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Case Report

A Case of Shield Ulcer with Corneal Scarring in a Child of Vernal -Keratoconjunctivitis

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Abstract

Vernal keratoconjunctivitis (VKC) is a chronic severe allergic inflammatory disease of the ocular surface in children and young adults. The disease is characterized by constant ocular irritation, frequent eye rubbing, photophobia, tearing, mucous discharge, and droopy lids .A 13 year old male child presented to us with severe itching, ropy discharge and redness since 6 years of age with excerbation from last 1 month.Examination shows giant papillae giving cobblestone appearance, punctate epithelial keratopathy with a macular corneal opacity in right pupillary area.Child was treated with topical olopatadine and ketorolac combination eye drops , carboxymethylcellulose eye drops, homatropine eye drops, ointment chloramphenicol and polymyxin B sulphate, tablet vitamin C and oral antihistaminics. Child improved in a span of 2 weeks.

Keywords: *Vernal Keratoconjunctivitis (VKC),Giant Papillae*

Introduction

Vernal keratoconjunctivitis (VKC) is a chronic severe allergic inflammatory disease of the ocular surface in children and young adults [1]. The disease is prevalent in dry hot climates, specifically at the Mediterranean basin, the Middle East, Central and West Africa, India, and South America. It is more prevalent in boys, starts during the middle of the first decade of life, and resolves after puberty [2]. The disease is characterized by constant ocular irritation, frequent eye rubbing, photophobia, tearing, mucous discharge, and droopy lids. The signs of the disease are mostly confined to the conjunctiva and include giant papillary reaction of the upper tarsal conjunctiva, and limbal hypertrophy or limbal infiltrates and nodules.

The cornea is affected as a result of the frequent mechanical injury caused by the rough surface of the giant tarsal papillae of the upper tarsal conjunctiva, which may disrupt the corneal epithelium. In addition, the inflammatory mediators that are secreted by the activated eosinophils and mast cells may further cause damage to the corneal epithelium. This constant damage to the cornea may cause severe complications, including shield ulcers and vernal plaques, keratoconus, corneal scarring, microbial keratitis, and limbal epithelial stem cell deficiency.

Case Report

A 13 year old male child from a nearby village presented to us with a history of severe itching, ropy discharge and redness of both eyes since 6 years of age with present excerbation from last 1 month in the month of May. On examination, visual acuity was 6/12 improving to 6/9 with pin hole OD and 6/9 OS, normal pupillary reactions and full ocular movements. On torch light examination, severe bulbar and palpebral conjunctival congestion, giant papillae in both upper palpebral conjunctiva (Figure 1a), limbal gelatinous membrane and presence of ropy discharge (asymmetrically -more in right eye) was found. On slit lamp examination, giant papillae giving cobblestone appearance was found (Figure 2). Corneal examination reveals punctate epithelial keratopathy with a circular to oval shape macular corneal opacity of approximately size 3x3 mm in right pupillary area (Figure 1b and Figure 3).

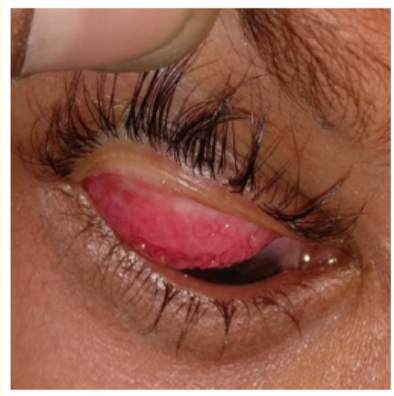

Figure 1a: *Torch light examination showing giant papillae in upper palpebral conjunctiva OD.*

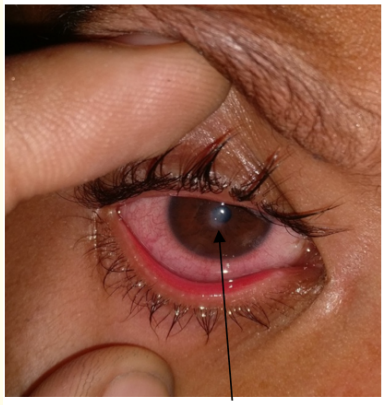

Figure 1b: *Central macular corneal opacity in pupillary area ,conjunctival congestion,and matted eyelashes.*

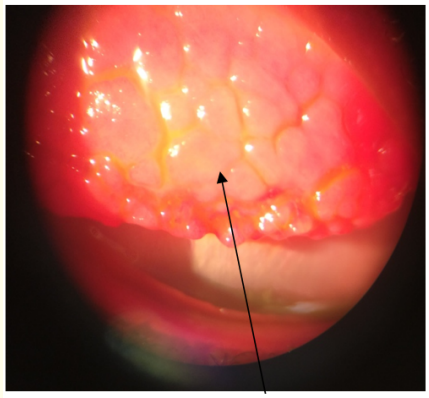

Figure 2: *Slit lamp examination showing giant papillae in upper tarsal conjunctiva giving cobblestone appearance.*

