

## Clinical Letter

### Successful treatment of solar urticaria with omalizumab

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Dear Editors,

We report a case of solar urticaria, which – compared to chronic spontaneous urticaria – showed a therapeutic response to the anti-IgE antibody omalizumab only after prolonged administration and at a higher dose. A rare photodermatosis, solar urticaria is characterized by pruritus, erythema, and wheals following light exposure. The action spectrum is usually in the UVA (320–400 nm) and visible-light (400–600 nm) wavelength range, less commonly in the UVB range (280–320 nm) [1]. Apart from consistent UV protection, treatment options include antihistamines and photohardening, but also cyclosporine A, chloroquine and intravenous immunoglobulins (IVIG), as well as extracorporeal photopheresis and plasma exchange [2]. In individual case reports, the condition has shown varying degrees of response to omalizumab [3–7].

A 51-year-old swimming pool mechanic – working outdoors and without any underlying atopic disorders – reported that, for more than ten years, he had developed pronounced erythema, wheals, and pruritus following brief exposure to light, even through window panes. Symptoms usually regressed spontaneously after two hours, and there was no improvement over the course of continuous exposure in the spring and summertime. In 2006, after spending a prolonged time in the sun, he had for the first time experienced a systemic reaction associated with diaphoresis and dizziness. The minimum urticarial dose (MUD) was 2.2 J/cm<sup>2</sup> with UVA; 2 mJ/cm<sup>2</sup>, with UVB. Phototesting yielded erythematous wheals within a few minutes (Figure 1). Lab tests showed an elevated antistreptolysin titer (without clinical correlate) as well as elevated ANA levels (1 : 160, ENA negative). Intracutaneous testing failed to show a positive serum factor (irradiation of the serum with 10 J/cm<sup>2</sup> UVA, corresponding to four times the MUD).

Besides physical and chemical UV protection, several-month-long therapeutic attempts were made using various antihistamines – also at increased doses – in combination with montelukast, chloroquine, and cyclosporine A. Therapeutic success was limited and did not last. Photohardening was infeasible due to frequent occupational engagements. We therefore initiated treatment with omalizumab at a dose of 300 mg every four weeks. As there was initially only minor subjective improvement (after three doses), we increased the omalizumab dose three times to (eventually)



**Figure 1** Phototesting with UVA<sub>1</sub> (26.2 J/cm<sup>2</sup>, 35.8 J/cm<sup>2</sup>, 52.4 J/cm<sup>2</sup>, 68.9 J/cm<sup>2</sup>, 80 J/cm<sup>2</sup>) and UVB (2 mJ/cm<sup>2</sup>, 6 mJ/cm<sup>2</sup>, 10 mJ/cm<sup>2</sup>, 15 mJ/cm<sup>2</sup>, 21 mJ/cm<sup>2</sup>, 25 mJ/cm<sup>2</sup>) showed marked urticarial reactions 20 minutes after irradiation.

450 mg every four weeks. On this regimen, there was significant improvement in pruritus, erythema, and wheals. The patient reported only minimal symptoms after being in the sun for half an hour. Phototesting confirmed the improvement, with erythema and minimal wheals above 35.8 J/cm<sup>2</sup> UVA and a minor response in the UVB range (Figure 2). Due to persistent effectiveness, we were subsequently able to reduce the omalizumab dose back to 300 mg every four weeks. Over the course of the 16-month treatment, the antibody was well tolerated.

The present case thus confirms previous case reports in which four solar urticaria patients showed a good response to omalizumab – partly with complete remission – at doses varying from 150 mg every four weeks to 400 mg every two weeks [3–5] (Table 1).



**Figure 2** Phototesting with UVA<sub>1</sub> (26.2 J/cm<sup>2</sup>, 35.8 J/cm<sup>2</sup>, 52.4 J/cm<sup>2</sup>, 68.9 J/cm<sup>2</sup>, 80 J/cm<sup>2</sup>) and UVB (2 mJ/cm<sup>2</sup>, 6 mJ/cm<sup>2</sup>, 10 mJ/cm<sup>2</sup>, 15 mJ/cm<sup>2</sup>, 21 mJ/cm<sup>2</sup>, 25 mJ/cm<sup>2</sup>) showed less pronounced urticarial reactions 20 minutes after irradiation on omalizumab 450 mg.

**Table 1** Case reports of omalizumab therapy in solar urticaria.

| Dose/interval          | Treatment duration            | Response                                           | Author                                                               |
|------------------------|-------------------------------|----------------------------------------------------|----------------------------------------------------------------------|
| Omalizumab 300 mg/ 4 w | At least 6 m                  | Complete remission n = 1                           | Metz M et al. [3]                                                    |
| Omalizumab 150 mg/ 2 w | At least 6 m                  | Complete remission n = 1                           | Metz M et al. [3]                                                    |
| Omalizumab 150 mg/ 4 w | 1 m [4]<br>4 m [6]<br>6 m [7] | Complete remission n = 1 [4]<br>No remission n = 2 | Güzelbey O et al. [4]<br>Duchini G et al. [6]<br>Müller S et al. [7] |
| Omalizumab 450 mg/ 2 w | 3 m                           | Partial remission n = 1                            | Waibel KH et al. [5]                                                 |

Abbr.: w, weeks; m, months

By contrast, there have been two other cases – a 24-year-old patient with UVA- and visible light-induced urticaria and a 64-year-old patient with UVA-induced solar urticaria – who, after 3–4 months, showed no improvement in symptoms on omalizumab 150 mg every four weeks [6, 7]. Possibly, the dose was too low. Dose-finding studies in chronic spontaneous urticaria have shown the most effective dose to be 300 mg every four weeks [8].

The mode of action of omalizumab in urticaria remains unclear. It is a recombinant humanized monoclonal IgG1<sub>kappa</sub> antibody that selectively binds to human immunoglobulin E (IgE), thus inhibiting the binding of IgE to the high-affinity receptor for the Fc region of IgE (FcεRI) on basophils and mast cells. This combined effect results in a significant reduction in the response to allergenic stimulation [9]. In solar urticaria, it is assumed that a chromophore in the skin or serum is altered by exposure to the individually relevant UV spectrum. In an IgE-mediated process, this chromophore then acts as mast cell activator [10].

In summary, our solar urticaria patient experienced marked subjective and objective improvement of his condition on omalizumab. Occasionally, a dose increase may be necessary to achieve a therapeutic response. We consider omalizumab an important therapeutic option for this type of physical urticaria as well.

#### Conflict of interest

None.

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