Case Report

Insights into recurrent bilateral double-headed hereditary pterygium: A case study

Abstract

Introduction: Pterygium is a fibrovascular growth of the palpebral conjunctiva extending over the corneal surface. It is more common in dry, dusty, windy environment. Sun exposure acts as an inciting factor for inflammation to occur and leading to pterygium development. However, bilateral double-headed hereditary pterygium presents a rare and intriguing manifestation.

Observation: Here we present a case of a 64 year old male with recurrent, bilateral double headed pterygium, shedding light on its hereditary predisposition and potential impact on the posterior segment. He was a field worker and a non-smoker with no history of trauma, systemic condition or any surgery except the pterygium excision in his left eye 6 years back. He also has increased retinal thickness with disc and macular oedema and inferior exudative retinal detachment.

Conclusion: Pterygium may have a hereditary predisposition and the inflammatory and degenerative involvement of the anterior segment in pterygium may also affect the posterior segment thus emphasizing the multifactorial nature of pterygium development

Keywords: bilateral, double-headed, hereditary, pterygium, recurrence, genetics, retinal diseases

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Abbreviations: UV, ultra violet; LE, left eye; RE, right eye; OD, right eye; OS, left eye; IOP, Intra-ocular pressures; OCT, Optical coherence tonometry; TGFβ, transforming growth factor beta; MMPs, matrix metalloproteinases; VEGF, vascular endothelial growth factor

Introduction

Pterygium, is a wing-shaped fibrovascular growth of the conjunctiva across the limbus onto the cornea,[1] and is divided into the head, the neck, and the body. The cap is the pigmentation line in front of the pterygium head and is deep to the epithelium, in the Bowman's membrane.[2]

Pterygium is seen in all countries of the world but its prevalence is higher in a country like India as it is a part of the "pterygium belt" located between 30° north and 30°south of the equator.[3]

The main histopathological changes in pterygium is elastotic degeneration of the conjunctival collagen.[4] Pterygium is usually seen to be occurring on the nasal side, may be because of the light coming to the temporal cornea and getting focused on the nasal cornea.[5] Double-head pterygium, that is, nasal and temporal pterygia in the same eye is rare. The incidence was found to be only 2.5% in a study by Dolezalová et al.[6] While bilateral double headed form is scarcely seen.[7] There are only two papers in the literature reporting bilateral symmetrical involvement of the nasal and temporal cornea.[8,9]

Various factors have been reported to be responsible for Pterygium development like exposure to ultra violet (UV) light which is reported to induce proinflammatory cytokines, chronic inflammatory cells, and growth factors[10] and may also be causing DNA damage in predisposed individuals.[11] Hereditary predisposition,[11] inflammation[12] and fibro vascular proliferation[13] are other factors in pterygium occurrence. But how they all are integrated is still not clear. In this manuscript, an interesting rare case of a bilateral double-headed recurrent pterygium is presented along with review of literature.

Case report

A 64 year-old male patient, came to our hospital with complaints of foreign body sensation in both eyes for two to three years along with blurred vision for last 10-12 months. He also complained of a mass in his eyes which he observed in his in left eye (LE) first almost 8 years back for which he was operated somewhere but it recurred after sometime. After 1-1.5 years he observed the similar problem in his right eye (RE) too. He was a field worker, non-smoker and had a family history of pterygium. He said that his father and brother also had the similar picture of growth on both sides of both eyes while his mother had unilateral nasal pterygium. His best corrected visual acuity was 6/24 OD (Right Eye) and 6/36 OS (Left Eye). On slit-lamp examination, he had bilateral double headed pterygium (Figure 1).

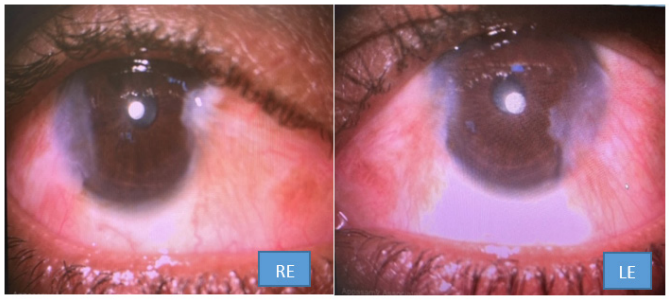

Figure 1 Bilateral double headed pterygium.

Schirmer test type 1 values were 10 and 12 mm respectively in RE and LE. Intra-ocular pressures (IOP) were14 and 16 mmHg by noncontact tonometer respectively in right and left eyes.

