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CASE SERIES ON VARIOUS PRESENTATIONS OF ALLERGIC CONJUNCTIVITIS.

Dermatology

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ABSTRACT

ocular allergy represents the most common condition encountered by ophthalmologists. Allergic conjunctivitis is often under diagnosed and undertreated. An understanding of the immunologic mechanisms, clinical features, differential diagnosis and treatment of ocular allergy may be useful to all who deal with these patients. Here, I am briefing the presenting features of allergic conjunctivitis, dermatological manifestations of atopic dermatitis in atopic conjunctivitis patients and few follow ups showing marked improvement with treatment.

KEYWORDS

presentation, symptoms, allergic conjunctivitis, periorbital swelling, papillae, dermatitis

INTRODUCTION:

Allergic conjunctivitis is an umbrella term used for all types of allergic conjunctivitis that includes seasonal allergic conjunctivitis (SAC), perennial allergic conjunctivitis (PAC), vernal keratoconjunctivitis (VKC), atopic keratoconjunctivitis (AKC), contact allergy and giant papillary conjunctivitis (GPC) [1]. Allergic conjunctivitis cases have increased in last decades but a single cause of this increase cannot be pinpointed because there is contribution of numerous factors like pollen grains, animal dander, house dust mite, genetics, air pollution in urban areas, pets, and early childhood exposure. Symptoms include ocular itching, swelling, discharge and photophobia. Itching may be quite incapacitating and severe [2]. An understanding of the cause, prevention of exposure, treating the cause and early treatment lead to better visual rehabilitation.

Cases:

6 years old male child presented with 2 month history of bilateral swelling around eyes with itching and watering. There was history of allergy to dust, sneezing, shortness of breath. On examination there was bilateral periorbital swelling, conjunctival chemosis with conjunctivochalasis, papillae on upper tarsal conjunctiva of both eyes (Figure A). He was diagnosed as a case of seasonal allergic conjunctivitis and started on topical antihistaminic, topical lubricants along with oral antihistaminic for his systemic symptoms and show marked improvement (Figure B). Bilateral Periorbital edema in a pediatric age group must rule out nephrotic syndrome, as in our case renal function test were absolutely normal and thus the case highlights the importance of clinical examination. Seasonal allergic conjunctivitis mostly present during spring and summer.

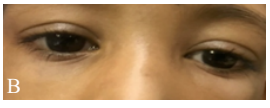



Figure A:- shows periorbital swelling, conjunctival chemosis, Figure B after treatment there is resolution of periorbital edema and conjunctival chemosis with residual left lower eyelid edema.

Case2:

9 years old male child not able to open his eyes because of marked photophobia, irritation, watering, itching both eyes. Symptoms increased during warm weather conditions, sunlight, wind and dust. On examination, everting upper eyelids revealed giant papillae on the upper tarsal conjunctiva, cobblestone like swellings along with tenacious mucus discharge (Figure C and D), hence we made a diagnosis of vernal keratoconjunctivitis and started him on antihistaminic, topical and low potency topical steroids along with lubricants and he showed marked improvement with the treatment.

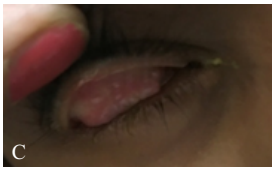
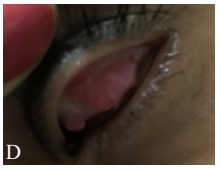
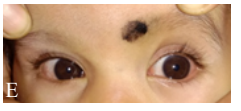



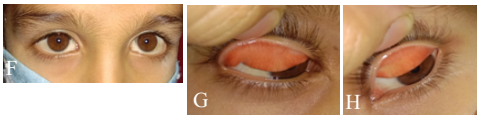
Figure C and D shows giant papillae on upper tarsal conjunctiva of both eyes on everting upper eyelids

Other Common Presentations Are:

there are varied clinical manifestations of ocular allergy in different patients in different age groups.



1 year old female child having itching in left eye, on examination she have **(Fig E)** conjunctival congestion and chemosis.



9 years old boy presents with complain of itching and discolouration of eyes as in **Figure F** and shows papillae on upper tarsal conjunctiva of both eyes with diffuse congestion of conjunctiva (**Figure G And H**) in chronic allergic conjunctivitis

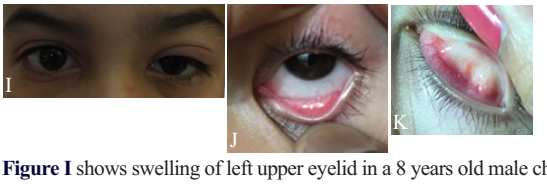


Figure I shows swelling of left upper eyelid in a 8 years old male child. **Figure J** shows presence of papillae on lower palpebral conjunctiva and plugged meibomian gland with its secretions on upper eyelid, **Figure K** shows presence of papillae, conjunctival chemosis and congestion of bulbar conjunctiva above upper tarsal conjunctiva on lid eversion.

