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Case report

Ocular surface disease: A case of vernal keratoconjunctivitis

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ABSTRACT

Vernal keratoconjunctivitis (VKC) is a recurrent, chronic allergic inflammation of the ocular surface associated with remodeling of the cornea and conjunctiva, with a potential to adversely affect the patient's vision and quality of life. It affects mainly children and young adults. The purpose of this interventional case report is to present a typical case of limbal VKC, which was unresponsive to antiallergic treatment alone.

A 5-year-old boy, who was previously unsuccessful with conventional anti-allergic therapy alone, presented with clinicopathological features pathognomonic of limbal VKC. The patient was successfully managed with a combination of palliative, anti-allergic and anti-inflammatory therapy. Prophylaxis with an anti-allergic pharmaceutical agent was initiated for long term use to prevent the release of allergic mediators.

This case highlights the importance of prescribing the most appropriate pharmaceutical medication for VKC since conjunctival fibroproliferative lesions are often resistant to anti-allergy therapy. Allergic inflammatory mediators are responsible for the fibroproliferative changes of the conjunctiva and corneal lesions in VKC. Thus, it is crucial to prescribe the most appropriate pharmaceutical medication that will prevent the release of allergic inflammatory mediators, control the allergic inflammatory cascade and prevent ocular surface damage. The fine points of this case are presented with a review of applicable literature.

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Vernal keratoconjunctivitis (VKC) is a recurrent, chronic allergic inflammatory disorder of the ocular surface [1,2]. It is a multifaceted ocular disorder that involves both the classic humoral allergic responses and the more complex cell-mediated immunologic processes. VKC is characterized by persistent conjunctival inflammation with an over-expression of mast cells, eosinophils, basophils, neutrophils, macrophages and lymphocytes [3,4].

VKC is bilateral in more than 96% of cases [5]. It generally affects pre-pubescent children and young adults from warm climates of the Mediterranean, Africa, Middle East, Asia, and Central and South America [2,6]. The male preponderance can be attributed to a possible role hormonal factors play in the development of VKC [3,7–9]. However, it usually resolves after puberty, about 4–10 years after onset, but progression towards atopic keratoconjunctivitis (AKC) can be observed at an adult age [2,8]. Although it reoccurs in spring and summer, Bonini et al. [3,5] reported that more than 60% of patients with VKC had recurrences in the winter. It has been reported that the longer patients suffer from VKC, there is a higher

probability of the seasonal presentation evolving into a perennial form of the disease [5]. Thus, depending on the climate and allergic disposition of the patient, it can be active throughout the year.

The classification of VKC is based on the main site of the papillary reaction: tarsal versus limbal [10]. The palpebral form is seen predominantly in Caucasians, whereas the limbal variety is the predominant form in non-Caucasians [11]. The tarsal form is characterized by irregular sized hypertrophic papillae that lead to cobblestone appearance on the upper tarsal plate [8], whereas the predominantly limbal (bulbar) form of VKC is characterized by gelatinous limbal infiltration [12–14]. Mixed VKC has both giant papillae on the upper tarsal conjunctiva and gelatinous infiltration of the limbus [15].

VKC can be characterized by two distinct groups (1) those patients that have a positive personal and family history of other allergic conditions and (2) those patients that have no personal or family history of other allergic disorders [8]. Approximately 50% of patients with VKC have a family history of allergic disorders such as asthma, rhinitis, eczema, and urticaria, and often are positive on standard allergen tests, such as prick or radioallergosorbent test (RAST) [3,5,8].

Diagnosis is usually based on typical history and clinical features of VKC. The symptoms of VKC include itching, burning, tearing, blepharospasm, foreign body sensation, photophobia, and mucus

