

Clinical and Experimental Dermatology / Early View

Letter to the Editor | [Full Access](#)

Successful treatment of severe recalcitrant vernal keratoconjunctivitis and atopic dermatitis associated with elevated IgE levels with omalizumab

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Abstract

We report the clinical case of an atopic patient with important manifestations impacting the quality of life at the cutaneous and ocular site. The condition resisted conventional treatments, and omalizumab had to be used to treat the disease successfully.



Dear Editor,

Atopic dermatitis (AD) is a chronic inflammatory skin condition, defined as extrinsic when

associated with elevated serum IgE levels and sensitization to various allergens.¹ It is linked to multiple allergic disorders, including conditions affecting the eyes. Of these, vernal keratoconjunctivitis (VKC) is one of the most serious, as it can lead to corneal scarring and vision loss if not treated properly. Treatment of VKC includes antihistamines for mild to moderate forms, while for more severe forms, eye drops containing steroids and tacrolimus, and eventually systemic steroid therapy or immunosuppressive agents such as systemic ciclosporin, may be used.² AD medications are represented by topical treatments with steroids and tacrolimus for mild to moderate forms and immunosuppressant agents such as systemic ciclosporin and biological drugs such as dupilumab.² However, dupilumab can worsen the ocular disease by superimposing conjunctivitis, a known adverse effect of this drug.³ Both VKC and AD are characterized by an immunological IgE-related cascade, and there have been several cases of IgE-mediated pathologies successfully treated with the use of omalizumab. Omalizumab acts by binding specifically to circulating human IgE, preventing its binding to the FcεR1 receptor on mast cells and basophils, thus inhibiting mast cell degranulation.⁴ In this case report, we describe a case of severe AD and VKC successfully treated with omalizumab.

A 13-year-old boy, originally from Pakistan, presented with skin desquamation associated with painful visual decrease, eye redness and diffuse itch, which had started several months previously. His medical history included obesity and metabolism disorders, diagnosed following scheduled visits to the auxological centre. The rest of the medical history was not relevant.

Physical examination revealed patches of skin affected by redness, surmounted by crusts and scratch marks, and concurrent dryness and marked lichenification on the limbs, including the hands (Fig. 1), trunk and periocular area (Fig. 2a).





Figure 1

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(a,b) Patient's hands (a) before any treatment, and (b) showing complete remission of eczematous manifestations after 4 weeks of omalizumab.

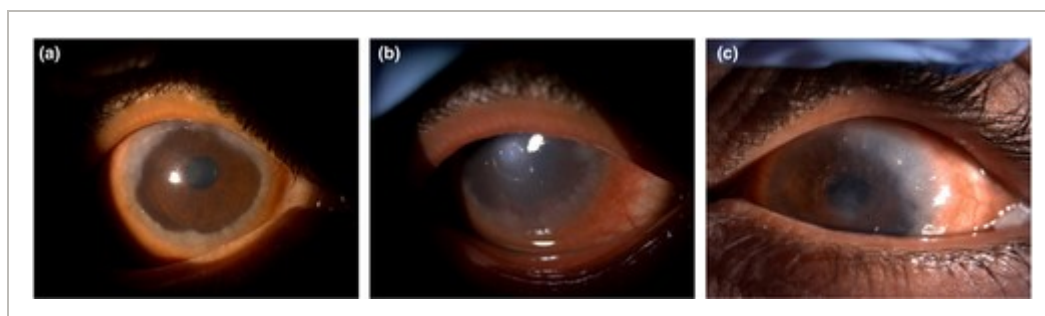


Figure 2

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(a–c) Slit-lamp biomicroscopy of the anterior segment showing (a) diffuse corneal de-epithelialization and limbal hyperplasia with Horner–Trantas dots at the first visit before any treatment; (b) marked worsening despite 3 months of topical and oral ciclosporin cycle therapy, with a central shield ulcer on the cornea and limbal neovascularization; and (c) resolution of the corneal ulcer and de-epithelialization, and disappearance of the new vessels, while some Trantas dots remained after 3 months of omalizumab. There was also improvement in the ocular symptoms.

As the patient fulfilled the London criteria for AD,⁵ with a total score of 32 as measured by the SCORing Atopic Dermatitis (SCORAD) instrument, a clinical diagnosis of AD was made, and VKC suspected.

Ocular examination showed that the patient had severe bilateral keratoconjunctivitis characterized by diffuse conjunctival injection, upper tarsal giant papillae, perilimbal Horner–Trantas dots, and macroerosions of the corneal epithelium. These features confirmed the diagnosis of VKC.²

Laboratory investigations revealed very high levels of total IgE (4555 kU/mL; normal level < 120 kU/mL), but IgE panels for inhalants and aliments did not exhibit specific allergies. The rest of the blood investigations were unremarkable.

We started a topical treatment with hyaluronic drops, ketotifen, corticosteroid and 1% ciclosporin eye drops and 0.1% betamethasone valerate ointment for the skin lesions. Due to no response to topical therapy alone, oral ciclosporin 5 mg/kg per day was added. After 1 month, there was complete resolution of skin disease but a worsening of the VKC (Fig. 2b). Thus, subcutaneous omalizumab 300 mg once monthly was started, and after 4 weeks, the patient reported benefits with a global reduction of symptoms. After 3 months, we observed

complete clearance of the skin conditions (SCORAD 0) (Fig. [1b](#)) and a significant improvement in clinical ocular signs and symptoms (Fig. [2c](#)). The patient is still receiving therapy at the same dose regimen with benefit.

In conclusion, this case shows the efficacy of omalizumab for the treatment of severe VKC associated with manifestations of extrinsic AD.

Acknowledgement

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