, in the present study conducted over several years, we could not observe any spontaneous remission in our patients.

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