

Base of Dermatology, Ministry of National Science and Technology, Hefei, Anhui, China; and ³Department of Dermatology, Affiliated Hospital of Jiujiang University, Jiujiang, Jiangxi, China
E-mail: wpg2370@163.com

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Chronic spontaneous urticaria in an 8-year-old girl treated with omalizumab

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Paediatric urticaria occurs in children at the ages of 6–11 years old. H1 antihistamines are the primary treatment of chronic spontaneous urticaria (CSU) in both children and adults.^{1,2}

An 8-year-old girl presented with a 16-month history of CSU refractory to 6 months of treatment with antihistamines (dosage increased four times), montelukast 5 mg/day and ciclosporin 5 mg/kg/day (minimum).

Physical examination showed evanescent coalescing weals 10–50 mm in diameter with sharp edges and arciform shape on the patient's trunk and limbs (Fig. 1a).

Laboratory investigations, including complete blood count, general biochemistry, C-reactive protein, erythrocyte sedimentation rate and thyroid function gave normal or negative results. Total serum IgE levels were 3281 IU/mL (normal levels < 100 IU/mL). Urticaria Activity Score in 7 days (UAS7) was 38 points.

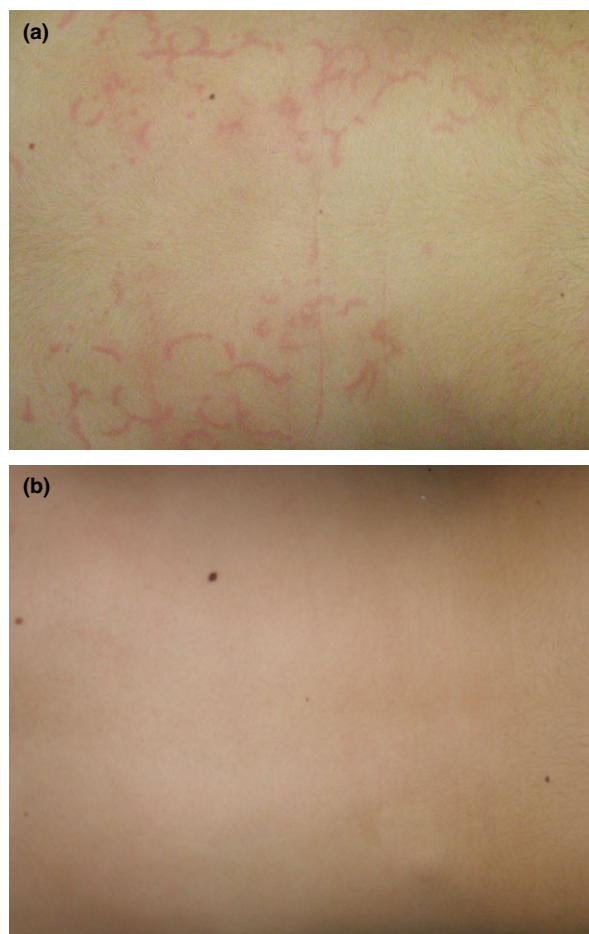


Figure 1 (a) Chronic spontaneous urticaria, presenting as weals with an arciform morphology affecting the patient's back; (b) excellent response to omalizumab after 1 month of treatment.

We started treatment with omalizumab at a dosage of 300 mg every 2 weeks, based on the current recommendations used for asthma. The patient showed an excellent response from the first dose, reaching UAS7 of 0 points within a month (Fig. 1b). After 6 months the dosage was decreased until it reached a maintenance dosage of 300 mg every 4 weeks, which is continuing at the moment. The patient remains in clinical remission, with good tolerance and without any adverse effects.

Omalizumab is approved by the US Food and Drug Administration and the European Medicines Agency (EMA) for use in CSU in patients > 12 years old. However, omalizumab is approved by the EMA for its use in bronchial asthma in the paediatric population from the age of 6 years.³ Chronic urticaria in the paediatric age group seems to have an autoimmune origin. Only

a small proportion of children with acute urticaria develop CSU, usually during adolescence.^{2,4} Thus, the need for treatment with omalizumab in children under the age of 12 years is rare. To our knowledge, only one previous report has described its use in CSU. Asero *et al.*⁵ described an 11-year-old boy with severe CSU and complete response after treatment with omalizumab, and reported no adverse effects with this treatment. In addition, recent publications describe the use of omalizumab in children < 12 years old with inducible chronic urticaria. In a recent review on treatment with omalizumab in children with various conditions mediated by IgE, the authors concluded that omalizumab has proven to be both safe and effective in asthma and atopic disorders, although the safety should be studied more carefully.⁴

The lack of response to conventional treatment for CSU in our patient and the good tolerance of omalizumab in children with asthma who are older than 6 years were the reasons for our choice of this drug in treating our patient.

In conclusion, controlled studies are needed on the use of omalizumab in children with CSU who are < 12 years old. However, omalizumab could be a safe and effective alternative in the treatment of patients with CSU who are < 12 years old and have not responded to first-line treatment.

L. Ossorio-García, D. Jiménez-Gallo, C. Albarrán-Planelles, C. Arjona-Aguilera and M. Linares-Barrios

Department of Dermatology, Hospital Universitario Puerta del Mar, Cadiz, Spain

E-mail: lidia.osga@gmail.com

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Imiquimod-induced vitiligo-like hypopigmentation after treatment for radiation keratosis

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Imiquimod is an immune response modifier with the chemical structure 1-(2-methylpropyl)-1H-imidazo [4,5-c] quinoline-4-amine. Imiquimod cream 5% is an immunomodulatory application that has been shown to be efficacious and has been approved for treatment of solar keratosis, superficial basal cell carcinoma and human papilloma virus (HPV)-induced genital warts.

The US Food and Drug Administration lists 68 reports of pigmentary changes out of a total of 1257 reports related to imiquimod lodged from 1997 to 2003: 51 of depigmentation, 7 of vitiligo, 17 of hyperpigmentation and 1 of hypopigmentation.¹ We report a further patient with vitiligo-like hypopigmentation caused by 5% imiquimod cream.

A 64-year-old man presented with plaques on his left hand. He had worked as a radiation engineer for 42 years.

On physical examination, several hyperkeratotic erythematous plaques were seen on the dorsal surface of the patient's left hand (Fig. 1a), and he was diagnosed by skin biopsy with multifocal radiation keratosis (Fig. 1b). In preference to surgery, imiquimod 5% cream was chosen as treatment, to be applied three nights per week. However, because of a severe inflammatory response at the application site (Fig. 1c), the patient discontinued the cream after 12 weeks.

At follow-up 4 months after stopping imiquimod, the patient reported hypopigmentation at the site of application. At the 60-month follow-up, the vitiligo-like hypopigmentation was still visible at the application site (Fig. 1d).

Histological examination of a skin biopsy specimen taken from the hypopigmented area was negative for radiation keratosis (Fig. 2a). Absence of melanin and melanocytes was confirmed by Fontana Masson stain (Fig. 2b), Schmorl method (Fig. 2c) and microphthalmia-associated transcription factor (MITF) (Fig. 2d).

We diagnosed this patient as having imiquimod-induced vitiligo-like hypopigmentation.