

UVA1 Irradiation is Effective in Treatment of Chronic Vesicular Dyshidrotic Hand Eczema

Sir,

Vesicular eczema of palms and soles (cheiropompholyx) is a common disease that often runs a chronic course. It is clinically characterized by recurrent pruritic vesicles and bullae frequently followed by persistent eczema with scaling, fissuring and hyperkeratosis. It is therefore a major cause of disability due to occupational dermatoses (1) and may result in long periods of sick leave. It can occur in both classic contact dermatitis and in atopic eczema, but often the cause remains unknown. To date, the conventional therapeutic approach mainly consists of topical application of corticosteroids, adstringent hand baths and PUVA administration. In the present study we investigated the efficacy of local UVA1 irradiation on chronic vesicular hand eczema. Palms and backs of hands of 12 patients with acute exacerbation were exposed to 15 UVA1 irradiations with a dose of 40 J/cm² per day over a period of 3 weeks. In 10 out of 12 patients, dyshidrotic hand eczema gradually improved and no relapse was reported during a 3-month post-treatment interval. Probably due to the small number of patients, no differences in responder rates regarding the underlying cause of the disease, such as classic contact dermatitis or atopic disposition, could be discovered in our study. Therefore local UVA1 phototherapy proved to be a promising new therapeutic approach in the treatment of vesicular hand eczema.

MATERIALS AND METHODS

Twelve patients with acute exacerbation of a chronic dyshidrotic hand eczema (8 females, 4 males, age: 19–56 years) were exposed to UVA1 irradiations (UVA1 Kaltlicht, Photomed, Gehrden) over a period of 3 weeks. In three patients, vesicular eczema of the hands was related to classical contact dermatitis. The remaining nine patients revealed a medical history of atopic diseases such as atopic eczema, hay fever or allergic asthma. All patients received five UVA1 irradiations per week in a dose of 40 J/cm² per day accumulating to an overall dose of 600 J/cm². No topical or systemic corticosteroids or antihistamines were allowed during phototherapy. The severity of hand eczema was assessed using the recently introduced Dyshidrosis Area and Severity Index (DASI), which combines objective criteria (vesicles, erythema, desquamation and area involved) and subjective criteria (pruritus) (5). The index represents the sum of score points of the level of severity

(1 point = mild; 2 points moderate; 3 points = severe) of the objective criteria vesicles (V), erythema (E) and desquamation (D) and of the subjective criteria pruritus (I) multiplied by the area affected (1 point = <20% to 5 points = 81–100% of hand surface). Consequently, the maximum score can amount to 60.

$$\text{DASI-Score} = V + E + D + I \times \text{points for affected area}$$

RESULTS

Phototherapy was well tolerated by all patients and no side effects were observed. During the first week of treatment, all but one of the patients reported a marked relief of pruritus. After the 3-week treatment period DASI scores significantly decreased with almost complete clearing of vesicular hand eczema in 10 out of 12 patients. A reason for the discrepancy between right and left hand after-treatment scores in patients nos. 9 and 11 could not be detected.

No relapse has since been noted in patients nos. 1–10 during a 3-month post-treatment follow-up (Table I). After a marked improvement during phototherapy, two patients (nos. 11, 12) reported exacerbations: Patient no. 11 revealed new eruptions of vesicles in the course of a bronchitis, whereas patient no. 12 suffered exacerbation after renewed contact with a well-known allergen (paraben). Differences due to the various underlying causes of chronic vesicular hand eczema (such as atopic or classical contact dermatitis) could not be detected in the short-term effectiveness of UVA1 irradiation or in the number of relapses.

