

disciplines (ENT-doctors, paediatricians, anaesthesiologists and oncologists), practising doctors treated anaphylactic patients regularly. The average number of treated patients per practice was low, but considering the high number of practising doctors, they deliver a broad number of anaphylactic cases.

The reported triggers of anaphylaxis are in line with data from the literature. However, the most common trigger factor of anaphylaxis reported by these doctors, who perform SCIT on a regular basis, was SCIT. This observation points to the necessity of gaining more information on severe reactions during SCIT, which are probably underreported.

There is a possibility that the reported anaphylactic reactions were not diagnosed properly, as only 19% of the practitioners were specialized in allergy. A recall-bias also needs consideration. Still, our data clearly show that SCIT seems to be an important cause of anaphylaxis in private practice and are in line with a previous survey from Bernstein et al. (4).

In conclusion, anaphylaxis is relevant in private practices and more epidemiological data are urgently needed.

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Successful treatment of solar urticaria with anti-immunoglobulin E therapy

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Solar urticaria is characterized by intense pruritus and wheal and flare-type skin reactions at the sites exposed to sunlight. The eliciting factor is UVA, UVB and/or visible light with individual action spectra for each patient (1). Nonsedating H1-antihistamines are the first line therapy for solar urticaria, but in severe cases conventional antihistaminic treatment is often unsuccessful. High-grade sun blockers are usually not sufficient, and light-hardening therapy, although sometimes quite effective, is not appropriate for long-term treatment because of the risk of skin tumour development.

We present the case of a 52-year-old caucasian female patient with severe solar urticaria who experienced wheals and pruritus induced by sun- and interior light. Minimal sun exposure, e.g. during the short walk to her car in the mornings, sufficed to induce cutaneous symptoms, and prolonged light exposure resulted on occasion in systemic symptoms including dizziness, headaches and

respiratory problems. Consequently, this patient experienced a major reduction in her quality of life because of her urticaria, which significantly interfered with her private and professional life.

The patient was diagnosed with solar urticaria in February 2007 by light provocation, revealing a threshold for UVA light of 1.4 J/cm². Since then, the patient has been treated with various nonsedating and sedating antihistamines (up to eightfold the recommended daily dose), histaglobin injections, various diets and *Helicobacter pylori* eradication therapy, none of which resulted in a significant reduction of her symptoms. Initially, we treated the patient with a UVA rush hardening regimen (2), which lowered the UVA threshold considerably. However, the patient still had light-induced symptoms and was unable to continue the twice weekly UVA exposures needed for maintaining the reduced UVA threshold.

The pathogenesis of solar urticaria is still poorly understood. One of the favoured hypotheses is that irradiation with light produces a photoallergen within the skin, which crosslinks specific immunoglobulin E (IgE) bound to the FcεRI-receptor on cutaneous mast cells. The ensuing mast-cell degranulation then leads to the release of a wide range of mediators, including histamine, which cause pruritus and the typical wheal and flare reaction. This hypothesis offers the possibility that anti-IgE treatment can be effective in the treatment of solar urticaria.

Omalizumab is a recombinant humanized monoclonal antibody against IgE. It acts by binding free IgE at the same site that IgE would bind to its high-affinity receptor (FcεRI) on mast cells, thereby reducing free IgE in the serum (3). Recently, successful treatment of cholinergic urticaria (4), cold urticaria (5) and chronic urticaria (6) with omalizumab has been reported. Because of these positive reports, and because none of the previous treatment strategies had been successful in our patient, we began to treat our patient with 150 mg omalizumab every 4 weeks. The patient does not suffer from allergic rhinitis or other allergies, had total IgE levels of 22.0 kU/l, and is otherwise healthy.

A possible new treatment alternative and new insight into the pathogenesis of solar urticaria.

Within 4 weeks after the first injection of omalizumab the patient reported back to us with a dramatic improvement of light-induced symptoms. Standardized light-provocation test with UVA and UVB 4 weeks after the first injection was completely negative, even at high doses of UV light (Fig. 1). The patient reported that she did not have a single wheal since the first injection of omalizumab, although she was frequently exposed to sun light. Furthermore, her respiratory

problems improved and she was able to stop antihistamine medication completely.

This is to our knowledge the first report of a successful treatment of solar urticaria with omalizumab. The observed rapid improvement of the symptoms during anti-IgE therapy supports the hypothesis that solar urticaria might be mediated by IgE. The entity of the photoallergen remains unclear. Further clinical trials should investigate whether

anti-IgE treatment is in general beneficial for patients with solar urticaria; these future investigations might also help to understand the pathogenic mechanism of the disease.

This case shows that solar urticaria can be rapidly and successfully treated with omalizumab at the recommended dosage, even in the context of relatively low total IgE levels.

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Figure 1. Omalizumab treatment effectively prevents light-induced symptoms in a patient with solar urticaria. The patient presented herself in our department before treatment with omalizumab (A) and on a sunny day 4 weeks after treatment (B). Before and 4 weeks after treatment, standardized light-provocation test (Saalmann-Multitester SBC LT 400) with UVA (lower row) and UVB (upper row) was performed (C, D). The numbers indicate the UV-doses applied in each field in J/cm² or mJ/cm², respectively. UVB irradiation shows only a mild erythema (C), irradiation with UVA shows characteristic wheals with a threshold of 1.4 J/cm² before treatment (C), and no detectable erythemas, wheals or pruritus 4 weeks after the start of the omalizumab therapy (D).

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