International Journal of Scientific Reports
Minhas S et al. Int J Sci Rep. 2019 Sep;5(9):254-256
http://www.sci-rep.com
pISSN 2454-2156 | eISSN 2454-2164
DOI: http://dx.doi.org/10.18203/issn.2454-2156.IntJSciRep20193764

Case Report

Clinical presentation of vernal keratoconjunctivitis in an immunocompromised patient: a case report

Shailender Minhas[1]*, Rajeev Tuli[2], Gaurav Sharma[2]

[1]Eye Mobile Unit, Zonal Hospital, Dharamshala, Himachal Pradesh, India
[2]Department of Ophthalmology, Dr. Rajendra Prasad Government Medical College, Kangra, Himachal Pradesh, India

Received: 01 August 2019
Accepted: 17 August 2019

***Correspondence:**
Dr. Shailender Minhas,
E-mail: shailendraminhas@gmail.com

ABSTRACT

Vernal keratoconjunctivitis (VKC) is a chronic bilateral inflammation of the conjunctiva, commonly associated with a personal or family history of atopy. It is characterized by severe itching, foreign body sensation, thick ropy discharge, photophobia and conjunctival injection. VKC has palpebral, limbal and mixed forms. The classical conjunctival sign in palpebral VKC is the presence of giant papillae, which are predominantly seen on the superior tarsal conjunctiva. The limbal form occurs in dark skinned individuals and the papillae tend to occur at the limbus and have a thick gelatinous appearance. Clinical findings and laboratory investigations support the presence of IgE mediated type 1 hypersensitivity reaction. Involvement of CD4 T helper (Th2) driven type IV hypersensitivity has also been confirmed. There has been an increase in the prevalence of allergic disorders in recent years and exaggerated manifestations of these diseases have been recognized in patients living with Human immunodeficiency virus.

Keywords: VKC, Immune-compromised state, Shield ulcer, CD4 counts

INTRODUCTION

Vernal keratoconjunctivitis (VKC) is a chronic, bilateral, recurrent, conjunctival inflammatory condition found in young individuals predisposed by their atopic background.[1] The disease shows marked seasonal (spring/summer) influence, probably secondary to vernal allergens, but perennial forms also exist. The onset of disease is generally before the age of 10 years. The mean age group of the disease is 4-20 years and resolves during late puberty. In the younger age group males predominate but in older age group male to female ratio is nearly equal.[2,3] VKC is both type I (IgE dependent) hypersensitivity reaction to exogenous antigens and Type IV (Th2 lymphocyte mediated) hypersensitivity reaction. B lymphocytes from the lymphoid follicles of the conjunctiva locally produce IgE. Chronic allergic inflammation of the ocular surface is mainly mediated by Th2 derived cytokines, IL4, IL5 and IL13 along with over expression of mast cells, eosinophils, neutrophils, chemokines, adhesion molecules, growth factors and fibroblasts. Once activated, Th2 lymphocytes play role in recruitment and activation of mast cells and eosinophils. Degranulation of mast cells leads to histamine release, causing classical allergic reactions of vasodilatation, edema, hyperaemia and recruitment of inflammatory cells.[8,10]

Clinical features

The main symptoms of VKC are itching, photophobia, foreign body sensation, redness, mucous production and burning sensation. Blepharospasm may also occur in severe cases. Conjunctiva develops papillae over upper tarsus and limbus shows gelatinous opacifications. The Giant papillae may increase the mass of upper lid markedly and hence pseudoptosis may be typical. Horner-Trantas dots are another important feature of

VKC which are collections of epithelial cells and eosinophils seen in the conjunctiva at the corneoscleral limbus. A ropy, lardaceous thread can be found in the inferior fornix.[1,2,4,5] Shield ulcer usually has its lower border in the upper half of the visual axis. There is an association of keratoconus in VKC patients.[4,6]

CASE REPORT

This 18-year-old male presented with itching, ropy discharge, redness, photophobia and foreign body sensation in both eyes for 2 months. Complaints of diminution of vision both eyes for 2 years and recurrences of symptoms since 10 years of age. His symptoms aggravated during summers and after exposure to dust and smoke.

