

Failure of omalizumab (Xolair®) in the treatment of a case of solar urticaria caused by ultraviolet A and visible light

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Summary

Key words:

omalizumab; solar urticaria; ultraviolet A; visible light

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Solar urticaria is a rare photodermatosis probably caused by a chromophore, that – if activated by light of a specific spectrum – binds to mast cell-bound IgE and elicits degranulation. In our patient an action spectrum in ultraviolet A and visible light range was found, in the autologous serum test the presence of a serum chromophore for the same action spectra could be demonstrated, which may underline this pathogenetic hypothesis. Symptoms did not improve using antihistamines and sun protection. Photo hardening was denied from the patient, immunosuppression and plasmapheresis were discussed but not considered. So a treatment with Omalizumab was started that recently was successfully used in 4 case reports. After 3 doses of Xolair® there was no changing in the phototesting results and after 4 doses no subjective improvement.

Accepted for publication:

10 August 2011

Conflicts of interest:

None declared.

Solar urticaria is a rare Immunoglobulin E (IgE)-mediated photodermatosis caused by a yet unidentified chromophore activated by light of a particular spectrum, presumably in the dermis and/or the serum. Assumably, binding of the activated chromophore to mast cell-bound IgE elicits degranulation (1).

The treatment of choice consists of sun protection and H1-antihistamines. Other treatment options are photo- or photochemotherapy hardening, plasmapheresis, cyclosporin A, and intravenous immunoglobulins. Recently, a case of successful therapy of solar urticaria with one single dose of omalizumab (Xolair®, Novartis Pharma, Basel, Switzerland) (2), a case of partial improvement after six doses (3), and two cases of complete symptom control after the first dose have been reported (4).

Case report

We present a 24-year-old patient who complained of a 3-year history of recurrent solar urticaria immediately after sun exposure (Fig. 1). Antinuclear antibodies panel and porphyrin profile were negative.

Phototesting was performed on the back using an increasing dose ranging from 3.6 to 9.0 J/cm² for ultraviolet A (UVA) and from 40 to 100 mJ/cm² for ultraviolet B (UVB). Phototesting for

visible light (VL) was performed during 10 min on the back at 10 cm with a slide projector using a filter for residual ultraviolet rays.

Phototesting with UVB, UVA and VL demonstrated an action spectrum in the UVA and VL range (Fig. 2). These results were supplemented by intradermal tests with autologous patient's serum previously irradiated with UVB (100 mJ/cm²), UVA (9 J/cm²) and VL (10 min, slide projector), respectively, which were positive at the UVA and VL test sites.

The patient did not react to UVB in the phototest nor in the irradiated autologous serum test. Symptoms did not improve using sunscreens, antihistamines and doxepine. The possibility of light hardening with UVA was considered, but the patient initially denied this treatment. Because of his young age, immunosuppressive drugs were not considered. Because of the positive skin tests with activated autologous serum, a treatment with plasmapheresis would have been an option. However, due to the time-consuming procedure and financial reasons, this was again denied. Therefore, treatment with omalizumab (Xolair®) was started. The total IgE was 77.1 kU/l, and the body weight was 63 kg. Therefore, a dose of 150 mg Xolair® s.c. every 4 weeks was chosen. We repeated the phototests after the third dose of omalizumab, and no change was observed compared with the

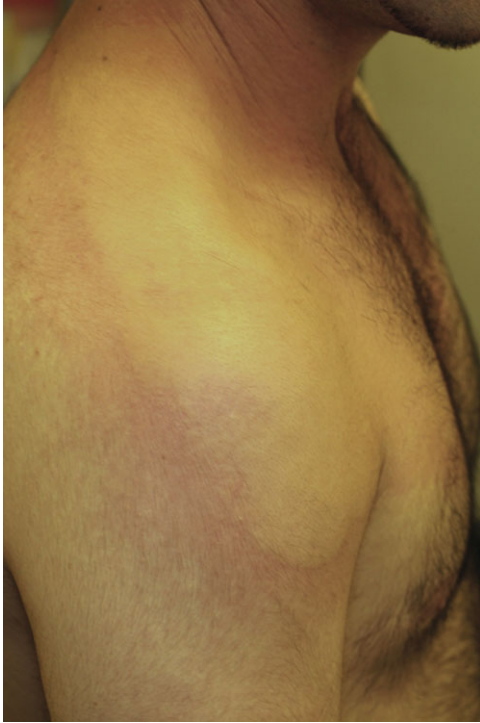


Fig. 1. Large urticarial wheals after exposure to sunlight.

baseline results. The patient did not report subjective improvement also after the fourth and last dose of omalizumab.

