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Clinical and Therapeutic Aspects of Polymorphous Light Eruption

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Key Words
Polymorphous light eruption · Ultraviolet A · Ultraviolet B · Phototesting · Phototherapy

Abstract
Background: Polymorphous light eruption (PLE) is an idiopathic eruption induced by ultraviolet (UV) radiation (UVR). ***Objective:*** Evaluation of the clinical aspects, diagnostic criteria of PLE in a major Swiss referral center. ***Methods:*** 25 patients with PLE were tested with a standardized protocol for the assessment of photodermatoses. ***Results:*** 25 patients (22 women vs. 3 men) were identified. Papular and papular-vesicular eruptions were the most common clinical presentations. 6 of 25 patients had a reduced minimal erythema dose (MED) für UVA and 8 of 25 patients had a reduced MED for UVB. Photoprovocation was positive in 11 of 20 patients for UVA and 7 of 20 patients for UVB. Photohardening with narrow-band UVB was successful in 8 of 10 patients. Combined UVA/UVB therapy had a satisfactory effect in 10 of 15 patients. Narrow-band UVB therapy was still successful after ineffective UVA/UVB therapy. ***Conclusion:*** The MED was of no value for the diagnosis of PLE. The typical lesions were reproduced by UVA and UVB photoprovocation. We recommend photohardening with narrow-band UVB (311 nm).

Introduction

Polymorphous light eruption (PLE) is a chronic light-induced skin disorder that appears to affect all ethnic groups [1]. There are several morphologic subtypes, but individual patients tend to keep their typical eruption over years [2–4].

In Europe, there is a strong female predominance, with female:male ratio ranging as high as 9:1 probably because there is a greater willingness in female patients to be seen by a doctor [3, 5]. An increasing incidence was reported for South America [6]. The clinical variants of PLE are papular, plaques, papulovesicular, erythema multiforme-like and hemorrhagic type [2, 3, 5].

Krutmann and Morita [7] suggested possible pathogenetic factors such as ultraviolet (UV)-mediated upregulation of adhesion molecules on the keratinocyte cell surface as well as UV-induced apoptosis in skin-infiltrating cells. The UV action spectrum is controversially discussed. Some authors reported that UVA is the wavelength that induces the specific skin lesions in most patients [5, 8, 9]. Other authors also found a significant number of positive tests for the UVB spectrum [2, 10–12].

In milder cases, prophylaxis of brusk UV exposure might be sufficient in combination with sunscreens that protect over the whole UV spectrum [13]. Otherwise so-called 'hardening' can be achieved by low-dose UV therapy several weeks before sun exposure [3, 14]. The purpose of this retrospective study was to evaluate the clinical aspects of this dermatosis, the photodiagnostic tests and phototherapy in 25 patients recruited from a major dermatological service in Switzerland.

Patients and Methods

45 patients (33 female vs. 12 male) were seen in our outpatient service and given the diagnosis PLE. 25 of them were referred for photodiagnostic procedures and phototherapy. The diagnosis of PLE was based on the following standard criteria: clinical symptoms; positive results of the photoprovocation tests, and clinical and histological analyses in five positive skin reactions [1, 4, 12]. Patients with a photosensitive dermatosis such as lupus erythematosus and porphyria were excluded by direct immunofluorescence, circulating antinuclear factor (titer $<1:160$) determination, and urinary and stool porphyrin determinations.

Of the 25 patients 22 (88%) were women and 3 (12%) were men with a mean age 44 (range 18–68) years for the women and 51 (range 30–74) years for the men. The average of duration of disease was 50 (range 6–120) months for the men and 104 months (4 days to 240 months) for the women. 11 patients (44%) had a papular type, 11 patients (44%) a papulovesicular type, 2 patients (8%) a plaque, and 1 patient (4%) an erythema multiforme-like type.

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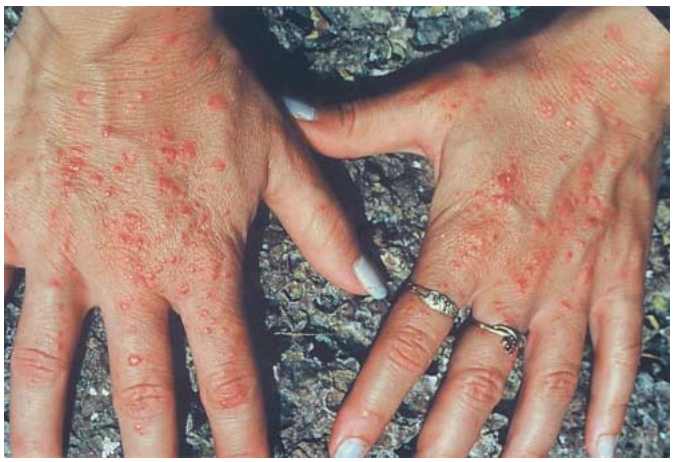

Fig. 1. Papular type of PLE associated with severe itching during holidays.

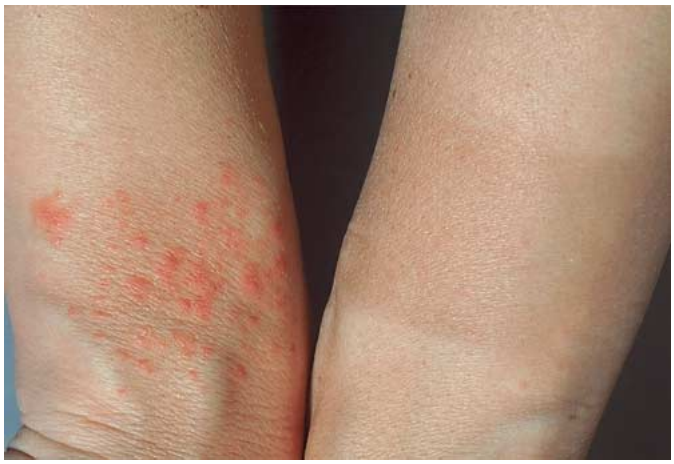

Fig. 2. Papular lesions of the patient shown in figure 1 after UVB (left) and pigmentation (right) after UVA photoprovocation.

