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Case report

Fixed solar urticaria to visible light successfully treated with fexofenadine

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Fixed solar urticaria (FSU) represents an uncommon form of urticaria related mostly to radiation from the ultraviolet (UVB, UVA) and visible spectrum. The exact pathomechanism has so far remained unknown. A 52-year-old woman with a 3-year history of urticated eruptions limited to certain skin areas is presented. Photobiological testing revealed positive reactions limited to the visible light range. The induced lesions appeared only in originally affected skin sites. The particular distribution of whealing supports the concept of specific alteration of mast cells in well defined areas. The clinical findings and the results of phototesting lead to the diagnosis of FSU to visible light. It is recommended to carry out phototesting in patients with FSU in the originally affected skin areas, usually covered and protected by the patient, to avoid false-negative results. Fexofenadine given in the conventional dosage can prevent recurrences and represents a successful treatment measure when dealing with this peculiar form of solar urticaria.

Key words: **fixed solar urticaria; visible light range; fexofenadine.**

Solar urticaria is characterized by itching, erythema and whealing occurring immediately after exposure to radiation from the ultraviolet (UVB, UVA) and visible light (1). Although its exact mechanism remains unknown, evidence supports an immunological pathogenesis (2). We report on a patient with localized solar urticaria in whom phototesting with a slide projector provoked whealing strictly localized to the skin areas usually affected. No urticariation could be induced by means of exposure testing with wavelengths of the UV spectrum.

Case report

A 52-year-old woman presented with Fitzpatrick skin type III and a 3-year history of itchy, urticated eruptions appearing regularly a few minutes after direct sun exposure. Skin lesions were located on the lower lateral neck, the lower third of the right arm (contralaterally no lesions), the upper third of the left forearm (contralaterally no lesions), the upper back, and the scalp. The eruptions faded within 30–60 min after cessation of irradiation. The patient experienced tachyphylaxia associated with 1-day tolerance right after urtication. All urticated lesions appeared throughout the year including winter. Filtering of sunlight through window glass or light clothing did not prevent urtication. There was no family history of photosensitivity or record of systemic medication. However, the patient reported to have suffered from infant eczema with recurrences between the age of 20 and 25 years. Physical and laboratory examinations, including complete blood cell count, liver function tests, serum urea and total serum IgE levels, yielded normal results. Antinuclear antibodies, antibodies against single-stranded DNA, double-stranded DNA, Sm, Ro, and La, were not detectable. Total porphyrin levels in plasma and in a 24-h urine specimen were normal. The patient was submitted to photobiological testing carried out with polychromatic irradiation (1000 W Xenon light, Dermolum UM-W®, Müller, Moosinning, Germany) filtered with a Schott WG 305 filter, and a high pressure metal halide UVA lamp (2000 W, Sunlab®). The intensity measured with the Centra UV®; meter (Osram, Munich, Germany) was 3.0 mW/cm^2 (in the UVB range) and 40 mW/cm^2 (in the UVA range). The MED (minimal erythematous dose) with the polychromatic irradiation was 33 mJ/cm^2, which is normal for phototype III. There were no immediate urticarial reactions after the polychromatic phototesting and after UVA (13 J/cm^2) phototesting. Thus, a sensitivity in the UVB and UVA spectrum could be excluded. Ten minutes after polychromatic and UVA phototesting, with exposure to normal daylight passing through window glass, the patient developed urticated lesions on her back excluding the skin areas previously exposed to UVB and UVA ir-

radiation. The reaction patterns were suspected to be solar urticaria to visible light. Further photobiological testing was performed including the visible light spectrum. The latter was carried out by conducting phototesting with a standard slide projector containing a halogen lamp (Tungsten Halogen Lamp TF3550®). The emission spectrum ranged from 400 to 800 nm with a maximum wavelength of 775 nm, and a major emission between 620 nm and 800 nm. The tested skin areas (back, upper extremities) were positioned at a distance of 150 cm. The maximum exposure time was 20 min. This series of phototesting was performed in a completely dark room to avoid any visible-light exposure. Both polychromatic phototesting and UVA phototesting did not reveal any whealing or skin reactions during immediate examination after testing, and during examination 30 min after testing as well as during the period between both readings. Phototesting conducted with the slide projector induced whealing after 5 min on the upper back (Fig. 1). This was considered to represent the patient's minimal urticaria dose (MUD).

