Ultraviolet-B Phototest in Patients with Atopic Dermatitis

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TORINUKI, W. Ultraviolet-B Phototest in Patients with Atopic Dermatitis. Tohoku J. Exp. Med., 1990, 162 (4), 367-368 — To evaluate the possibility of ultraviolet (UV) rays as an aggravating factor of atopic dermatitis (AD), UV-B phototest was performed in patients with AD. Resultingly, minimal erythema doses for UV-B were all within normal range and AD lesions were not reproduced by irradiation. UV-B may not be an important factor for skin aggravation in patients with AD.

Atopic dermatitis (AD) is a chronically relapsing skin disorder of early infancy, childhood, and adolescence. Ultraviolet (UV) radiation exacerbates some cases of AD (Frain-Bell and Satchard 1971). However, the role of UV in such exacerbations may be difficult to establish because of reactions to seasonal airborne antigens. The purpose of this study is to evaluate the possibility of UV as an aggravating factor of AD, and I report the result of UV-B phototest in AD.

A total of 77 patients with AD, 38 males and 39 female, were selected for this study. They ranged in age from 1 to 29 years (mean, 10 years). The diagnosis was made on the basis of the clinical appearance and distribution of skin lesions, the clinical course, and the family history of atopic diseases. All patients fulfilled the criteria for AD (Hanifin and Rajka 1980).

First, patients and the parents were asked whether AD eruption was exacerbated by exposure to sunlight. Only 2 of 77 patients answered “perhaps”, but the remaining 75 patients said “unlikely or no”. Therefore, there was no clear evidence of photoaggravation in the group I studied.

Second, the study details were fully discussed with each patient, and informed consent for phototesting was obtained in 20 patients; 2 patients had answered “perhaps”, 18 patients “unlikely or no”, as mentioned above. Phototest was performed with UV-B, on clinically normal skin on the back, during June to September of 1989. The result of phototestings was not influenced by the season chosen for testing, if the untanned back-skin was selected as test site. For light source, seven tubes (Toshiba FL20SE-30, Tokyo), emitting mainly UV-B peaking at 315 nm, were used, housed in reflector unit of a Dermaray (Eisai, Tokyo). The irradiance of UV-B, which was measured by a Topoon UV radiometer UVR-305/365 (Eisai, Tokyo), was 1.0 mW/cm² at a distance of 30 cm. At first, in 20 patients the 24 hr minimal erythema dose (MED) of UV-B was determined; resultingly, MED for UV-B were all within normal range in our clinic (58-173 mJ/cm²). Next, 3×3 cm adjacent test site was irradiated on 12 of 20 patients to induce skin lesions with single exposure of 5 MED, because skin lesions were successfully reproduced with single exposure of 3-5 MED of UV-B, in some patients suffering from polymorphous light eruption.

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(Miyamoto 1989). Test reaction was read at 3 and 7 days after irradiation. However, the AD lesions were not reproduced by irradiation and only brownish pigmentation was observed in test area.

The majority of individuals with AD experienced improvement after exposure to sunlight more often than impairment (Berg 1989), and the eczema in patients with AD improved with UV-B (Britton et al. 1988). Many patients with AD who live in northern countries regularly experience an improvement during the summer months, and many find that a vacation in a sunny climate may be extremely beneficial. For these reasons, artificial UV irradiation has been used as a treatment for AD, with reports of benefit from UV-B (Jekler and Larko 1988). Though Frain-Bell and Satchard (1971) described five atopic girls who had eczematous or pruriginous changes in light-exposed areas, phototesting was negative and this syndrome may be difficult to distinguish from Hutchinson's summer prurigo.

Therefore, from the present study and the review of literatures, UV-B may not be an important factor for skin aggravation in patients with AD.

References