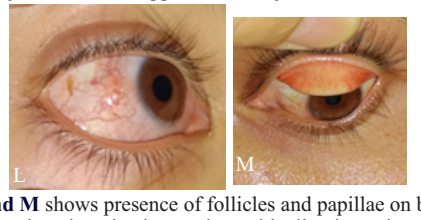


Figure L and M shows presence of follicles and papillae on bulbar and upper tarsal conjunctiva in a patient with allergic conjunctivitis.

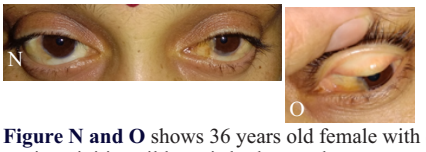
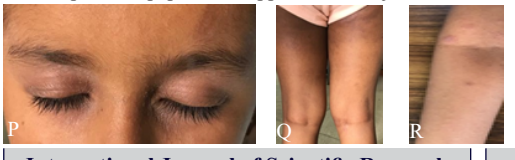


Figure N and O shows 36 years old female with chronic allergic conjunctivitis, mild ptosis both eyes due to excessive rubbing, yellowish discoloration of both eyes, pseudo pterygium formation, corneal pannus , papillae on upper tarsal conjunctiva on lid eversion.



6 years old male child with atopic conjunctivitis have dermatological manifestations of atopic dermatitis.
Figure P shows periorbital excoriation and pigmentation, **Figure Q** shows excoriation and pigmentation of skin of gluteal region and **Figure R** shows eczematous plaques on antecubital region.

There were many patients presented with similar complaints and treated with antihistaminic, tear substitutes and low potency steroids and showed marked improvement in their symptoms. Few patients also put on immunotherapy.

DISCUSSION:
Allergic conjunctivitis is caused by an allergen induced inflammatory response in which allergens interact with IgE bound to sensitized mast cells resulting in clinical ocular allergic expression. The pathogenesis of allergic conjunctivitis is predominantly an IgE - mediated hypersensitivity reaction. Activation of mast cells induces enhanced tear levels of histamine, tryptase, prostaglandins and leukotrienes. This is responsible for early or acute inflammatory response that lasts for 20-30 minutes. Mast cell degranulation also induces activation of vascular endothelial cells, which in turn expresses chemokines and adhesion molecules such as inter cellular adhesion molecule(ICAM), vascular cell adhesion molecule (VCAM), other secreted chemokine include regulated upon activation normal T cell expressed and secreted (RANTES) chemokines, monocyte chemo attractant protein (MCP), interleukin (IL)-8, eotaxin, macrophage inflammatory protein (MIP)-1 alpha. These factors initiate the recruitment phase of inflammatory cells in the conjunctival mucosa, which lead to ocular late phase reaction [3]. Seasonal allergic conjunctivitis and perennial allergic conjunctivitis are the most common forms of ocular allergies. These types of allergy usually affect 15-20% of the population. Signs and symptoms of SAC and PAC are the same. The difference is the specific allergens to which the patient is allergic. SAC is usually caused by airborne pollens. Signs and symptoms usually occur in the spring and summer, and generally abate during the winter months. PAC can occur throughout the year with exposure to perennial allergens. Diagnostic features of SAC and PAC consist of itching, redness, swelling of conjunctiva and periorbital swelling.

VKC is a disease of warm weather months and warm climates, more common in tropics. Young people are typically affected. In this form, a non specific hypersensitivity occurs that explains the ocular symptoms induced by non-specific stimuli- such as wind, dust, sunlight- which is not related to allergen levels in the environment. Skin tests and serum IgE antibody tests to common allergens are often negative [4]. VKC is a chronic allergic inflammation of the ocular surface mediated mainly by Th2-lymphocyte; in a complex pathogenesis have a role also the over expression of mast cells, eosinophils, neutrophils, Th2- derived cytokines, chemokines, adhesion molecules, growth factors, fibroblasts and lymphocytes. IL-4 and IL-13 are involved in the formation of giant papillae by inducing the production of extra cellular matrix and the proliferation of conjunctival fibroblasts [3]. VKC has 3 clinical forms: palpebral, limbal and mixed. It is common in males. Symptoms include ocular itching, redness, swelling and discharge. Patient has very severe photophobia. The most characteristic sign is giant papillae on upper tarsal conjunctiva. These 'cobblestone like 'swellings may be several millimeters in diameter, usually 10-20 are found on upper tarsal conjunctiva, can be seen easily by flipping the upper eyelid. The giant paillae are filled with inflammatory cells and edema. Neutrophils, plasma cells, mononuclear cells and eosinophils are found in abundance. There is mast cell activity within giant papillae and also foud in the conjunctival epithelium, where they are not seen normally. So, the tears of patient with VKC contains high amount of IgE and mast cell mediators. Histamine, leukotrienes, prostaglandins and kinase may be found in the tears of VKC patients. Punctate keratitis and formation of shield ulcer commonly seen in VKC, may interfere with vision. Tranta's dots consist of clumps of necrotic eosinophils, neutrophils and epithelial cells. The dots represent almost pure collections of eosinophils. These cells collect in crypts, which are formed by invaginations at the junction of cornea and conjunctiva. Tranta dots tend to appear when VKC is active and disappear when symptoms abate. Shield ulcers occur in superior part of cornea; these are noninfectious, oval shaped, circumscribed epithelial ulcer with underlying stromal opacification. After the ulcer heals, an anterior stromal opacity can persist. The massive eosinophil infiltration and activation in the conjunctiva is responsible for corneal complications [5].

