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# ALLERGIC CONJUNCTIVITIS: A COMPREHENSIVE REVIEW OF THE LITERATURE

Ophthalmology

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KEYWORDS

## INTRODUCTION

Ocular allergies encompass a group of hypersensitivity disorders to normally harmless substances, known as allergens and can be observed as the only dominant presentation of an allergic sensitisation, or are associated with rhinitis, asthma, atopic dermatitis or food allergy. [1] Allergic conjunctivitis is an inclusive term that encompasses seasonal allergic conjunctivitis (SAC), perennial allergic conjunctivitis (PAC), vernal keratoconjunctivitis (VKC), and atopic keratoconjunctivitis (AKC). However, AKC and VKC have clinical and pathophysiological features quite different from SAC and PAC, in spite of some common markers of allergy.[2] Ocular allergy can involve all of the components of the ocular surface, including the lid and lid margin, conjunctiva and the lacrimal system. Corneal involvement is typically restricted to the two most severe forms of ocular allergy, vernal keratoconjunctivitis (VKC) and atopic keratoconjunctivitis (AKC), which requires particular care in their management. [3] Besides causing irritating symptoms and affecting vision of the patient, it also affects the quality of life and poses an economic burden on the patient. The purpose of this review article is to provide a comprehensive overview of the classification, epidemiology, signs, symptoms, and pathophysiology and treatment options available for allergic conjunctivitis.

### Classification And Nomenclature:

Allergic conjunctival disease is classified into multiple disease types according to the presence or absence of proliferative changes, complicated atopic dermatitis, and mechanical irritation by foreign body [10] or on pathophysiology, according to the different hypersensitivity mechanisms introduced by Gell and Coombs. In 2001, the European Academy of Allergy and Clinical Immunology (EAACI), introduced a revised nomenclature that proposed a distinction between allergic and non allergic hypersensitivity reactions: allergic diseases were further divided into IgE- and Non-IgE-mediated hypersensitivities.[4]

### Epidemiology:

Approximately15–20% of the world population is affected by some form of allergic disease; [4] ocular symptoms are estimated to be present in 40–60% of allergic patients.[6] 30% of the population in industrialized countries has such as atopic dermatitis, asthma, rhinitis, or conjunctivitis.[5] The prevalence is greater in Western countries than in Asia or Africa. [8]Approximately 20% of the population of United States are affected with ocular allergy. [7]

### Pathophysiology And Clinical Entities:

Seasonal and perennial allergic conjunctivitis-

Seasonal allergic conjunctivitis (SAC) is the most common form of all ocular allergy disease, and is usually triggered by exposure to airborne pollens produced by plants that cause hay fever, the signs and symptoms typically occurring in spring and summer. The pathomechanism involves an IgE-mediated type-I hypersensitivity, with the early response clinically lasting for 20-30 min.

Perennial allergic conjunctivitis (PAC)is milder than SAC, and is a chronic condition that occurs throughout the year. [1]

### Vernalkeratoconjunctivitis-

VKC is a potentially severe, chronic, allergic condition, causing bilateral recurrent inflammatory disorder of the conjunctiva and cornea (Fig.1). [9] VKC has 3 clinical forms- limbal, palpebral and mixed. Large papillae of different shape and size, usually greater than 1 mm in diameter, on the upper tarsal conjunctiva characterize the tarsal form (Fig. 2), while Trantas' dots and infiltrates on the limbus are typical of the limbal form. [18]VKC is characterized by infiltration of the conjunctiva by a variety of inflammatory cell types, especially eosinophils. Although VKC has previously been thought of as an IgE-mediated disease several other immunologic pathways have also been implicated. Patients with VKC have been shown to have an increased number of activated CD4+ T-lymphocytes, predominantly Th2, indicating that there is a hypersensitivity reaction to an unknown pathogen Increased levels of inflammatory cytokines IL-3, IL-4, and IL-5 have also been demonstrated.[16] This chronic limbal inflammation probably affected the microenvironment of the stem cells, resulting in poor stromal support. When pathologic insults destroy the limbal stem cells, the corneal surface invariably heals with conjunctival epithelial ingrowth, neovascularization, chronic inflammation, and/or recurrent epithelial defects constituting LSCD. [17] Although the disease is most often self-limiting, and will often resolve after puberty. [16] Shield ulcer is an uncommon, incapacitating corneal manifestation that occurs in 3 to 11% of patients suffering from vernal keratoconjunctivitis.[14]

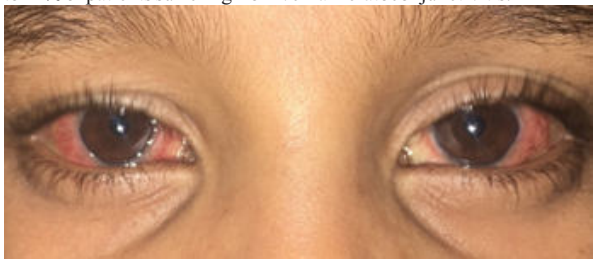

**Fig.1** Bilateral Conjunctival Hyperaemia In Vernal Keratoconjunctivitis.

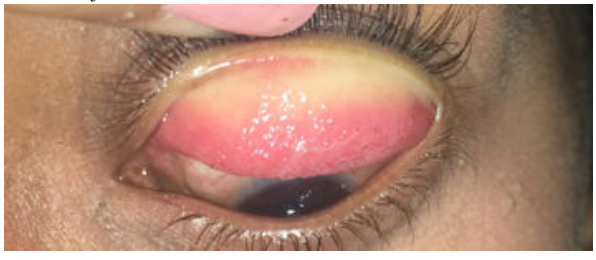

**Fig.2** Palpebral conjunctival papillae

Fig.3 Limbal conjunctiva Horner-Trantas dots.