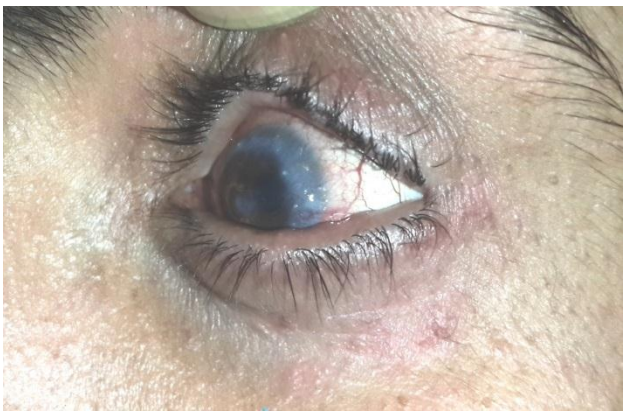

Figure 1: Shield ulcer in the pupillary region, conjunctivilization of the cornea, conjunctival congestion, neovascularization of cornea and Horner Taranta's spots.

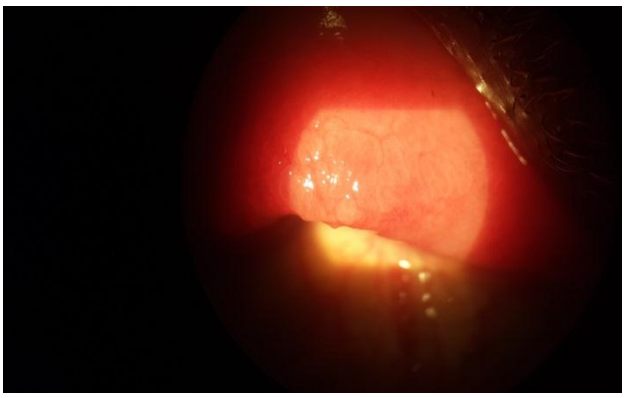

Figure 2: Giant papillary hypertrophy.

Eye examination

Visual acuity, unaided at the time of examination was 6/36 in R/E, 6/36 in L/E and the BCVA was 6/18 and 6/24 in R/E and L/E respectively. Lid margins showed anterior blepharitis along with meibomian gland disease Gd IV in both eyes. Thickened lid margins were noted in both eyes, conjunctivalization of cornea was seen in both the eyes. Corneal neovascularization (Figure 1) and giant papillary hypertrophy (Figure 2) was present bilaterally. Central nebular opacity in the right eye and shield ulcer (Figure 1) in the left eye were also noted. Horner-Trantas dots were also present in the left eye.

Schirmer's test-1 was 20 mm in right eye and 18 mm in the left eye. TBUT was 6 sec in right eye and 8sec in the left eye. Keratometry readings were K1=41.75R/E, K2=50.25R/E and K1=52.75L/E, K2=60 L/E.

General examination

His ESR was 55 mm in 1sthr, WBC count 7600/mm^3, neutrophils 47%, lymphocytes 39%, E=10%, Monocytes 4% and Basophils 0%. His Mantoux was negative, HIV status was positive and CD4 count was 204 cells.

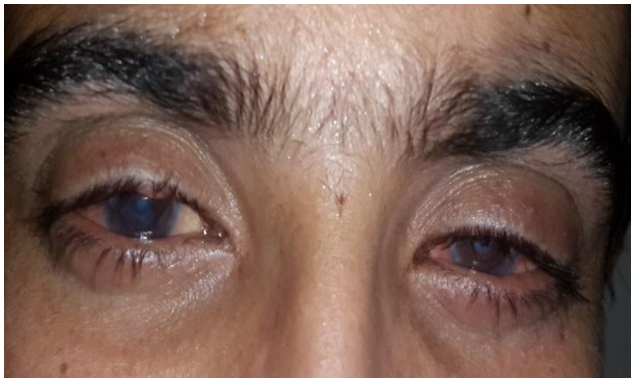

Figure 3: CD 4 COUNT 204, at the time of presentation.