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hyper-secretion resulting in mucoid discharge. The signs of VKC include pseudoptosis due to heavy giant papillae on the upper tarsal conjunctiva, filamentous mucus discharge, eyelid edema, limbal and conjunctival edema, perilimbal conjunctival pigmentation, cicatrizing conjunctivitis, the presence of aggregates of epithelial cells and eosinophils at the limbus (Horner-Trantas dots), pseudogerontoxon, upper limbal pannus, superficial punctate keratitis, neovascularization of the cornea, and corneal shield ulcers [6,9,16,17]. Giant papillary hypertrophy of the tarsal conjunctiva and gelatinous infiltrate at the limbus are hallmark features of VKC. The differential diagnoses of VKC include acute allergic conjunctivitis, giant papillary conjunctivitis (GPC) and atopic keratoconjunctivitis (AKC). VKC usually responds to a combination of non-pharmacologic therapy and pharmacologic therapy using anti-allergic and anti-inflammatory medications. Although VKC has a good prognosis; complications due to delayed and inadequate treatment may lead to fibrovascular reaction, tissue remodeling and permanent visual changes [5,8].

This paper presents a typical case of limbal VKC that was unresponsive to anti-allergic treatment alone. It will review the disease process of VKC and discuss the pharmaceutical agents that are most effective for treating this chronic, recurrent ocular surface disease. Since it is a relatively rare form of ocular allergy, understanding the disease process will aid clinicians in prompt diagnosis and effective management of VKC, with the primary objective of preventing the development of serious ocular sequelae. The clinicopathological features of vernal keratoconjunctivitis with emphasis on tissue remodeling as well as the principles of clinical management are presented.

1. Case summary

A 5 year-old boy presented to the eye clinic for the first time with a chief complaint of red and itchy eyes, of approximately three weeks duration. Additionally, the patient had watering eyes and stringy, white, thick discharge. The parent reported that the itching had been intermittent over the last 12 months. The mother reported the use of olopatadine 0.1% (Patanol, Alcon Laboratories, Inc., Fort Worth, Texas) ophthalmic solution and ketotifen fumarate 0.025% (Zaditor, Novartis Pharmaceuticals Corporation, East Hanover, New Jersey) ophthalmic solution twice a day in both eyes (OU), as prescribed by another physician. Neither medication seemed to achieve therapeutic relief. His ocular history was otherwise unremarkable. Present medical history was remarkable for both asthma and severe seasonal allergies. His entrance visual acuities at distance were 20/30 OD (right eye) and 20/40 OS (left eye). Color vision, pupils, confrontation fields, tonometry and ocular motility were unremarkable. Biomicroscopic examination showed grade II (CCLRU (Cornea and Contact Lens Research Unit) Grade Type 2) papillary hypertrophy of the upper tarsal conjunctiva, diffuse grade III (CCLRU Grade Type 3) conjunctival injection, perilimbal conjunctival hyperpigmentation, and large limbal papillae and Horner-Trantas dots OU (Fig. 1a and b). Corneal evaluation revealed punctate epithelial erosions adjacent to the limbal infiltrates OU. The diagnosis of limbal form of VKC was based on the pathognomonic presence of gelatinous limbal infiltrates and Horner-Trantas dots in the absence of characteristic giant papillae on the palpebral conjunctiva. He was prescribed prednisolone acetate ophthalmic suspension 1% (Pred Forte, Allergan, Inc., Irvine, CA), dosed every 3 h OU, and nedocromil sodium ophthalmic solution 2% (Alocril, Allergan, Inc., Irvine, CA) dosed twice a day OU. In addition to the pharmacologic therapy, the mother was advised to apply cold compresses at least twice daily and carboxymethylcellulose sodium ophthalmic solution 0.5% (Refresh tears, Allergan, Inc., Irvine, CA) four times daily as palliative therapy. The patient

