

REVIEW OF ALLERGIC CONJUNCTIVITIS

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ABSTRACT

Eye allergies are one of the most common conditions faced by allergists and ophthalmologists. Allergic conjunctivitis is often underdiagnosed and therefore undertreated. Basic and clinical research has improved our understanding of the cells, mediators, and immunological events that occur in ocular allergy. New drugs have improved the effectiveness and safety of ocular allergy treatment. Understanding the immune mechanisms, clinical features, differential diagnosis, and treatment of ocular allergies may be of benefit to all professionals working with these patients. The purpose of this review is to focus on all forms classified as ocular allergy, including seasonal allergic conjunctivitis, perennial allergic conjunctivitis, spring allergic conjunctivitis, atopic keratoconjunctivitis, contact allergy, and giant papillary conjunctivitis. A systematic review of the literature.

KEYWORDS: Allergy, Conjunctivitis, Diagnosis, Symptoms, Treatment.



INTRODUCTION

Allergic diseases have increased dramatically in recent decades. Ocular allergy is one of the most common eye diseases encountered in clinical practice. No single cause for this increase can be identified, so experts consider the contribution of a number of factors, including genetics, urban air pollution, pets, and childhood exposures.^[1] As more people require treatment for allergies, the associated costs have also increased significantly. Eye allergies can themselves cause irritating symptoms, and severe conditions such as atopic keratoconjunctivitis can eventually lead to vision loss. Allergic conjunctivitis is a general term that includes seasonal allergic conjunctivitis (SAC), perennial allergic conjunctivitis (PAC), vernal kerato conjunctivitis (VKC), and atopic kerato conjunctivitis (AKC). However, despite some common allergic markers, AKC and VKC have significantly different clinical and pathophysiological characteristics from his SAC and PAC.^[4] Contact lenses and prosthetic eyes associated with giant papillary conjunctivitis (GPC) are also often included in the group of ocular allergies. However, these should not be considered true allergic diseases, but rather chronic ocular microtrauma-related diseases that must be treated by an ophthalmologist in collaboration with a contact lens specialist.^[3]

Understanding the immune mechanisms, clinical features, differential diagnosis, and treatment of ocular allergies may be of benefit to all professionals working with these patients. To achieve this objective, we systematically reviewed the literature and focused on all forms classified as ocular allergy.

Prevalence Rate

Allergic conjunctivitis is a very common ocular disorder in patients and is frequently overlooked, misdiagnosed, and undertreated. Allergic conjunctivitis is prevalent in 6%–30% of the general population.

Classification of Acute Tonsilitis

Allergic conjunctivitis is an inclusive term that encompasses.

- 1) Seasonal allergic conjunctivitis
- 2) Perennial allergic conjunctivitis
- 3) Vernal keratoconjunctivitis,
- 4) Atopic keratocongiuntivitis

Aetiology

Seasonal allergic conjunctivitis (hay fever conjunctivitis) is caused by airborne mold spores and pollen from trees, grass, and weeds. Depending on the life cycle of the offending plant, it tends to peak in the spring, late summer, or early fall and disappear during the winter.

Perennial allergic conjunctivitis (atopic conjunctivitis, atopic keratoconjunctivitis) is caused by dust mites, animal dander, and other nonseasonal allergens. These allergens, especially those present in the home, tend to cause symptoms year-round.

Vernal keratoconjunctivitis is a more severe form of conjunctivitis and is most likely caused by allergies. It most commonly occurs in men between the ages of 5 and 20 who have eczema, asthma, or seasonal allergies. Vernal keratoconjunctivitis usually recurs each spring and resolves in the fall and winter. Many children outgrow this condition as adults.

**Diagnosis**

The diagnosis of ocular allergy is primarily clinical, but there are laboratory tests that can be useful in supporting the diagnosis. Allergists can perform skin testing for specific allergens by scratch tests or intradermal injections of allergen. In-vitro tests for IgE antibodies to specific allergens are widely used. Allergic tests would help in differentiating intrinsic and extrinsic forms and would, therefore, be helpful in the treatment.^[5]

Treatment

The primary behavioral change in all types of allergic conjunctivitis is avoidance of the provoking antigen. However, because the eyes have a large surface area, eye exposure to airborne allergens is often unavoidable. Artificial tear substitutes are responsible for the barrier function and help improve the initial defense at the level of the conjunctival mucosa.

These drugs help to dilute various allergens and inflammatory mediators that may be present on the ocular surface and help clear the ocular surface of these drugs. If symptoms are not sufficiently relieved by avoiding non-pharmacological strategies, pharmacological treatments can be applied locally or administered systemically to reduce allergic reactions.

The mainstay of ocular allergy treatment is the use of antiallergic drugs, such as antihistamines, multi-acting antiallergic drugs, and mast cell stabilizers. For example, the topical H1 antihistamine levocabastine hydrochloride rapidly reduces ocular inflammation when administered topically to the eye. Topical antihistamines competitively and reversibly block histamine receptors, reducing itching and redness, but their effects are only short-term. These drugs have no effect on other proinflammatory mediators, such as prostaglandins and leukotrienes, which remain uninhibited. Due to their limited duration of action, they must be administered frequently, up to four times per day, and topical antihistamines can cause eye irritation, especially with long-term use. Combined treatment with decongestants and antihistamines has been shown to be more effective and is administered as eye drops up to four times a day. Decongestants act primarily as vasoconstrictors and effectively reduce erythema. However, side effects include burning or stinging during infusion, mydriasis, and rebound hyperemia and drug-induced conjunctivitis when used chronically.^[8] Therefore, these treatments are only suitable for short-term symptom relief and are not recommended for use in patients with angle-closure glaucoma.