DISCUSSION

Local UVA1 phototherapy proved to be highly effective in the treatment of chronic vesicular hand eczema and seems to be a promising new therapeutic approach. The conventional therapeutic approach to treating chronic vesicular dyshidrotic hand eczema mainly consists of the topical application of corticosteroids and adstringent hand baths. Because of the chronic course of the disease the administration of corticosteroids is required for longer periods, provoking side effects related to the drug. Acute exacerbations with bullous eruptions (cheiropompholyx)

Table I. Effects of UVA1 on chronic vesicular hand eczema

No.	Age/sex	Score results before treatment (left/right hand)	Score results at end of treatment (left/right hand)	Non-parametric Wilcoxon matched-pairs signed-ranks test	
1	56/F	32/34	1/1	<i>Mean left hand</i>	<i>Mean right hand</i>
2	35/M	33/33	1/1		
3	45/F	36/36	2/2	19.83 before treatment	20.41 before treatment
4	53/F	10/10	2/2	4.25 after treatment	3.50 after treatment
5	19/F	12/12	2/2		
6	45/F	16/16	4/4		
7	23/M	14/14	6/6	<i>Left side</i>	<i>Right side</i>
8	30/F	11/11	3/3		
9	47/M	21/21	6/1	2-tailed $p=0.002$	2-tailed $p=0.002$
10	50/F	16/16	1/1		
11	33/M	10/15	8/4		
12	37/F	27/27	5/15		

and spreading of lesions to other parts of the body are frequently seen and may sometimes require systemic steroid therapy. The successful use of cyclosporine has also been reported (2). Topical PUVA photochemotherapy has been successfully applied; however, in order to achieve persistent remission, long-term topical PUVA treatment is necessary which is associated with the possibly increased risk of skin cancer development (3). High doses of UVA1 (340–400 nm) have been successfully integrated into the therapeutic approaches of treating severe atopic eczema. The marked improvements experienced in these cases were attributed to effects on IgE-bearing epidermal Langerhans' cells as well as modified secretory patterns of dermal mast cells and keratinocytes (4).

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Tanja Schmidt, MD, Dietrich Abeck, MD, Kerstin Boeck, MD, Martin Mempel, MD and Johannes Ring, PhD, MD
Department of Dermatology and Allergy, Biederstein, Technical University Munich, DE-80802 Munich, Germany.

Failure of Gluten-free Diet in Celiac Disease-associated Alopecia Areata

Sir,

Alopecia areata (AA) is quite a common disorder and occasionally may coincide with internal diseases. Celiac disease is the last of such alleged associations. Even its improvement with gluten-free diet has been advocated (1, 2).

A 32-year-old woman had had AA for 14 years. Initially, she had been treated with intralesional corticosteroids with temporary benefit. Scalp hairs regrew to shed again when the treatment was stopped, eventually resulting in alopecia universalis. In 1993, high titres of IgA antiendomysial (EmA) and anti-gliadin antibodies (AGA) were found. The patient, though asymptomatic, underwent endoscopy and jejunal biopsy, and celiac disease was diagnosed. Since then, she has been on a gluten-free diet for 4 years. The raised EmA and AGA titres became normal within a few months. All other laboratory tests have been within normal limits. Despite the good control of the celiac disease, no improvement of alopecia was obtained.

Her brother had high titres of IgA EmA and AGA and villos atrophy at the jejunal biopsy. For years, he had also had vesicular lesions on both elbows and back, which had been interpreted as atopic dermatitis. Direct immunofluorescence showed granular deposits of IgA at the top of dermal papillae consistent with the diagnosis of dermatitis herpetiformis (DH). DH improved with a gluten-free diet, which he followed steadily for 4 years. He was still on diet when AA developed on his legs, wrists and neck. IgA EmA and AGA were then negative; routine laboratory tests were within normal limits or negative and DH lesions were not flaring up. AA improved spontaneously about 2 months later.

DISCUSSION

The association between AA and celiac disease has been reported with 1% of patients having IgA EmA and AGA. IgG AGA were also found in 4% and 10% of AA patients tested with indirect immunofluorescence and ELISA, respectively. According to Volta et al. (2), the prevalence rate of celiac disease in patients with AA is 1 : 116, a figure higher than that of celiac disease in general population (1 : 305). In addition, three patients had a complete or partial regrowth of the hair after being submitted to the gluten-free diet (1, 2).

We confirm the possible association of the two diseases, but fail to do so for the effect of the diet. In fact, one of our patients developed AA when on diet, the other one deriving no benefit at all.

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P. Bondavalli, G. Quadri, A. Parodi* and A. Rebora
Department of Dermatology, University of Genoa, Viale Benedetto XV n.7, IT-16132 Genoa, Italy.

* Corresponding author.