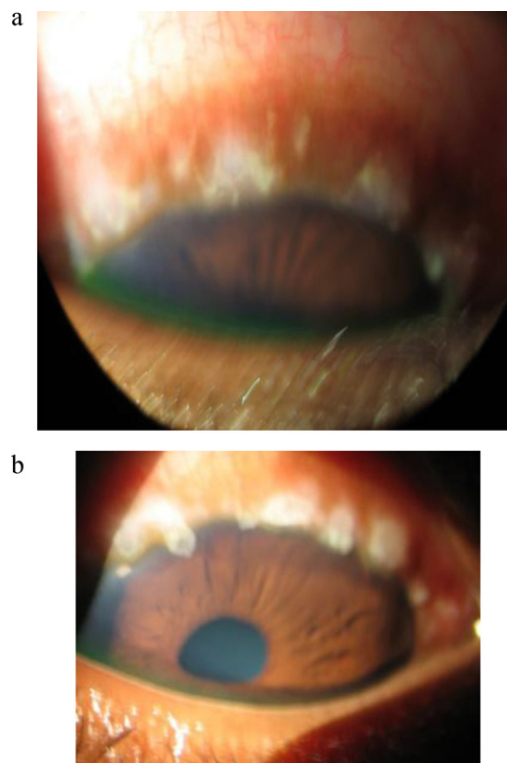


Fig. 1. (a) Gelatinous limbal infiltration with Horner-Trantas dots in Limbal VKC OD. (b) Gelatinous limbal infiltration with Horner-Trantas dots in Limbal VKC OS.

was advised to avoid rubbing his eyes since it will make the condition worse and/or delay the response to therapeutic management. The importance of adhering to the recommended treatment protocol and the complications of undertreated VKC was reviewed with the parent. The patient was scheduled to return in five days for re-evaluation.

The patient returned on the 6th day for his first follow-up visit. Although his symptoms were slightly improved, he still had persistent itch and redness of both eyes. The parent reported compliance with the recommended therapeutic protocol. The Alocril 2% ophthalmic solution was switched to cromolyn sodium 4% ophthalmic solution dosed four times daily OU, because his medical insurance plan did not authorize Alocril 2% ophthalmic solution. Visual acuities, pupils, ocular motility, tonometry, confrontation fields remained stable and unremarkable. Biomicroscopic examination showed grade II (CCLRU Grade Type 2) papillary hypertrophy of the upper tarsal conjunctivae, perilimbal conjunctival hyperpigmentation, and small limbal papillae OU (Fig. 2a and b). The conjunctival injection was now mainly concentrated in the superior limbal area OU. The assessment of the condition at this visit was that the limbal VKC was responding to the prescribed palliative and pharmacological therapeutic modality. In light of the complaints of ocular pruritus on the follow-up visit, they were advised to resume using olopatadine 0.1% ophthalmic solution dosed twice-daily OU to provide antihistaminic coverage. They were advised to continue with the current therapeutic regimen and return to the clinic in 2 weeks for a follow up assessment.

He returned as scheduled, with marked improvement of both signs and symptoms. His entrance visual acuities at distance were 20/30 OD and 20/20 OS. Pupils, ocular motility, tonometry, confrontation fields remained stable and unremarkable. Biomicroscopic examination showed bilateral grade I (CCLRU Grade Type 1) papillary hypertrophy of the upper tarsal conjunctiva and perilimbal conjunctival hyperpigmentation. There was no bulbar

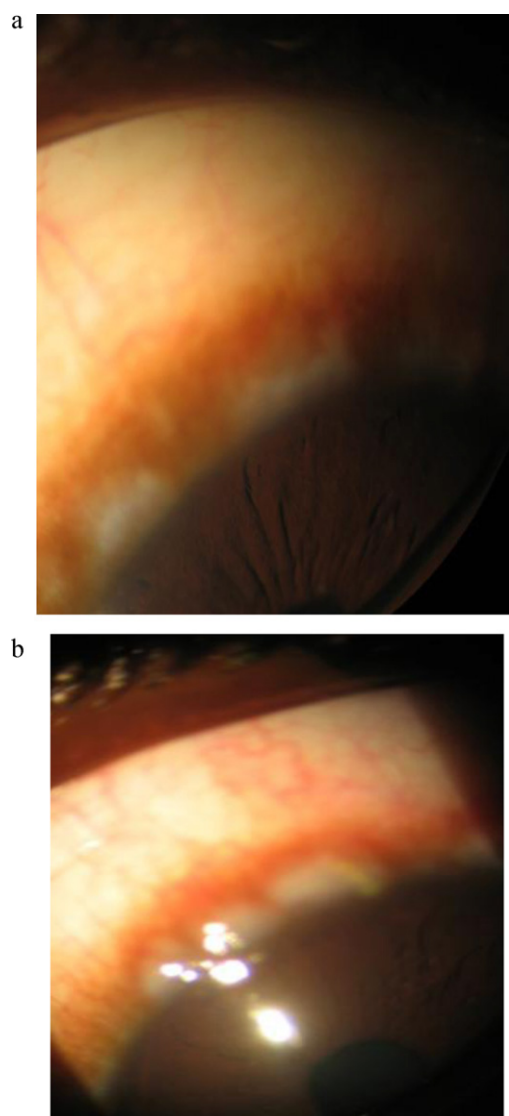


Fig. 2. (a) Limbal VKC resolving OD. (b) Limbal VKC resolving OS.