In addition to anterior segment findings, the patient demonstrated significant posterior segment involvement in the right eye. Fundus examination revealed an **inferior exudative retinal detachment** extending from the 5 to 8 o'clock position without retinal breaks. (Figure 2) The macula showed loss of foveal reflex with associated

retinal thickening. OCT imaging confirmed **cystoid macular edema (CME)** with increased central macular thickness and disc edema suggestive of inflammatory or vascular etiology. No vitreoretinal traction was observed (Figure 3) (Figure 4). The absence of retinal tears or tractional components suggested a non-rhegmatogenous etiology. Systemic evaluation did not reveal hypertension, diabetes, inflammatory, or neoplastic causes.

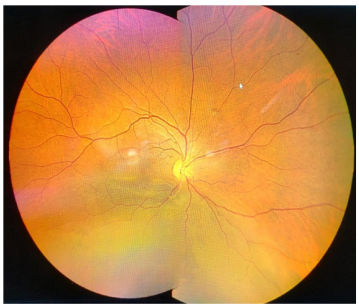

Figure 2 Inferior Retinal Detachment RE.

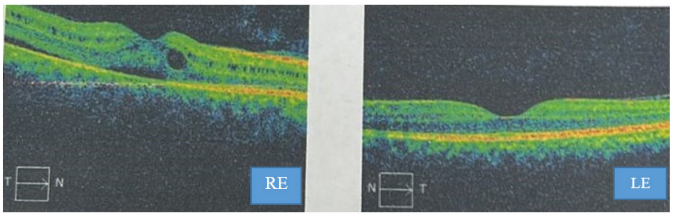

Figure 3 OCT images of Macular thickness.

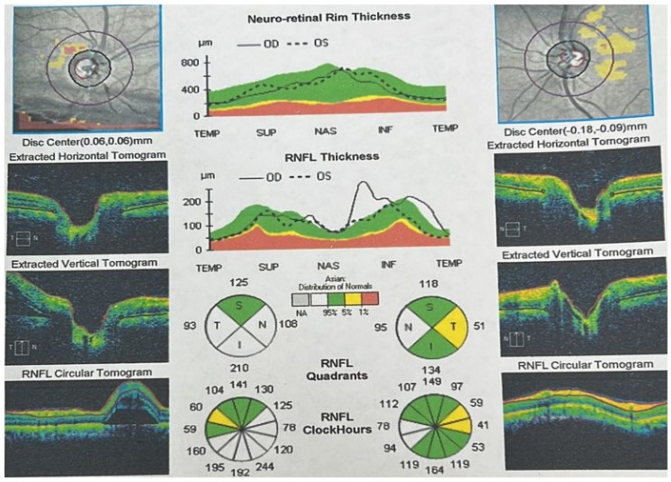

Figure 4 OCT image of RNFL thickness.

Considering the recurrent bilateral double-headed pterygium with significant visual impairment and associated posterior segment pathology, a staged management approach was adopted. The patient was first referred to the retina service where he was started on topical NSAIDs, topical corticosteroids and Oral anti-inflammatory therapy. Close monitoring with serial OCT was done. As the retinal detachment was exudative and shallow without macular-threatening progression, conservative management was preferred initially. Ocular surface inflammation was managed prior to surgery using lubricating eye drops for associated dry eye and UV protection advice was given.

After stabilization of macular edema, surgical excision was planned sequentially. A conjunctival limbal autograft with intraoperative Mitomycin-C (0.02%) was performed in the left eye first (less posterior involvement). The graft was secured using fibrin glue to reduce postoperative inflammation. Surgery for the right eye was deferred until posterior segment stability was confirmed. Post operatively he was prescribed topical steroid-antibiotic combination for 6 weeks, lubricating eye drops and an advice for regular OCT follow-up for right eye macular edema. At 3-month follow-up, the left eye showed no recurrence. The right eye demonstrated reduction in macular thickness with stable inferior detachment.

Discussion

This case was reported because of the rare incidence of hereditary, bilateral, recurrent and double sided Pterygium.

In some studies, the primary risk factors for pterygium were reported as old age, male sex, ultraviolet exposure, outdoor work and smoking.[14,15] In contrast to above studies, one study did not find outdoor occupations as a risk factor for pterygium.[16] Family history of pterygium and blepharitis are also found to be risk factors.[17] Our patient was a male field worker of age 64 years with a positive family history, but a non-smoker with no blepharitis. One study[18] mentioned a strong association between dry eye and pterygium and dry eye was an accompanying entity in our patient too.