Atopic keratoconjunctivitis (AKC) is a bilateral chronic inflammatory disease of the ocular surface and eyelid. Its pathomechanism involves both a chronic degranulation of the mast cell mediated by IgE and immune mechanisms mediated by Th1 and Th2- lymphocyte derived cytokines [3]. It is considered the ocular counterpart of atopic dermatitis or atopic eczema. They may have Dannie Morgan fold under the eyes, hyper linearity of palms. Eczematous lesions may be found on the eyelids, or any place on the body, are red and elevated. They often occur in the antecubital or popliteal regions. Eczematous lesions are itchy; eyelid skin may be chemotic with fine sandpaper like texture. Later, these lesions end up with excoriation and pigmentation of skin. There may be conjunctival congesion, chemosis followed by conjunctival scarring. AKC may also develop atopic cataracts, typically are anterior shield like cataract but nuclear, cortical and even sub capsular cataract may develop. Corticosteroid therapy in AKC may contribute to cataract formation. The appearance of AKC and VKC is similar; both may be associated with giant papillae and tranta spots. VKC usually resolves by age of 20 years, whereas AKC can persist throughout life [6].

Contact allergy or allergic contact dermatitis, is not an IgE- mediated allergy, it is a type IV delayed hypersensitivity response that lead to release of cytokines. It consists of two phases, sensitization phase and elicitation of inflammatory response. In sensitization phase, antigen presenting cells processed antigen- MHC class II complex interacts with T lymphocytes, resulting in the differentiation of CD4+ T-lymphocyte into memory T- lymphocyte. In the elicitation phase, the interaction between the antigen – MHC-II complex and memory T-cells stimulates the proliferation of T- cells. The memory T-lymphocytes during proliferation release cytokines [8]. Allergens are generally simple chemicals, low molecular weight substances that combine with skin protein to form complete allergens. Examples include poison ivy, poison oak, neomycin, nickel, latex, atropine and its derivatives. Contact allergy involves the ocular surface, eyelids and periocular skin. Although contact allergic reactions usually occur on the skin, including the skin of the eyelids, the conjunctiva may also support contact allergic reactions. Initial sensitization with a contact allergen may take several days. Upon re-exposure to the allergen, an indurated, erythematous reaction slowly develops. The reaction may peak 2-5 days after re-exposure. The delay in the development of the reaction is due to slow migration of lymphocytes to the antigen depot. The term 'delayed hypersensitivity' is sometimes given to these reactions. Contact allergic reactions are generally associated with itching. Treatment consists of withdrawing and avoiding contact with allergen [9]. Severe reactions can be treated with topical and systemic corticosteroids.

The diagnosis of ocular allergy is primarily clinical but allergists can perform skin testing for specific allergens by scratch test or intradermal injections of allergen. In vitro tests for IgE antibodies to specific allergens are widely used [9].

Treatment options include avoidance of offending antigen is primary behavioral modification for all types of allergic conjunctivitis. Artificial tear drops provide a barrier and help to improve the first line defense at the level of conjunctival mucosa; it also helps to dilute various allergens and inflammatory mediators that are present on ocular surface. In case of no relief with above measures, pharmacologic treatments may be applied locally or given systemically to diminish the allergic response. The mainstay of management of ocular allergy involves the use of antiallergic therapeutic agents such as antihistamine, multiple action anti-allergic agents and mast cell stabilizers [10]. In recent years, several multimodal anti-allergic agents have been introduced, such as olopatadine, ketotifen, azelastine, epinastine and bepotastine. They exert multiple pharmacological effects like histamine receptor antagonist action, stabilization of mast cell degranulation and suppression of activation and infilteration of eosinophils. These drugs are becoming the drug of choice for providing immediate symptomatic relief but when no adequate control with above drugs, anti inflammatory drugs are used [11,12]. Non-steroidal anti inflammatory drugs are used as additive drugs to reduce conjunctival hyperemia and pruritus [12]. Corticosteroids remain the most potent pharmacological agent used in more severe variants of allergic conjunctivitis, both acute and chronic forms. Flourometholone and loteprednol are the low potency steroids which are commonly used for some allergic conjunctivitis cases. They are given for short period of time i.e. for 2-3 weeks and show dramatic response in these patients [13]. Immunotherapy with cyclosporine and tacrolimus is also given to

those patients who require long term steroids for control of allergic conjunctivitis [14].

CONCLUSION:

We must be aware about the various presentations of allergic conjunctivitis, dermatological manifestations, their etiologies, control, prevention and treatment either short term or long term.

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