conjunctival injection and no evidence of gelatinous limbal infiltrates OU (Fig. 3a and b). The limbal VKC was judged to be resolved, and a taper schedule was started for the topical corticosteroid (Pred Forte 1% ophthalmic suspension) on a 4-3-2-1 schedule over one month. They were advised to return to the clinic in one month for a follow up assessment.

On his follow-up visit a month later (third follow up visit), the patient was asymptomatic. His entrance visual acuities at distance were 20/25 OD and 20/25 OS. Pupils, ocular motility, tonometry, fundus appearance, confrontational fields remained stable and unremarkable. Biomicroscopic examination was unremarkable OU. The limbal VKC was assessed as completely resolved, and as such, they were advised to discontinue the Pred Forte 1% and cromolyn sodium 4%. The disease process and the chronic, recurrent nature of VKC was reviewed with them and for this reason, they were instructed to continue the long-term use of olopatadine 0.1% dosed twice-daily OU and carboxymethylcellulose sodium ophthalmic solution 0.5% PRN OU for maintenance therapy. They were advised to return to the clinic in two months for a follow up assessment.

2. Discussion

When a patient presents with complaints of red, itchy eyes, it is crucial to obtain a detailed history in addition to a meticulous biomicroscopic and external eye examination. A complaint of recurrent or persistent itching should alert the clinician that an allergic disorder of the ocular surface is the likely underlying etiology. The patient's complaint of red, itchy eyes in conjunction with the presence of papillary hypertrophy of the tarsal conjunctiva, Horner-Trantas dots and gelatinous limbal infiltration were features pathognomonic of limbal VKC (Fig. 1a and b). Corneal involvement in this patient was limited to mild punctate epithelial keratitis. This finding is considered a sign of corneal involvement in patients with limbal VKC [8,18]. Epithelial macro-erosions, shield ulcer formation, plaque formation, corneal vascularization, secondary microbial keratitis, keratoconus, pellucid marginal degeneration, keratoglobus, superior corneal thinning, hydrops, pseudogerontoxon and corneal opacification are other signs of corneal involvement in patients with VKC [9,15,19,20]. It was of note that biomicroscopic examination revealed the presence of perilimbal conjunctival hyperpigmentation in this patient. There is an abundance of melanocytes and mast cells in the limbus. Perilimbal conjunctival hyperpigmentation (Fig. 4), a golden brown pigmented limbal thickening, is seen in most non-Caucasian patients with VKC [14,21]. Luk et al. [21] suggested that it may be a by-product of complex immune interactions and pathways involving growth factors or interleukins that stimulates the melanocytes to produce the perilimbal pigment.

The immunopathogenesis of VKC is multifactorial [7]. VKC could be an IgE-mast cell/Th2-eosinophil or non-IgE-mast cell/Th2-eosinophil mediated hypersensitivity reaction with additional nonspecific hypersensitivity responses [8,13]. In addition, climatic, environmental, hormonal, genetic and neural factors may influence the pathogenesis of VKC [10,22,23]. Cytological and immunohistochemical studies indicate that VKC is a Th2-lymphocyte-mediated allergic inflammatory disease with an over expression of Th2-lymphocyte-derived cytokines, chemokines, adhesion molecules, eosinophils, growth factors, fibroblast, enzymes and other cells and mediators within the tear film and ocular surface [8,9]. The Th2-lymphocyte-derived cytokines favor a local hyperproduction of IgE (interleukin (IL)-4, IL-9, IL-13), recruitment and activation of eosinophils (IL-5, GM-CSF) and differentiation and activation of mast cells (IL-3) [3,7,8,23]. The histopathological and clinical presentation of VKC is influenced by the inflammatory cells and mediators involved in the disease process [24]. Histopathological studies of conjunctival tissue from VKC patients show infiltration of the conjunctiva by inflammatory cells (eosinophils, mast cells, CD4⁺T-lymphocytes, dendritic cells, macrophages, epithelial cells, fibroblasts, dendritic cells bearing IgE) and mediators (cytokines, chemokines, histamine, growth factors and matrix metalloproteinases) as well as post-inflammatory tissue remodeling [1,8].