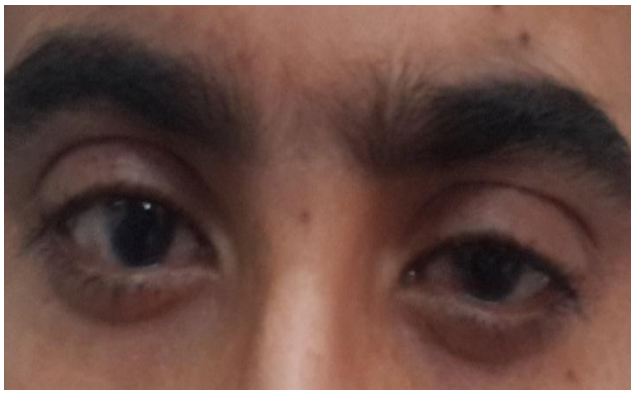

Figure 4: CD 4 COUNT 538, 3 months after initiating therapy.

DISCUSSION

Very few case reports exist in the literature related to clinical profile of VKC in HIV positive patients. VKC is a Th2 subset of T lymphocyte driven type IV hypersensitivity disease and considered to be uncommon in HIV positive patients. Diseases like allergic rhinitis, asthma and cutaneous rashes may be more severe in HIV positive patients, reasons for which may be an increase in

serum IgE levels in these individuals contrary to the fact that inflammatory and allergic disease manifestations will be less profound in those with suppressed immunity.

It was further found that in HIV, not only the quantity of CD4 T helper cells was less but they also had abnormal function and this may have led to increased manifestations of VKC in our patient.[7-9]

Though the exact mechanism of how the Th2 Lymphocytes function is not still clear, it is postulated that cytokines such as IL-4 undergo changes in HIV infection, in which increased concentration of IL-4 may be observed favoring IgE synthesis.

Th1 Lymphocytes produce Interferon-γ which acts as suppressor of IgE, but in HIV positive patients Th1 Lymphocytes are depleted, as a result more IgE is formed.

There is HIV related polyclonal hyperactivity of B cell, which further increases the production of IgE.[9]

Another theory postulates class switching of T lymphocytes to B lymphocytes leading to production of IgE, this may explain why the manifestations of VKC were more in our patient.

Few studies are found that specifically address VKC with HIV in patients of younger age group (18 years or less). Considering the diversity of information that can be explored in this population and characteristics that various studies show, further research is needed on the relevant issue.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Bonini S, Lambiase A, Marchi S, Pasqualetti P, Zeccaro O, Magrini L, et al. Vernal keratoconjunctivitis revisited: A case series of 195 patients with long term follow up. Ophthalmology. 2000;107:1157–63.
2. De Smedt S, Wildner G, Kestelyn P. VKC: an update. Br J Ophthalmol. 2013;97:9–14.
3. Kosrirukvongs P, Vichyanand P, Wongsawad W. Vernal Keratoconjunctivitis in Thailand. Asia Pac J Allergy Immunol. 2003;21:25–30.
4. Neumann E, Gutmann MJ, Blumen Kranz N, Michaelson IC. A review of four hundred cases of vernal conjunctivitis. Am J Ophthalmol. 1959;47:166-72.
5. Saleh. A. Al-Akily, Mahfouth. A. Bamashmus. Ocular complications of severe VKC in Yemen. Saudi. J Ophthalmol. 2011;25(3):291-4.
6. Barreto J Jr, Netto MV, Santo RM, Jose NK, Bechara SJ. Slit-Scanning topography in VKC. Am J Ophthalmol. 2007;143(2):250–4.
7. Wright DN. (HIV) infection. J Allergy Clin Immunol. 1990;85:445-52.
8. Abu El-Asrar AM, Al-Mansouri S, Tabbara KF, Missotten L, Geboes K. Immunopathogenesis of conjunctival remodeling in vernal keratoconjunctivitis. Eye. 2006;20:71.
9. Linhar et al. Allergy, Asthma Clin Immunol. 2014;10:35
10. Mossman TR, Coffman R. Two types of mouse helper T cells clone: Implications from immune regulation. Immunol Today. 1987;8:233.

Cite this article as: Minhas S, Tuli R, Sharma G. Clinical presentation of vernal keratoconjunctivitis in an immunocompromised patient: a case report. Int J Sci Rep 2019;5(9):254-6.