Conclusion

We report a case of solar urticaria triggered by UVA and VL, which are the most frequently described action spectra in the literature (5). These results were confirmed by the positive intradermal test with autologous serum which had previously been irradiated. This supports the hypothesis that a light-activated chromophore in the serum elicits mast cell degranulation after binding to IgE receptors.

Recently, four cases with complete or partial resolution of solar urticaria after treatment with omalizumab were published. The total IgE levels were between 22.0 and 851.0 KU/l.

In our case, after four doses of Xolair®, there was no subjective or objective improvement, and no changes were demonstrated in the phototests. The treatment of solar urticaria is an ongoing challenge. Omalizumab may represent an option if other therapies fail or are contraindicated. However, our case shows that this treatment is not always successful. Possibly, a higher dose and longer treatment would have been more effective.

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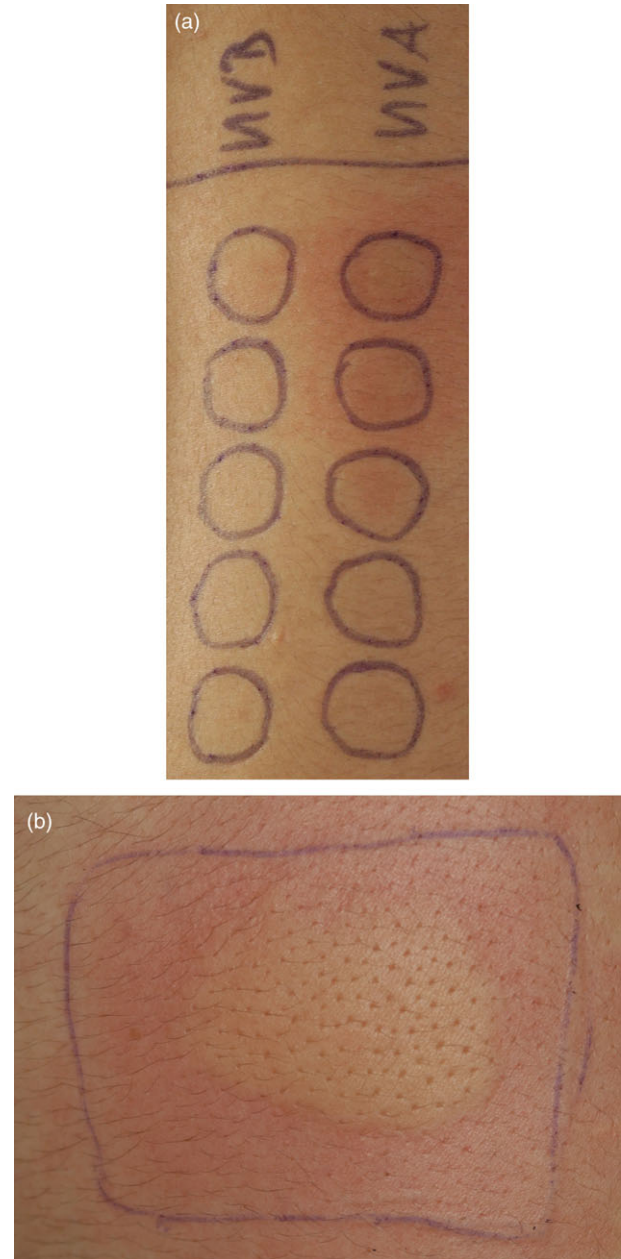


Fig. 2. (a) Wheals at the UVA test site after 10 min (minimal urticaria dose 6,3 J/cm²). (b) Wheal for visible light after 10 min irradiation (slide projector, equipped with a 415 nm edge filter).

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