The clinicopathological features of limbal VKC are characterized by the presence of gelatinous limbal infiltrates, papillary hypertrophy of the tarsal conjunctiva, corneal lesions and Horner-Trantas dots. Gelatinous limbal infiltrates and papillae on the tarsal conjunctiva are fibroproliferative lesions of the conjunctiva, and are pathognomonic features of tissue remodeling in VKC. Histopathological studies of giant papillae revealed the presence of eosinophils, mast cells, conjunctival fibroblast, deposited extracellular matrix (ECM) molecules, growth factors, and Th2-lymphocytes [25]. Th2-lymphocyte-derived cytokines (IL-4 and IL-13), pro-inflammatory cytokines (TNF- α , IL-1 α and IL-1 β), eosinophils, macrophages, matrix metalloproteinase, and growth factors (vascular endothelial growth factor (VEGF), transforming growth factor (TGF)- β , basic fibroblast growth factor (bFGF), and platelet-derived growth factor (PDGF)) play an important role in the

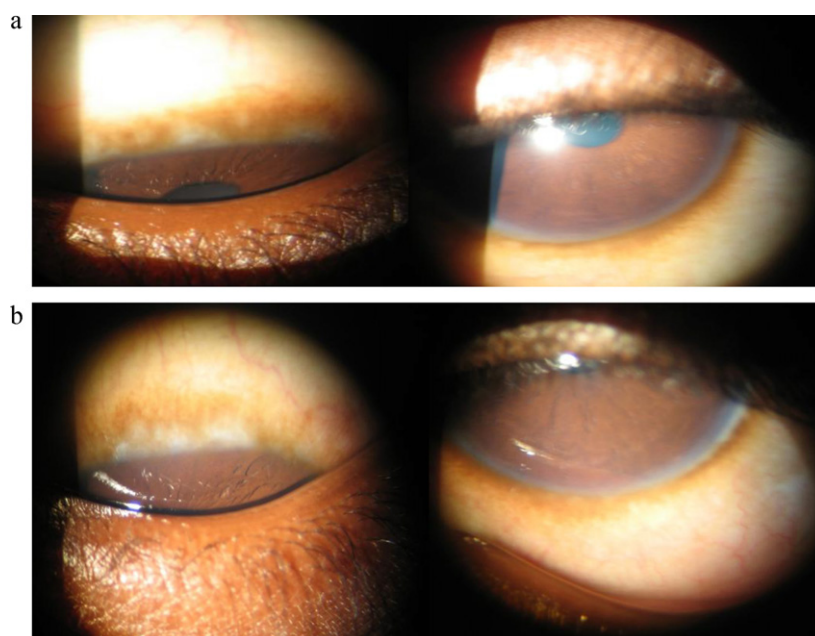


Fig. 3. (a) Limbal VKC resolved OD. (b) Limbal VKC resolved OS.

pathogenesis of tissue remodeling of the ocular surface of patients with VKC. The growth factors are expressed by conjunctival resident cells (epithelial cells, fibroblasts, mast cells) and inflammatory cells (eosinophils and macrophages) [26–28]. The matrix metalloproteinase (MMP-2 and MMP-9) are expressed by eosinophils, macrophages, conjunctival resident cells, corneal epithelial cells and corneal fibroblasts. Th2-lymphocyte-derived cytokines, pro-inflammatory cytokines and growth factors are responsible for the development of fibroproliferative lesions of the conjunctiva in VKC. These lesions occur as a result of hyperplasia of the conjunctival fibroblast, increased deposition of ECM molecules, and infiltration of inflammatory cells [27–29].