Pterygium has a high recurrence rate and its prevention has not been successful because the pathogenesis of pterygium is still not very clear. Multiple theories are there to describe the pathogenesis of pterygium. It was hypothesized that exposure to excessive sunlight is correlated with collagen degeneration but this has now been discredited as a mechanism of pterygium development as some studies have shown that the pterygium is infrequent in individuals highly exposed or a lower exposure may be frequent in the pterygium patients,[19] so it seems that the level of sunlight exposure may not be important for the pterygium to occur, as can also be seen in our report where although the patient is an outdoor worker but his brother and father were indoor workers.

It may be that ultraviolet light acts as a trigger for pterygium development in those predisposed to pterygium formation by inducing pro-inflammatory cytokines. However, the degree of induction varied in pterygia exposed to the same level of ultra violet light[10] but inflammation which is thought to be a type of hypersensitivity,[20] has been proposed to be the final step in the formation of pterygium though the exact mechanism is still not clear. Inflammation activates transforming growth factor beta (TGFβ)[21] which stimulates the fibroblast to synthesize collagen[22] which is deposited as fleshy pterygium. TGFβ also inhibits Matrix Metalloproteinases (MMPs)[23] and this inhibition minimizes collagenolysis.[24] Transforming growth factor β upgrades the VEGF (vascular endothelial growth factor)[25] which stimulates neo-vascularization hence inflammation in addition also promote the vascularisation on pterygium.

The coexistence of inferior exudative retinal detachment and cystoid macular edema in this case is unusual and not classically associated with pterygium. Exudative retinal detachment typically results from breakdown of the outer blood-retinal barrier due to inflammatory, vascular, or neoplastic causes. Similarly, cystoid macular edema arises from increased vascular permeability mediated by inflammatory cytokines such as VEGF and prostaglandins. It is plausible that in genetically predisposed individuals, a heightened inflammatory milieu may extend beyond the anterior segment, contributing to posterior segment vascular permeability changes.

However, a direct causal relationship cannot be definitively established. The posterior segment findings may represent a

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coincidental inflammatory or idiopathic exudative process. The coexistence raises the possibility of shared inflammatory mediators contributing to both anterior and posterior segment pathology. Further studies are required to determine whether chronic ocular surface inflammation may influence posterior segment vascular stability.

In this case report, three of the members of the same family were having bilateral double headed pterygium while the fourth member that is the patient's mother had only nasal pterygium on one side. This shows that heredity could be associated with the pterygium occurrence. Heredity involvement in pterygium development has been acknowledged in literature but under-emphasized.[26,27] Mode of inheritance may be Mendelian or non Mendelian. Mendelian inheritance may be autosomal dominant, autosomal recessive or sex linked while non Mendelian inheritance maybe multifactorial or mitochondrial. Pterygium inheritance is reported to be autosomal dominant based on a study of one or two families. But if there is incomplete penetrance sometimes it leads to skipped generation[28] as may be the case in our report as none of the children of our patient was having pterygium till date. In multi factorial inheritance, genes may interact with environment,[29] or, two or more genes coding for different proteins in different loci may modify one another's effect to produce phenotypes.[30] Affected individuals then tend to cluster in families.

Management of primary double-headed pterygium requires careful surgical planning to minimize recurrence. Options include, Bare sclera excision (high recurrence rate 30–80%), Conjunctival autograft (gold standard; recurrence 2–15%), Amniotic membrane transplantation, Adjunctive Mitomycin-C, Fibrin glue-assisted graft fixation. For double-headed pterygium, techniques which may be incorporated are split conjunctival autograft, vertical or horizontal graft division, two separate conjunctival grafts. In recurrent bilateral double-headed pterygium, management becomes even more challenging due to conjunctival scarring, and higher fibrovascular proliferation. So, the preferred strategies in recurrent cases include Limbal conjunctival autografting to restore limbal stem cell barrier; Intraoperative Mitomycin-C (0.02% for 2–3 minutes); Use of amniotic membrane when conjunctiva is insufficient; Fibrin glue to reduce surgical time and inflammation and strict postoperative anti-inflammatory regimen (topical steroids tapered over 6–8 weeks). Meticulous removal of Tenon's tissue and fibrovascular remnants is critical in recurrent cases.