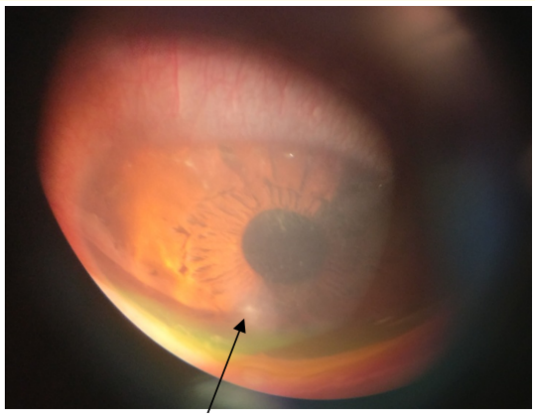

Figure 3: *Showing macular corneal opacity ,punctate epithelial keratopathy, and conjunctival congestion.*

Child was started on topical olopatadine 0.1% and ketorolac (0.5%) combination eye drops BD, carboxymethylcellulose 0.5% eye drops 4 times, homatropine 2% eye drops BD, ointment chloramphenicol and polymyxin B sulphate at bed time, tablet vitamin C 500mg OD and tablet levocetirizine 5 mg at bed time. Child improved in a span of 2 weeks (An informed and written consent was taken from the father of the child before taking photographs and eliciting information for publication).

Discussion

The corneal epithelium has a barrier function, which secludes the corneal stroma and corneal keratocytes from the inflammatory environment of the conjunctiva. However, this barrier function may be damaged in VKC. Eosinophils and eosinophil derived factors are responsible for the evolution of corneal ulcers. The two granule proteins that are secreted from activated eosinophils are Major Basic Protein and Eosinophil Cationic Protein. These two proteins are cytotoxic to corneal epithelial cells in vitro, and inhibit corneal epithelial wound healing in an organ culture model [3,4], and are found at the base of shield ulcers and plaques in VKC [5,6]. Shield ulcers and plaques in VKC usually evolve at the upper third of the cornea. The reported incidence among patients with VKC is 3 - 11%, with a subsequent permanent reduction in visual acuity in 6% of all patients [1]. In a recent large series of shield ulcers from India, the overall incidence of ulcers among patients with VKC was 4.6%, and the annual incidence ranged from 3 to 8% [7]. These are chronic epithelial abnormalities, which result from a combination of two mechanisms [5,7]. The first is the mechanical damage caused by the constant friction between the giant papillae of the upper tarsal conjunctiva against the upper third of the corneal epithelium. The second mechanism involves secretion of inflammatory mediators from activated eosinophils, which infiltrate the conjunctiva. These mediators include the Major BasicProtein and the Eosinophil Cationic Protein, which are toxic to the corneal epithelium [5]. The secreted proteins accumulate with time on the denuded stromal surface of the ulcer, thus forming a dense plaque, which prevents epithelialization.Chronic corneal ulcers and plaques in VKC can lead to further complications such as microbial keratitis [8,9], amblyopia [8,10], and very rarely a corneal perforation [11].

Chronic corneal ulcers and plaques in VKC can lead to further complications such as microbial keratitis [8,9], amblyopia [8,10] and very rarely a corneal perforation [11]. The evolution of corneal ulcers and plaques was described by Cameron, who presented a large series of 66 ulcers in 55 eyes [12]. Punctate epithelial erosions, which evolve into coarse erosions, and later coalesce to form macro-erosions, precede the development of shield ulcers (Table 1).

Microbial keratitis is one of the most severe complications of VKC [8,9,13-15], usually resulting from infection of recurrent shield ulcers [11]. The incidence of infections is 9 - 10% among eyes with shield ulcers, as reported in the two large series form India and Saudi Arabia [11,12].

Epithelial keratopathy	Corneal ulcers
Punctate erosions Coarse keratopathy Macroerosion	Grade 1 – Transparent ulcer base Grade 2 – Translucent ulcer base opaque white or yellow deposits Grade 3 – Elevated plaque

Table 1*: Development of shield ulcers and plaques [12].*

Since the child was a known case of recurrent VKC since long time, when presented to us during this episode, had severe inflammation along with a pre-formed macular corneal opacity.We were able to provide relief from allergic symptoms but the long term visual acuity was not so good due to central macular corneal opacity.

Conclusion

Although VKC is primarily an inflammatory disease of the conjunctiva, the cornea may be involved in a significant number of patients, causing complications such as shield ulcers and plaques, infectious keratitis, keratoconus, LSCD, and scarring. These may cause temporary visual loss, and if not managed aggressively, may result in permanent visual loss. As treatment is available to most of these complications, it is important to recognize these problems and treat them accordingly.

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