The cornea lack immune cells (with the exception of Langerhans' cells at the periphery of the corneal epithelium) and is composed of three structural cells (epithelial cells, fibroblasts, and endothelial cells) that may function as modulators of the allergic inflammation in VKC. Although the cornea is not the primary site of allergic inflammation in VKC, it has been reported that corneal involvement occurs in more than 50% of patients with VKC [27]. Th2-lymphocyte-derived cytokines and pro-inflammatory cytokines in the tear film stimulate the proliferation and migration of conjunctival and corneal fibroblasts. The activated corneal fibroblasts express eotaxin and vascular cell adhesion molecule (VCAM)-1 that mediate the infiltration and activation of eosinophil. The activated eosinophil releases MMP-2, MMP-9 and eosinophil-derived cytotoxic mediators. The active forms of MMP-2 and MMP-9 in tear film of VKC patients may play an important role

in the pathogenesis of corneal lesions by mediating the degradation of collagen type IV and laminin of the corneal epithelial basement membrane. The exposure of the corneal epithelium to eosinophil-derived cytotoxic mediators and MMPs in the tear film can compromise the barrier function of the corneal epithelium. The breakdown of the barrier function of corneal epithelium could lead to the development of persistent corneal epithelial defects in patients with limbal VKC [27,28,30].

The differential diagnoses of VKC should include acute allergic conjunctivitis, giant papillary conjunctivitis (GPC) and atopic keratoconjunctivitis (AKC). Unlike VKC, the pathogenesis of allergic conjunctivitis is predominantly an IgE-mediated hypersensitivity reaction; moreover, eosinophil and Th2-lymphocytes do not play a major role in the pathogenesis of allergic conjunctivitis, as noted by the absence of corneal damage in seasonal allergic conjunctivitis (SAC) and perennial allergic conjunctivitis (PAC) [4,31]. Although the histologic appearance of the conjunctiva in VKC and GPC is almost identical, the main difference is that the number of eosinophils and basophils is greater in VKC than in GPC [32,33]. The level of eosinophilic major basic protein (EMBP), which may be responsible for injury to the corneal epithelium, is increased in patients with VKC but not in those with GPC [32]. The perennial nature of the atopic keratoconjunctivitis distinguishes it from vernal keratoconjunctivitis, which typically has a seasonal pattern. Furthermore, AKC tends to have lower eyelid and inferior palpebral conjunctival involvement and begin in the late teens, whereas VKC is characterized by superior tarsal conjunctival papillary hypertro-

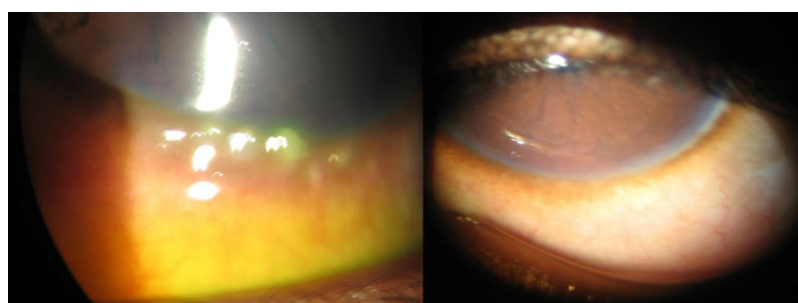


Fig. 4. Perilimbal conjunctival hyperpigmentation.

phy and tends to affect mostly children and young adult males [34].

The presence of limbal papillae and Horner-Trantas dots in the absence of characteristic giant papillae on the tarsal conjunctiva is distinctive of VKC. The diagnosis of VKC is usually based on the collective consideration of all clinical signs and symptoms. Although diagnosis can be made based on typical clinical signs and symptoms of the disease, tear and conjunctival cytology can be a quick and noninvasive diagnostic tool used to help in difficult or subtle cases [7,8]. Conversely, skin prick test or RAST are not considered useful in diagnosing VKC since it has been reported that more than 50% of patients with VKC have negative results on these tests [3,7].

