Novel treatment (new drug/intervention; established drug/procedure in new situation)

Omalizumab for severe atopic keratoconjunctivitis

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Summary

A 35-year-old patient received omalizumab (300 mg twice a month) for 2 years for a severe atopic keratoconjunctivitis (AKC) in order to reduce the risk for steroid-induced keratitis. After 4 months, quality of life and ocular symptoms improved, and the use of oral steroids was strongly reduced. The treatment was well tolerated. This observation suggests that omalizumab may be a good option for the treatment of severe AKC, especially to avoid side effects of immunosuppressive treatments, as suggested for other allergic diseases. Specific trials should be designed for allergic eye diseases.

BACKGROUND

Atopic keratoconjunctivitis (AKC) is a severe disease, sometimes leading to visual loss. Immunosuppressive treatments may induce severe side effects. Omalizumab is probably a promising and safe therapy for severe allergic diseases other than asthma. However, specific studies have to be designed to assess the role of anti-immunoglobulin E (IgE) treatment in severe ocular allergic diseases. Eye specialists should be aware of such possibilities.

CASE PRESENTATION

A white 35-year-old patient presented to the outpatient clinic for uncontrolled asthma associated with severe AKC, rhinitis and eczema.

The patient suffered from a severe bilateral AKC, which started when he was 13 years old. The patient was born in Southern Africa and came back to France when he was 5 years old. Perennial ocular symptoms gradually increased, requiring wide local treatment. Despite scleral lenses, topical steroids and ciclosporin, tacrolimus on the eyelids, as well as oral tetracycline and H1-antihistamine, the patient required a continuous low dose of oral steroid (5 mg/days), with frequent pulses during seasonal exacerbations. He was unable to go out from April to September because of ocular pain and recurrent keratitis, and consequently lost his job. The patient was sensitised to d-pteronyssinus (0.41 kU/l), Timothy grass (48.9 kU/l), wheat (20.7 kU/l) and plane tree (0.76 kU/l). Total IgE level was 200 UI/ml.

The patient also showed persistent rhinitis, eczema and persistent asthma. Asthma control was poor (Asthma Control Test of 16/25), probably due to little observance of inhaled steroids treatment. Lung function tests were normal.

TREATMENT

Because of ocular disease-induced severe handicap, and to avoid the risk of steroid-induced keratitis, the patient received omalizumab (300 mg subcutaneous twice a month). The treatment was started in February, for a maximal effect expected during the grass pollen season. The treatment was well tolerated.

OUTCOME AND FOLLOW-UP

Conjunctivitis rapidly improved, with decrease in ocular pain and inflammation (figure 1). The patient could be weaned from continuous oral steroid therapy 1 month later. During the first pollen season, he required oral prednisone for few days only and was able to go out about 4 days a week. During the first year of treatment with omalizumab, total prednisone consumption was reduced from 2000 mg to less than 100 mg. The patient recognises that his quality of life had improved. Asthma control score also rapidly improved, as well as eczema and rhinitis.

DISCUSSION

Clinical expression of AKC involves conjunctiva, eyelids and cornea, with a wide spectrum of symptoms such as intense itching, tearing and redness. In the most severe forms, corneal damage can lead to visual loss.¹

AKC is a complex chronic inflammatory disease of the ocular surface. Frequent association with other allergic manifestations, high serum and tear IgE levels, and presence of positive FCR1-mast cells in the conjunctiva suggest that the disease is mediated by immediate hypersensitivity. Both conjunctival epithelial cells and inflammatory cells infiltrating conjunctival tissues (eosinophils, T lymphocytes, mast cells, basophils) are responsible for the secretion of both Th1 and Th2 cytokines that induce progressive remodelling of the conjunctival connective tissue, leading to mucus metaplasia, conjunctival thickening, neovascularisation and scarring, responsible for the corneal complications of the disease.²

Topical antihistamines combined with mast cell stabilisers are the cornerstone of the ocular allergy treatment, but more aggressive treatments such as topical or systemic immunosuppressive drugs (steroids, tacrolimus, ciclosporin A) may be required in the most severe forms. However, such treatments may have serious side effects. Omalizumab, a monoclonal anti-IgE antibody, is successfully used for the treatment of persistent atopic asthma,³ with few side effects. Omalizumab has also shown beneficial effects in other IgE-mediated diseases, such as atopic dermatitis,

Figure 1 Left eye after treatment, without any corneconjunctival inflammation.

urticaria, eosinophil-associated gastrointestinal diseases and seasonal rhinoconjunctivitis.⁴ The costs of the treatment do not support the use of omalizumab for ordinary allergic rhinoconjunctivitis, but its use has been proposed for the treatment of severe ocular allergy,⁵ despite no clinical data at this time being available, except for an unpublished series of six patients.⁶ This observation suggests that omalizumab may be a good option for the treatment of severe AKC, especially to avoid side effects of immunosuppressive treatments. Specific trials should be designed for allergic eye diseases.

Learning points

- ▶ AKC, in the most severe forms, can lead to visual and social handicap.
- ▶ AKC is a complex chronic inflammatory disease of the ocular surface, associated with local IgE production and features for immediate hypersensitivity.
- ▶ Omalizumab may be a good option for the treatment of severe AKC.

Competing interests None.

Patient consent Obtained.

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