Skin Type

In accordance with the classification of Fitzpatrick, 4 patients (16%) had skin type I, 5 patients (20%) skin type II, 14 patients (56%) skin type III, and 2 patients (8%) had skin type IV.

Minimal Erythema Dose

For minimal erythema dose (MED) testing we used a Waldmann system (Waldmann Lichttechnik, Villingen-Schwenningen, Germany). The Waldmann UV800 has an emission spectrum from 285 to 350 nm, and UV test from 330 to 440 nm.

The MED for UVA and UVB was determined in all 25 patients. It was normal (range 1.5–9.0 J/cm^2) for UVA in 19 patients (76%) and abnormal in 5 females (22%) and 1 male (33%) patient. The MED for UVB was normal (range 0.08–0.036 J/cm^2) in 17 patients (68%) and abnormal in 5 women (22%) and 3 men (100%).

Photoprovocation Test

20 patients were subjected to photoprovocation by exposing skin areas (5 × 8 cm) at the predilection site with UVA 100 J/cm^2 and 1.5 × MED UVB over 3 consecutive days. A reaction was considered positive when a typical skin lesion and/or histology was generated by this test (fig. 2). The photoprovocation for UVA was positive in 11 patients (55%) and negative in 9 patients (45%). The photoprovocation tests showed a pathological reaction to UVA in 66% (n = 2) of the men and 40% (n = 9) of the women, and to UVB in 33% (n = 1) of the men and 27% (n = 6) of the women. The reaction varied from burning, itching, severe erythema, papules, vesicles, plaques, and painful sensation and corresponded well to the clinical symptoms after natural UV exposure.

Table 1. Criteria for evaluating the efficacy of therapy

Excellent (+++)	No more relapses and disappearance of patients' complaints after treatment
Good (++)	Reduction in the frequency of relapses and clinical symptoms
Improved (+)	Relapses but subjectively reduced clinical symptoms during outdoor activities
Failure (0)	Identical clinical symptoms during therapy

Photopatch Test

These tests were performed in 2 patients (8%). 1 male patient had photocontact allergy to chlorothiazides but this was without clinical relevance.

Results

The effect of therapy was based on the patients' case history and wherever necessary by contacting the patients by phone. The number of relapses and symptoms during outdoor activities was assessed (table 1). 15 patients (60%) were treated with prophylactic combined UVA + UVB radiation: 5 patients (33%) failed to improve; 4 patients (27%) improved; 2 patients (14%) had good, and 4 patients (27%) excellent results. There were relapses in 9 patients (40%). 2 patients (8%) were treated with prophylactic psoralens + UVA (PUVA) therapy: 1 patient responded well, and 1 patient responded excellently. A relapse was seen in 1 patient. 10 patients (45%) were treated with prophylactic UVBnb (311 nm) radiation: 2 patients failed to respond; 2 patients improved, and 6 patients had an excellent response. The rate of relapse was 50% [4]. 5 patients did not experience a new episode of skin eruption during the next high-dose UV exposure. Patients who did not respond adequately to UVA + UVB therapy were successfully treated with UVBnb (311 nm). Patients were successfully treated with PUVA after combined UVA + UVB and UVBnb (311 nm) treatments were unsatisfactory.

Discussion

There are reports from the southern hemisphere that the incidence of photosensitivity disorders including PLE is rising, probably due to increased UV exposure because of the Antarctic ozone hole [6]. PLE is a common chronic idiopathic photoderma-

tosis. Its clinical presentation and history are typical, and it can be easily differentiated from photoprovoked dermatosis and photoallergic disorders [1–5]. In our series of patients and in other studies, the papular and papular-vesicular presentation covers approximately 80% of all patients. All skin types are affected. The MED is typically reduced only in a minority of patients and therefore has no diagnostic significance. Photoprovocation tests are therefore mandatory using UVA and UVB. Our data and data from several extensive studies [1, 2, 11, 12] demonstrate that the majority of cases are sensitive to UVA irradiation. However, also UVB is the major inducer of the skin lesions in approximately 30% of the patients [2]. There is a single large study that could not confirm UVB as an inducer of PLE lesions [4]. This is probably due to the test setting applied (light source, number of UV doses applied).

From our clinical experience, phototesting results are mandatory to advise the patients correctly concerning the choice of sunscreen [15] and behavior (sun tan studios, sun exposure behind window glass) [2]. Since only severe cases of PLE are referred to our clinic, prophylactic measures such as the avoidance of brisk UV exposure and the use of highly efficient sunscreens did not prevent the reappearance of the typical skin eruptions in our patients. Therefore, photohardening with various phototherapeutic options was performed, including combined UVA/UVB treatment, systemic PUVA therapy and narrow-band UVB therapy [10, 12, 14, 16–18]. Between 1994 and 1998 combined UVB therapy was the most common therapy. However, the retrospective evaluation of our small patient population suggests that this treatment option is of minor efficacy. If we compare the outcome of UVA/UVB treatment with the results of narrow-band UVB, we feel that narrow-band UVB is far more efficient and probably achieves a similar efficacy as PUVA therapy. Therefore, narrow-band UVB should be the first choice in the photohardening of PLE patients. In special situations, such as exclusive induction of the PLE lesions by UVB as shown in figure 1, 2, correct application using narrow-band UVB might be not possible. In these rare cases, PUVA therapy is a valid alternative.

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