

Photosensitive Psoriasis Vulgaris Inducible by a Single Suberythematous Dose of Ultraviolet B Irradiation

Timo Buhl, Ullrich Krüger, Steffen Emmert, Hans Peter Bertsch and Rotraut Mössner

Department of Dermatology and Venereology, University of Göttingen, von-Siebold-Str. 3, 37075 Göttingen, Germany. E-mail: buhl@med.uni-goettingen.de
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Sir,

Although ultraviolet (UV) irradiation is a standard treatment for psoriasis, it can lead to an exacerbation of psoriatic skin disease in some patients. Psoriatic lesions can be triggered by both UVA and UVB irradiation; in general doses above the minimal erythema dose (MED) are required to trigger photosensitive psoriasis. We report here a patient with photosensitive psoriasis (PP) in whom new psoriatic lesions could be induced by a single suberythematous dose of UVB irradiation.

CASE REPORT

A 67-year-old Caucasian woman with no history of light dermatosis presented with chronic stationary plaque psoriasis and with a suberythrodermic skin condition in formerly unaffected areas. Prior to admission to our clinic, skin lesions had exacerbated under several sessions of therapeutic UVB irradiation. Routine blood parameters showed 13.2/nl leukocytes (normal 4.0–11.0/nl) with an isolated increase in "bands" (juvenile neutrophilic granulocytes) to 16% ($\leq 8\%$), CRP 56.4 mg/l (≤ 8 mg/l), ESR 23 mm/h. All other blood parameters were within normal limits. Skin biopsies and serological findings, including auto-antibody testing, ruled out autoimmune disorders such as subacute lupus erythematosus. Phototesting revealed an MED of 100 mJ/cm² (normal ≥ 25 mJ/cm²) for UVB and 20 J/cm² (normal ≥ 20 J/cm²) for UVA (light source: Philips TL 20W/12 (UVB), Philips TL 20W/09 (UVA)). Five days after phototesting, the patient developed thin erythro-squamous plaques in all UVB test fields (doses between 25 and 150 mJ/cm²). During the following days, the psoriatic lesions increased in size, exceeding the borders of the UVB phototesting area. The skin biopsy taken from such a lesion revealed a psoriasiform dermatitis with microabscesses within parakeratotic isles of the stratum corneum, suggestive of an early eruptive form of psoriasis vulgaris. A urinary tract infection without clinical symptoms was treated with antibiotics (ciprofloxacin

500 mg/day for 3 days) after PP was diagnosed. In addition to topical therapy with corticosteroids and salicylic acid we started a psoralen+ultraviolet A (PUVA) therapy combined with oral acitretin (0.4 mg/kg body weight). After 2 weeks of treatment (initial UVA dose 0.3 J/cm², maximum dose 0.5 J/cm², cumulative dose 3.2 J/cm²) without a significant improvement of the skin condition we switched the therapy regimen to oral therapy with cyclosporin A (4 mg/kg body weight). This led to a rapid improvement in skin lesions and to a normalization of the blood cell count. The patient was still free of disease at 6 weeks of cyclosporin A therapy.

DISCUSSION

PP is a clinically well-known, but poorly defined, psoriasis subset. Scandinavian investigators found 7 of 35 patients with PP to have a lowered MED for UVB irradiation (1). In this study 18/35 patients had a history of polymorphous light eruptions (PLE) with secondary evolution of psoriasis (Koebner phenomenon), 17/35 patients had no signs of preceding PLE reaction but developed new psoriatic lesions between 11 and 48

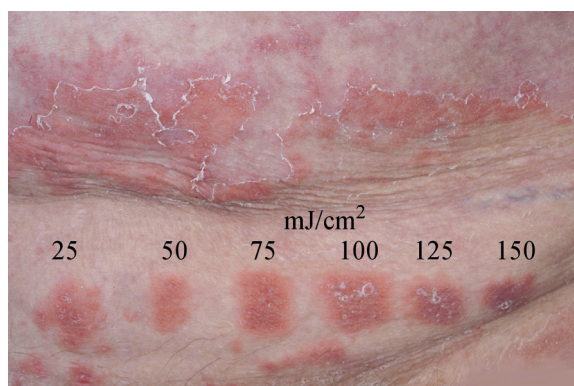


Fig. 1. Photo-testing area for determination of minimal erythema dose for UVB, 6 days after irradiation.



Fig. 2. (a and c) Erythematous squamous papules and plaques on the elbow and abdomen before treatment with cyclosporin A (6 days after photo-testing). (b and d) After treatment with cyclosporin A for 6 weeks.

