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Occupational UVC-induced exacerbation of atopic dermatitis in a welder

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Welding generates UV radiation mainly within the UVC-range as well as infrared radiation (1). Welders are well protected by means of a welding helmet, but unintentional exposure often occurs and may lead to erythema. The symptoms are usually mild and the cause is obvious to the welder, so this usually does not present a problem.

In up to 10% of patients with atopic dermatitis, this skin disease is aggravated by solar exposure or by phototherapy. This reaction is commonly categorized as photosensitivity reaction (particularly to UVB in patients with skin types I and II); other patients seem to experience exacerbation due to the infrared in the solar spectrum and thus sweating (2). Little is known about the direct effect of UVC on atopic dermatitis.

Case Report

We report a case of a welder in which exacerbation of atopic dermatitis occurred repeatedly upon occupational exposure to UVC, and which subsided only after the patient was removed from the welding site.

A 32-year-old male patient with a positive family history of atopic dermatitis and a personal history of childhood scalp eczema, allergic asthma and allergic rhinoconjunctivitis presented to our occupational dermatoses clinic. The patient had suffered from eczematous skin lesions since the age of 15 years, with exposure to sun light leading to aggravation. At the age of 25 years, the patient commenced work as a welder, which resulted in aggravation of the skin lesions especially in the face and neck area. As a protective measure, a welding shield covering the face and the neck with a viewing window was provided. The shield was mainly used during seam welding for longer periods. During spot welding and welding of only minor seams, as well as for delicate work or during welding with limited space, the shield was not used for short periods of time. Furthermore, several other welders worked at a distance of 2 to 5 m, resulting in UVC exposure from

the fellow welders. The patient's colleagues experienced no skin problems. When the patient stopped welding at the age of 26 years, his skin improved considerably. At the age of 31 years, circumstances forced him to work as a welder again, which led to a prompt aggravation of the atopic dermatitis, with lesions predominantly in the region of the face and the neck. In the following months, the patient repeatedly suffered from rapid onset of episodes upon welding and regression of inflammation when he was off work. The overall state of the skin deteriorated and treatment with topical and systemic steroids no longer sufficed. The patient had to be admitted to hospital for further treatment. Skin lesions cleared under therapy with topical corticosteroids and systemic cyclosporine A. Intermittent aggravation, however, again occurred upon a 1-day UVC-exposure due to welding. After this episode, the patient changed his job and had no further occupational UV exposure. In the follow-up over the next 4 months, his skin condition remained stable.

Atopy screening revealed sensitization against dust mites (*Dermatophagoides pteronyssinus* and *D. farinae*), cat and horse hair, parakeet feather, pollen of grass, hazel, birch and alder as well as yellow of egg, peanut, malt, sodium disulfite and carmine. Serum eosinophil cationic protein (ECP) was 22 mg/l (normal value <18 mg/l); serology revealed extreme elevation of IgE to 7900 kU/l (normal value <26 kU/l).

To determine the minimal erythema dose (MED, areas at the back were irradiated with a series of 6 exposures of filtered UVA with doses from 1.5 to 9.0 J/cm² and of UVB with doses from 0.025 to 0.25 J/cm² respectively. The MED in the UVA range was lowered with development of erythema already at a dose of 5 J/cm² (normal range 20–50 J/cm²). The MED in the UVB range was normal with erythema at 0.05 J/cm² (normal range 0.02–0.05 J/cm²).

A sunscreen preparation (Anthélios, Trademark of La Roche Posay, Kronberg, Germany) was applied to an area of 5×5 cm on the forehead 1 h prior to UV ex-

posure upon welding. The absorption spectrum of this photoprotectant is predominantly within the UVA and UVB range, with almost no effect in the UVC range. No significant difference was seen between the resultant dermatitis on the protected skin and the adjacent skin where no sunscreen preparation had been applied.

Comment

Arc welding equipment often develops temperatures of up to 6000°C with an intensity of 600 W/cm². The resulting UV radiation, mainly within the UVC range, can reach even distant persons (directly or indirectly by reflection) and thus may cause adverse effects in these individuals (3, 4). Barth et al. (5) demonstrated that non-welders working near welding stations are exposed to doses of UV radiation which exceed the recommended limits by about 8×. Evaluation of dermatologically relevant emission/effect and thus risk is difficult, partly due to individual differences in susceptibility. The possibilities of individual hazard monitoring are thus limited.

The primary protection measure provided at any welding site is a welding shield. These shields are, however, rather inconvenient to use and welders are often tempted to do soome of their work without this protection. In many work places, there are several welding stations at a distance of between 2 to 5 m from one another, leading to inadvertent UV exposure of fellow workers.

The literature on the effects of UV exposure due to welding on the skin is scarce. Shehade et al. (6) described 1 case in which work-related photodermatitis occurred in a patient due to spot welding without use of the protective face mask. Bruze et al. (7) recently described an unusual case in which dermatitis on the cheeks in a welder was caused by reflection of UVC by an additional protective hood made of which cloth meant to protect the head and neck. Roelandts & Huys (8) describe 2 cases of UVC-induced erythema after accidental exposure to UVC radiation associated with broad-band photosensitivity, suggesting a persistent light reactor state. Our case is special, since our patient suffers from atopic dermatitis, which in itself goes with an impairment of the immune system, thus presenting a further variable not present in the cases described to date. While cases of UVA- and UVB-photosensitive atopic derma-

titis have been well described in the literature (9–12), this is the first case of UVC-induced deterioration of atopic dermatitis. Since the use of the photoprotectant Anthélios "L", which also has practically no absorption in the UVC range, did not prevent work-related aggravation of dermatitis, we suggest that the enhanced UVC exposure is the cause of dermatitis in our patient. Infra-red exposure, however, cannot be ruled out as an additional cause.

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