The mechanism of action of mast cell stabilizers is unknown. They may increase calcium influx into cells to prevent membrane changes and/or reduce membrane fluidity prior to mast cell degranulation. As a result, mast cell degranulation is reduced and the release of histamine and other chemotactic factors present in already formed and newly formed states is prevented. Mast cell stabilizers do not alleviate existing symptoms and can be used prophylactically to prevent mast cell degranulation due to subsequent allergen exposure. Mast cell stabilizing drugs can also be used topically in the eye and may be appropriate for more severe conjunctivitis. They require a loading period that must be applied before antigen challenge. Therefore, poor compliance must be considered as a potential disadvantage. In recent years, several multimodal antiallergic drugs have been introduced, such as olopatadine, ketotifen, azelastine, epinastine, and bepostatin, which have effects such as histamine receptor antagonism, stabilization of mast cell degranulation, and inhibition of mast cell

activation and infiltration. It exerts multiple pharmacological effects such as inhibition. Due to eosinophils. Ketotifen inhibits eosinophil activation, leukotriene formation, and cytokine.

Azelastine is a second generation selective H1 receptor antagonist that inhibits platelet activating factor (PAF) and also acts by blocking the expression of intercellular adhesion molecule 1 (ICAM-1).^[41] Epinastine acts on both H1 and H2 receptors (the latter action may be effective in reducing eyelid swelling) and also has mast cell stabilizing and anti-inflammatory effects.

These drugs are becoming the agents of choice for immediate symptom relief in patients with allergic conjunctivitis.

Anti-inflammatory drugs are used if the above anti-allergic drugs are not sufficient to control the inflammatory process of allergies. Nonsteroidal anti-inflammatory drugs (NSAIDs) can be used as adjuncts to reduce conjunctival hyperemia and pruritus, especially those associated with prostaglandin D2 and prostaglandin E2.

Corticosteroids are among the most effective drugs used for more severe forms of ocular allergy and are also effective in treating acute and chronic forms of AC. Corticosteroids have immunosuppressive and antiproliferative properties because they can interfere with the transcription of Th2-derived cytokine genes and transcription factors that regulate the differentiation of activated T lymphocytes into Th2 lymphocytes. These have some limitations, including negative effects on the eyes, such as: B. Delayed wound healing, secondary infection, increased intraocular pressure, and cataract formation. Therefore, these treatments are suitable for short courses (up to 2 weeks). However, if needed for a long period of time, an eye exam should be performed, including a baseline assessment for cataracts and intraocular pressure measurements.^[7] The effectiveness of immunotherapy against ocular symptoms caused by conjunctival antigen challenge was first demonstrated in 1911, and this proven method can be considered for long-term control of AC. Some recent studies have focused on nasal symptoms rather than eye symptoms, while others have confirmed the effectiveness of immunotherapy for eye symptoms.^[12]

Allergen-specific immunotherapy is an effective treatment for patients with allergic rhinoconjunctivitis who have specific IgE antibodies against allergens. The main goal of this treatment is to induce clinical tolerance to a specific allergen. This reduces the seasonal

increase in IgE specific to that allergen and increases the production of specific IgG4 and IgA. These effects are mediated by increased production of IL-10 and TGF- β 1.^[6]

However, the immune response to the administration of an allergen is not an indicator of the effectiveness of the treatment, and the treatment itself can cause systemic reactions, the frequency and severity of which depend on the type of allergen administered. Traditionally, immunotherapy is administered by subcutaneous injection. However, sublingual (oral) immunotherapy (SLIT) is gaining popularity among allergists. SLIT requires further evaluation for ocular allergy relief. It has been shown to control eye symptoms and symptoms, although eye symptoms may be less responsive than nasal symptoms. Oral antihistamines are often used to treat allergic symptoms of the nose and eyes. These newer second-generation antihistamines are preferred over first-generation antihistamines because they are less likely to cause side effects such as drowsiness. However, second-generation antihistamines can cause dry eyes, which can compromise the tear film's protective barrier and also worsen allergy symptoms. Therefore, it has been suggested that a combination of eye drops may be more effective in treating allergic eye symptoms. Although intranasal corticosteroids are highly effective in treating the nasal symptoms of allergic rhinitis, evidence is conflicting that they may also be effective in treating ocular symptoms.^[9]

Pediatric Allergic conjunctivitis

Allergic conjunctivitis is common in childhood, peaking in late childhood and adulthood. Patients often have other atopic diseases such as eczema, asthma, and most commonly rhinitis. Symptoms include bilateral lesions, pruritus, lacrimation, mucus discharge, erythema, mild lid edema, and edema. AKC and VKC occur less frequently but can be more severe. Therefore, involvement of a pediatric ophthalmologist may be necessary to prevent avoidable vision loss in severe cases.^[10]

Ayurvedic View

In Ayurveda, Simple Allergic Conjunctivitis can be linked to Vataja and Kaphaja Abhishyanda. It is characterized by symptoms such as Toda (Pricking pain), Sangharsha (foreign body sensation), Achchashruta (watery discharge), Alpa Shopha (mild chemosis), Vishushka Bhava (feeling of dryness), and Parushya (dryness).

CONCLUSION

The term “allergic conjunctivitis” is an umbrella term that encompasses a variety of clinical entities and is based on the assumption that the classic type I hypersensitivity mechanism is responsible for all clinical forms of allergic eye disease. However, IgE and non-IgE-mediated mechanisms are involved in the development of allergic eye diseases. A large number of mediators, cytokines, chemokines, receptors, proteases, growth factors, intracellular signals, regulatory and inhibitory pathways, and other unknown factors and pathways are differentially expressed in different allergic diseases, leading to different clinical aspects, yield diagnostic features and response to treatment. Therefore, a new classification system is desired, preferably derived from the diverse pathophysiological mechanisms that operate in different forms of ocular allergy.

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