Alopecia Areata after Omalizumab Treatment for Chronic Spontaneous Urticaria

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Omalizumab is the first targeted biologic therapy approved for the treatment of chronic spontaneous urticaria (CSU) (1). It binds selectively to human IgE, preventing binding of IgE to its high-affinity receptor (FcεRI), thus affecting the urticaria immunological cascade on several levels (2). Omalizumab has a very good safety and tolerability profile; the global pattern of side-effects and adverse events related to the drug is similar to that occurring in placebo-treated patients (3). The Summary of Product Characteristics (SmPC), reports some side-effects (muscle pain, joint swelling and hair loss), but there is insufficient data to estimate their frequency (4). We describe here a patient treated with omalizumab for CSU who developed alopecia areata (AA) after 14 weeks of first exposure.

CASE REPORT

A 27-year-old man with no significant medical history presented with a 7-month history of poorly controlled, antihistamine-resistant CSU. He had been treated previously in another dermatology centre with daily fexofenadine, 180 mg 4 times a day, and montelukast, 10 mg once a day, with only mild improvement. The laboratory examinations, whole blood count, liver function tests, renal function tests, serum electrolytes, erythrocyte sedimentation rate, CRP, thyroid function tests, C3 and C4, were normal, antinuclear antibodies were negative and total IgE was 74.9 kU/l (normal 2–78 kU/l). The 7-day urticaria activity score was 38.

Because high-dose antihistamines and montelukast did not improve the patient's condition, treatment with omalizumab was proposed at a single dose of 300 mg/month for 6 months. The patient improved significantly after the first dose, and from the second month onwards, switched from taking fexofenadine, 180 mg 4 times a day, to use it regularly on demand. However, after 14 weeks of omalizumab treatment, he noted patches of hair loss on his right frontal and temporal scalp (**Fig. 1**). He was seen in our clinic, the pull test at the periphery of the plaque was positive, and dermoscopy revealed black dots. AA was diagnosed clinically (the patient refused scalp biopsy). The patient characterized his hair loss as a major, distressing side-effect, significantly affecting his quality of life. He is currently being treated with intralesional triamcinolone acetonide, 5 mg/ml every 4 weeks.

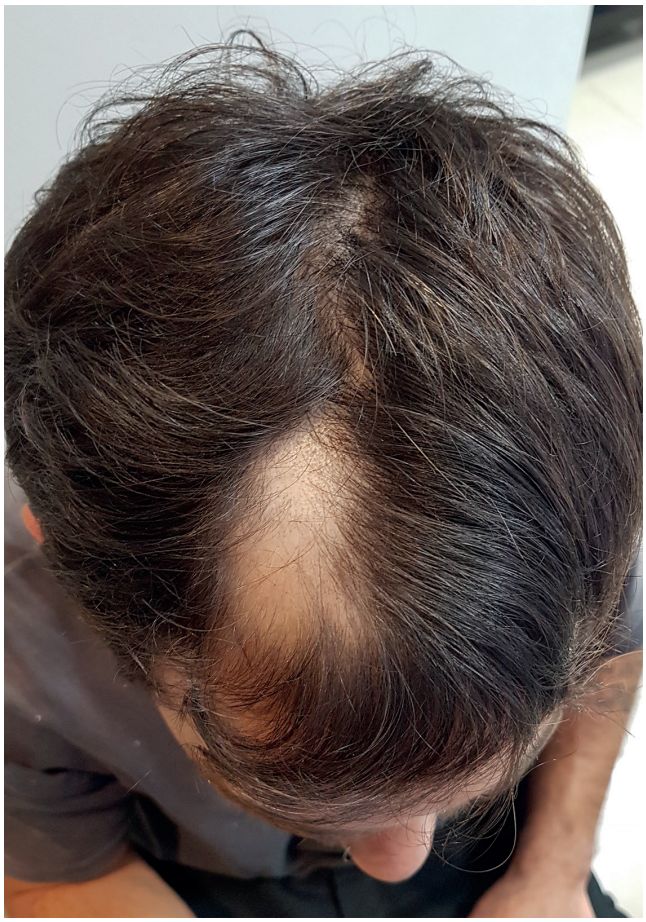

Fig. 1. Clinical photograph of alopecia areata after 14 weeks of treatment with omalizumab.

DISCUSSION

We report here a chronological relationship between omalizumab treatment and subsequent development of AA. Although a chronological relationship cannot be interpreted as causal, this option cannot be ruled out. There are no reports of AA in patients on omalizumab therapy in the existing medical literature. Although hair loss is listed among the side-effects in the SmPC of omalizumab, all previous reports related to diffuse and transient types of hair loss and not to AA (5, 6). AA represents a multifactorial autoimmune organ-specific disease and its pathogenesis involves T-cell autoantigens, type 1 T helper (Th1)/interferon-γ, Th2, PDE4, interleukin (IL)-23 and IL-9 pathways (7). It can be speculated that

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the downregulation of Th2 pathways might amplify the Th1 pathway and promote the development of AA with omalizumab use (8). Moreover, as mast cells have some impact on hair cycle regulation, downregulation of mast cell activity by omalizumab may play some pathophysiological role in drug-induced AA (9).

In conclusion, AA may be a dermatological adverse event of omalizumab use in patients with CSU.

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