VKC is a chronic allergic inflammation of the ocular surface associated with remodeling of the cornea and conjunctiva, and as such, a topical corticosteroid was utilized to bring the Th2-lymphocyte-mediated immune hypersensitivity reaction under control. To achieve this, the patient was prescribed Pred Forte 1% ophthalmic suspension and Alocril 2% ophthalmic solution. In addition to the pharmacologic therapy, the mother was advised to apply cold compresses at least twice a day and an ocular lubricant (Refresh tears) four times a day as palliative therapy. The patient was also advised to avoid rubbing his eyes, to prevent delay of therapeutic response. Unfortunately, because his medical insurance did not permit Alocril 2% ophthalmic solution, it was switched to cromolyn sodium 4% ophthalmic solution dosed four times a day in both eyes. The ultimate goal of the therapeutic strategy employed in this case, was to suppress and/or control the allergic inflammation.

The supportive therapy of this limbal form of VKC included avoiding the trigger factors, avoiding eye rubbing, applying cool compress, and administering ocular lubricants. The ocular lubricants were given to aid in stabilizing the tear film, lubricating the ocular surface, and diluting and flushing away allergens and allergic mediators [8]. Avoidance of eye rubbing was advised, since the mechanical degranulation of mast cells may exacerbate the intensity of the itching [34]. In addition, the mechanical microtrauma from chronic eye rubbing can induce the release of neutrophil chemotactic factors (NCF) from the injured conjunctival cells, as well as trigger mast cell degranulation. The released NCF attracts inflammatory cells to the site of conjunctival trauma or irritation. The interaction of these recruited inflammatory cells with the mediators released following mechanically-induced mast cell degranulation can exacerbate the VKC [8,31,33,35].

The standard pharmacologic treatment of VKC consists of topical mast cell stabilizer, anti-histamine, multimodal anti-allergic agents with mast cell stabilizer and antihistamine effects, corticosteroid and immunosuppressive preparations [10]. The management strategy adopted in this case was influenced primarily by the severity of the clinical presentation. The patient had moderate-to-severe limbal VKC, and as such, he was treated concurrently with topical steroid, mast cell stabilizer and multiple action anti-allergic pharmaceutical agents to achieve adequate therapeutic relief. Topical corticosteroid was considered the therapeutic agent of choice for treating this case because they down-regulate the allergic inflammation by blocking phospholipase A₂; inhibits histamine synthesis in mast cells by inhibiting histidine decarboxylase; reduces the level of unbound histamine on the ocular surface by increasing histaminases enzyme; and reduces production of late-phase allergic mediators [36]. In light of the complaints of ocular pruritus on the first follow-up visit, olopatadine 0.1% was added to the treatment regimen to provide antihistaminic coverage due to its proven efficacy in providing immediate relief from ocular pruritus. Cromolyn sodium, a mast cell stabilizer, requires a loading period of two weeks to achieve maximum relief from ocular pruritus [36]. Nedocromil sodium was the preferred choice of mast cell stabilizer because it is now considered to be a multiple action anti-allergic