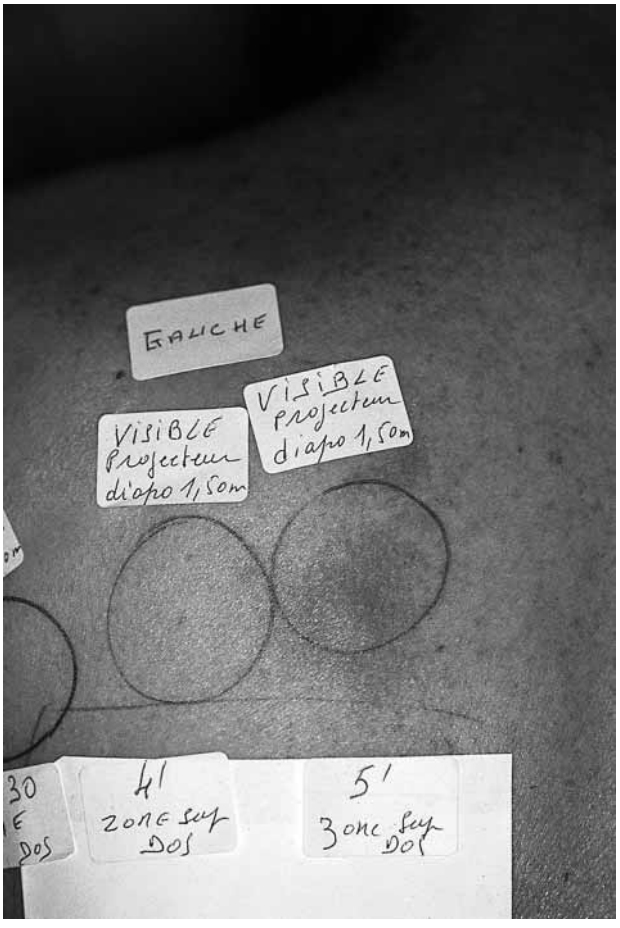

Fig. 1. Whealing induced by phototesting with a slide projector on the upper back after 5 min exposure. There was no reaction after 4 min exposure.

Eruptions appeared only on the patient's originally affected skin areas: upper back, upper third of the left forearm (contralaterally no lesions, also when exposed to four MUDs), and lower third of the right arm (contralaterally no lesions, also when exposed to four MUDs). Due to the results of phototesting we made the diagnosis of FSU solely to visible light spectrum. The patient was treated successfully with sun protection measures and fexofenadine, active metabolite of Terfenadine (Telfast® 180 mg once per day). After stopping the antihistamine treatment, 24 h later the patient developed urticarial eruptions in the affected skin. These eruptions disappeared when fexofenadine was taken again. Since then, the patient has followed sun protection measures and a fexofenadine regimen of 90 mg per day. So far, she has not had any recurrences.

Discussion

Fixed solar urticaria (FSU) represents an uncommon disorder. It was first observed by Reinauer et al. in 1993 who detected urticarial eruptions in three patients which appeared shortly after exposure to sunlight (1). In those patients, whealing remained localized to certain skin areas and could be reproduced in the same sites with similar morphology and distribution pattern. Solar urticaria (SU) induced solely by visible light has been previously described but represents a rare form (2). Our patient demonstrated symptoms of FSU induced by visible light. Such clinical reaction patterns are highly unusual. Recently, Patel et al. reported a case of FSU in the UVB range which progressed to polymorphic light eruption (PLE), suggesting that a common inducing photoallergen is reponsible for this particular type of SU (3). The investigation of SU is usually performed by phototesting in the UVA and UVB range in an area not chronically exposed to sunlight. The usual way of performing phototests with visible light in solar urticaria is by using a xenon lamp in combination with a monochromator, which is time consuming. Visible phototesting can be also carried out with a slide projector or with lasers, which represent high-intensity sources of monochromatic irradiation. Such laser phototesting might be useful to determine the exact action spectrum. The reproduction of urticarial eruptions strictly limited to certain skin sites has been explained by mast cell alterations in well defined areas shown by electron microscopy performed in affected and unaffected skin (4). Reinauer et al. found that intradermal injection of the patient's plasma activated by *in vitro* irradiation induced wheals only in the affected skin sites in two patients, showing that a local factor exists (1). Thus, phototesting in FSU patients should focus on affected skin areas to avoid false-negative results. The lack of severe symptoms

and systemic anaphylactic reactions in patients suffering from FSU is related to a decreased release of histamine due to localized small areas of the skin in contrast to generalized SU. Treatment of FSU comprises antihistamines, classically terfenadine (5, 6), and light hardening by phototherapy, photochemotherapy (PUVA), or repeated exposures to natural sunlight as well as plasmapheresis. Our patient was treated successfully with fexofenadine, first 180 mg per day, and in the later course of the disease 90 mg per day. Despite the well-known efficacy of terfenadine in solar urticaria, there has been no previous report concerning the efficacy of fexofenadine in solar urticaria. Fexofenadine appears to be safer than terfenadine, in particular in higher dosages (6).

In conclusion, FSU represents a less severe form of SU. Its frequency seems to be underestimated due to the lack of severe clinical symptoms and because it has rarely been reported. Fexofenadine is considered to be efficient in the treatment and the disease control of FSU to the visible light spectrum. Furthermore, photobiological testing should focus on the originally affected skin areas, usually covered and protected by the patient, to avoid false-negative results.

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