### Atopic keratoconjunctivitis-

AKC in children is defined as the presence of severe allergic conjunctivitis with atopic dermatitis that is diagnosed before 16 years of age. [11]The immunopathogenesis of AKC is not fully understood, but involves the release of different cytokines by the effector cells( mast cells, eosinophils, T-cells, conjunctival epithelial cells).[15] The clinical signs are represented by the hyperaemia of conjunctiva and episcleral vessels, papillae on upper tarsal conjunctiva, and the presence of comcomitant blepharitis.[22]

### Giant papillary conjunctivitis-

Giant papillary conjunctivitis (GPC) has been reported as a complication of contact lens wear since 1974. It is characterised by papillary hypertrophy of superior tarsal conjunctiva.

Prior to the wide spread use of contact lens, this reaction was predominantly seen in patients with immunoglobulin E (IgE) mediated ocular allergies including allergic conjunctivitis and vernal keratoconjunctivitis (VKC).[12]

**Treatment options:**

**Supportive –**

Supportive therapy includes artificial tears, goggles and cold eye pads. Artificial tears dilute the concentration of allergens and inflammatory mediators and flush the ocular surface of these agents. Goggles decrease the amount of allergen reaching the ocular surface while cold eye pads offer for symptomatic relief. Avoidance of allergen should be tried wherever possible.[20]

**Medical –**

1. Antihistamines- Antihistamines act via histamine receptor (HR) antagonism to block the inflammatory effects of endogenous histamine and prevent or relieve the associated signs and symptoms. [18] Topical antihistamines, such as levocabastine and emedastine alleviate ocular allergy quickly by binding to histamine receptors but are only effective in mild cases.[21]

2. Mast cell stabilisers- Mast cell stabilizer inhibits the degranulation of mast cells and Suppress release of mediators (e.g.histamine, leukotriene, thromboxaneA2), consequently, the early phase reaction to type I allergy is inhibited, and conjunctival local infiltration of inflammatory cells is curtailed, resulting in a reduction of the late phase reaction.[10]

3. Dual acting agents- New antihistamines that combine mast cell stabilizing properties and histamine receptor antagonism, such as alcaftadine, azelastine, bepotastine, epinastine, ketotifen, and olopatadine, are presently available and show evident benefits in treating all forms of ocular allergy. [19] These agents are well tolerated and are not associated with any oclular side effects.

4 Non-steroial anti-inflammatory drugs- These drugs block the cyclooxygenase pathway; as a result, they reduce prostaglandin and thromboxane synthesis. The marketed topical agents (0.5% ketorolac, 0.1% diclofenac, 0.5% pranoprofen and 0.03% flurbiprofen) have demonstrated their efficacy in a metaanalysis, in application to itching and conjunctival hyperemia versus placebo.[24]

5. Corticosteroids- Although topical corticosteroids are highly effective in relieving the signs and symptoms of acute flare up of allergic conjunctivitis, their prolonged use can result in unwanted and serious sequelae. [23]

6. Immunomodulators- Cyclosporine and tacrolimus are the two calcineurin inhibitors that block the IL-2 induced inhibition of TH-2 cells and inhibit histamine release from the mast cells. [23]

**Surgical –**

When medical therapy remains ineffective, procedures like excision, cryocoagulation, excision with Mitomycin-0.02% or CO2 laser are used to manage papillary hypertrophy. Supratarsal corticosteroid injections, topical cyclosporine, cryotherapy, surgical or laser assisted excision of giant papillae with or without mitomycin, excimer laser phototherapeutic keratectomy, amniotic membrane graft and cultivated corneal epithelial cells transplant, have been tried for nonhealing shield ulcer with varying degrees of success.[14]

**Experimental –**

Researches in VKC and SAC confirm the role of AMCase in human conjunctival allergic pathologies and the measurement of AMCase showed a sensitivity and specificity of 100 % respectively, addressing the use of AMCase assay in the biochemical diagnosis of VKC and SAC. The activity of AMCase was significantly high in VKC. The role of AMCase in allergic diseases is suggestive of new possibilities for its control, using specific inhibitors of AMCase or modulating its expression.[13]

**CONCLUSION –**

The clinical diagnosis of allergic conjunctival disease is challenging due to a wide range of overlapping entities that might respond differently to conventional therapy. Treating patients with severe ocular allergy requires adequate knowledge of the immunopathologial mechanisms involved the use of novel treatments and the involvement of an interdisciplinary treatment group.

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