agent due to its H1 receptor antagonist properties and inhibitory effects on allergic inflammatory cells, mast cells and eosinophils [36,37]. The patient's previous insufficient response to multimodal anti-allergic therapeutic agents is likely due to the presence of fibroproliferative lesions on the conjunctiva. It has been reported that corneal lesions and conjunctival proliferative changes in VKC are often resistant to anti-allergic therapy but are responsive to topical steroidal therapy or immunomodulatory agents [27,38]. Fluorometholone 0.1% ophthalmic suspension was not used in this case because of the moderate-to-severe presentation. Although clinical studies have shown that fluorometholone-based corticosteroids reduces the clinical expression of VKC, including papillary hypertrophy and Tranta's dots [39,40], they are usually indicated for mild to moderate ocular surface inflammatory conditions [41]. Although a site-specific topical corticosteroid with a proven safety profile such as loteprednol etabonate 0.5% would have been preferable over prednisolone acetate 1.0% for long term use, issues with medical insurance coverage precluded its use. Although its utilization in controlling the chronic allergic inflammatory process in VKC [8,42] maybe warranted, maximum strength topical corticosteroids, such as prednisolone acetate 1.0%, are usually considered when site-specific topical corticosteroids are not effective in controlling the allergic inflammation [8]. Topical cyclosporine has been proven to be effective in the long-term treatment of both limbal and tarsal VKC. It is prescribed, when steroids are contraindicated or deemed unsafe, for treating the severe form or steroid resistant cases of VKC. Although cyclosporine ophthalmic emulsion may be considered a suitable alternative to corticosteroids, in the presence of corneal complications caused by epithelial toxic allergic mediators, such as eosinophil major basic protein (EMBP), topical steroidal therapy is the preferred choice [3,7,8,43,44], since the therapeutic effect of dampening the inflammatory process is achieved more rapidly with corticosteroid. In this case, appropriate therapeutic measures were taken to avoid the longstanding permanent inflammatory sequelae in VKC that may lead to tissue remodeling and permanent visual damage [2]. Furthermore, topical steroidal therapy was essential for treating VKC-induced keratopathy since if left untreated, the punctate epithelial keratitis could evolve into corneal macroerosions and perhaps into a shield ulcer. Notably, multimodal anti-allergic therapeutic agents are less likely to have any effect on the Th2-lymphocyte-mediated clinicopathological features of VKC since the corneal and conjunctival fibroproliferative lesions are often resistant to anti-allergy therapy [27].

Patients may develop a visual impairment secondary to corneal damage, corticosteroid-induced cataract and glaucoma, microbial keratitis or limbal tissue hyperplasia [2,5]. Limbal infiltrates may cause transitory modifications of corneal astigmatism whereas keratoconus is the most frequent corneal ectasia associated with VKC. Chronic eye rubbing and activation of metalloproteinase may play a role in the development of keratoconus in VKC [8]. Therefore, it is important to counsel young patients about avoiding eye rubbing. Totan et al. [45] reported that approximately 27% of patients with VKC in their study had keratoconus, whereas Khan and colleagues [46] reported an incidence of 7%. The higher incidence reported by Totan et al. could be due to their use of computer-assisted videokeratographic maps that aided in the detection of subclinical keratoconus in patients with VKC, which could be overlooked using traditional methods such as biomicroscopy and keratoscope [45]. Therefore, evaluation of patients with VKC for subtle signs of keratoconus could be considered an important aspect of the management plan.

Although the long-term prognosis of patients with VKC is generally good, it has been reported that 6% of patients develop visual complications from corneal damage [3]. It has been reported that the bulbar (limbal) form of VKC may have a worse long-term prognosis than the tarsal form [23]. However, the majority of patients

with VKC have spontaneous resolution of the disease after puberty without any further symptoms or visual complication [3].

3. Conclusion

In this interventional case report the clinicopathological features and management of VKC with an emphasis on selecting the most appropriate therapeutic intervention has been discussed. The management of VKC presents a particular challenge to the clinician. It calls for formulation of appropriate ocular allergy differentials and adequate knowledge of their clinicopathological features. An adequate knowledge of the treatment mechanisms and clinical pharmacology of pharmaceutical agents used in the management of VKC is necessary; as such knowledge will enable the clinician to prescribe the most appropriate pharmaceutical medication that will best address the allergic reaction.

VKC is a chronic form of ocular allergy with increased potential for developing ocular complications that can adversely affect visual prognosis. As such, the therapeutic strategy in managing this case involved preventing the release of allergic mediators, controlling the allergic inflammatory cascade and preventing ocular surface damage. This case has demonstrated the importance of prescribing topical steroidal therapy for VKC since the corneal lesions and conjunctival fibroproliferative lesions do not often respond to anti-allergy therapy. The long-term maintenance therapy for this young patient called for the use of a pure mast cell stabilizer or multimodal anti-allergic agent to be used preferably prior to the allergy season or all year round depending on the allergic exposure and duration of the allergic expression. The correct management of VKC is imperative to avoid any adverse effects on the patient's quality of life, as well as, the ocular surface damage that might occur if this condition is under-treated or left untreated.

Statement on proprietary interest

The authors have no proprietary interest in any of the therapeutic agents mentioned in this paper. The authors report no conflict of interest.

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