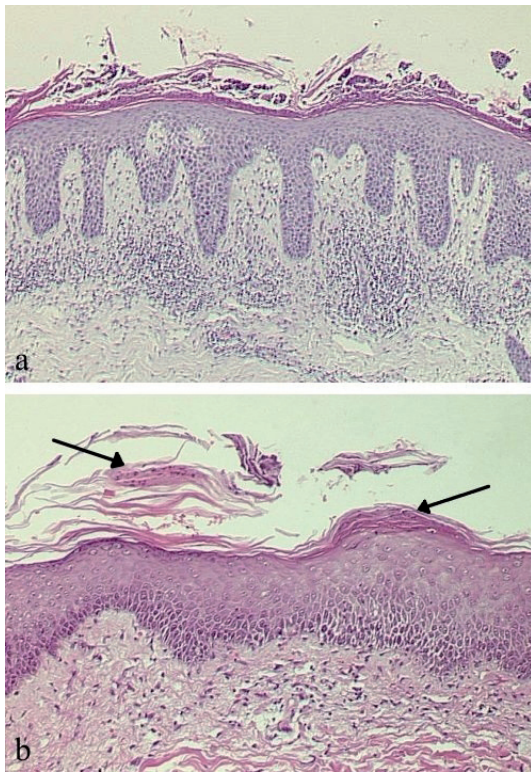


Fig. 3. (a) Biopsy taken from an erythroquamous plaque on admission: psoriasiform hyperplasia with elongated rete ridges, absent granular zone and confluent parakeratosis containing layered collections of neutrophils. Dilated tortuous capillaries and lymphocytes around vessels. Haematoxylin and eosin (H&E) $\times 50$. (b) Skin sample taken from the 100-mJ/cm² phototesting area on the lower abdomen 6 days after irradiation: irregular acanthosis, neutrophils in mounds of parakeratosis (arrows). Slight spongiosis in the lower part of the epidermis. H&E $\times 100$.

days after provocation with high doses of UVA and less frequently with UVB exposure. Bernard (2) hypothesizes the existence of yet another group of patients with PP with a negative photoprovocation: even in the absence of a pathological MED, these patients have an exceedingly low threshold for the Koebner phenomenon (ultra-Koebnerization), which can be diagnosed by triggering isomorphic responses by types of trauma other than irradiation.

Patients with PP have a statistically significant higher frequency of skin type I, a heredity of photosensitivity, advanced age, difficulties in therapy, and face and hands involvement compared with non-photosensitive patients (3, 4). Other causes of photosensitivity should be excluded in such patients. The patient in this case report met the criteria advanced age, difficulties in therapy, and face and hands involvement. She was not able to provide any information on other family members' photosensitivity. Her skin was assessed as type II. Due to the poor definition and the low prevalence of PP, no clinical trials have been conducted in patients with PP. PUVA (5), UVB (6) and systemic immunosuppressive

drugs such as cyclosporin A (7) have been used successfully in the treatment of PP in individual patients.

The patient described here showed no clinical or serological evidence for an underlying cause of PP, and in particular no disorders concomitant with photosensitivity. The role of the urinary tract infection without any clinical symptoms remains unclear, but it is unlikely that this infection caused the exceptional skin reaction to UVB irradiation. The antibiotic treatment was not started until PP was diagnosed, ruling out any photosensitizing drug reaction to ciprofloxacin. Although T-cell dysregulation is considered to be an important feature of psoriasis, there have been some reports stating that elevated amounts of neutrophils in the peripheral blood (as in the patient described here) may also play a role in defining the activity of psoriasis. A more severe disease might be associated with an increase in the inflammatory response in the peripheral blood (WBC count, ESR, CRP) and particularly with an activation of neutrophils (8, 9). These findings are supported by studies of the spontaneous mouse mutation flaky skin with some features of psoriasis, including a prominent infiltrate of neutrophils and microabscesses within a hyperkeratotic epidermis. A marked reduction in these psoriasis-like symptoms was achieved by injection of neutrophil-depleting monoclonal antibodies into these mice (10).

Kudoh et al. (11) published one case of photosensitive psoriasis who developed a macule in the area exposed to a suberythematous dose of UVB. In contrast to our case it took the psoriatic lesion 3–4 weeks to develop after phototesting. In another patient with a long history of psoriasis, new skin lesions developed in the UVB-testing fields after 5–6 days (6). Interestingly, therapeutic UVB-irradiation led to a complete remission of the disease in this patient after 6 weeks. To the best of our knowledge all other reports of PP describe the occurrence of psoriatic lesions after application of much higher UV irradiation doses.

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