Conclusion

This case highlights the rare occurrence of hereditary recurrent bilateral double-headed pterygium with concomitant posterior segment pathology. While pterygium is primarily an anterior segment disorder, chronic inflammation and genetic predisposition may contribute to a broader ocular involvement. Management of recurrent bilateral double-headed pterygium requires a multimodal strategy including limbal conjunctival autografting, judicious use of Mitomycin-C, and long-term inflammation control. Careful evaluation of posterior segment findings is essential before surgical intervention. A multidisciplinary approach ensures optimal anatomical and functional outcomes.

Limitations

Genetic analysis of the patient and his family members was not done. Also the ocular picture of father and brother was not possible as father was not alive and brother was living in different state.

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None.

Declaration

a) Ethical Approval and Consent to participate: NA

b) Consent for publication: "Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the editor-in- chief of this journal.

c) Availability of supporting data: Pubmed, Google scholar

d) Competing interests: None

e) Authors' contributions: All helped in literature search, documentation of case and writing of this case report

Conflict of interests

The authors declare that they have no known competing financial interests or personal relationships that appeared to influence the work reported in this study.

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References

1. Dushku N, Reid TW. Immunohistochemical evidence that human pterygia originate from an invasion of vimentin-expressing altered limbal epithelial basal cells. *Curr Eye Res.* 1994;13:473–481.
2. Dushku N, Hatcher SL, Albert DM, et al. P53 expression and relation to human papillomavirus infection in pingueculae, pterygia, and limbal tumours. *Arch Ophthalmol.* 1999;117(12):1593–1599.
3. Kim KW, Park SH, Kim JC. Fibroblast biology in pterygia. *Exp Eye Res.* 2016;142:32–39.
4. Spencer WH. *Ophthalmic Pathology: An Atlas and Textbook.* 3rd ed. Vol 1. Philadelphia, PA: W.B. Saunders; 1985:174–176.
5. Maloof AJ, Ho A, Coroneo MT. Influence of corneal shape on limbal light focusing. *Invest Ophthalmol Vis Sci.* 1994;35:2592–2598.
6. Dolezalová V. Is the occurrence of a temporal pterygium really so rare? *Ophthalmologica.* 1977;174:88–91.
7. Elhamaky TR, Elbarky AM. Outcomes of vertical split conjunctival autograft using fibrin glue in treatment of primary double-headed pterygia. *J Ophthalmol.* 2018;2018:9341846.
8. Kurtul BE, Kakac A, Karaaslan A. Bilateral double-headed recurrent pterygium: a case presentation and literature review. *Med Hypothesis Discov Innov Ophthalmol.* 2020;9(2):85–90.
9. Wolter-Roessler E, Seitz B, Naumann GO. Pterygoid corneal dystrophy. *Klin Monbl Augenheilkd.* 2002;219(9):677–681.
10. Di Girolamo N, Wakefield D, Coroneo MT. UVB-mediated induction of cytokines and growth factors in pterygium epithelial cells involves cell surface receptors and intracellular signaling. *Invest Ophthalmol Vis Sci.* 2006;47(6):2430–2437.
11. Young CH, Lo YL, Tsai YY, et al. CYP1A1 gene polymorphism as a risk factor for pterygium. *Mol Vis.* 2010;16:1054–1058.
12. Golu T, Mogoanta L, Streba CT, et al. Pterygium: histological and immunohistochemical aspects. *Rom J Morphol Embryol.* 2011;52(1):153–158.
13. Kria L, Ohira A, Amemiya T. Immunohistochemical localization of basic fibroblast growth factor, platelet-derived growth factor, transforming growth factor-beta and tumor necrosis factor-alpha in the pterygium. *Acta Histochem.* 1996;98(2):195–201.

14. Nangia V, Jonas JB, Nair D, et al. Prevalence and associated factors for pterygium in rural agrarian central India: The central india eye and medical study. *PLoS One.* 2013;8(12):e82439.

15. Liu L, Wu J, Geng J, et al. Geographical prevalence and risk factors for pterygium: a systematic review and meta-analysis. *BMJ Open.* 2013;3(11):e003787.

16. Akinsola FB, Mbadugha CA, Onakoya AO, et al. Pattern of conjunctival masses seen at Guinness Eye Centre LUTH Idi-Araba. *Nig Q J Hosp Med.* 2012;22(1):39–43.

17. Malekifar P, Esfandiari H, Behnaz N, et al. Risk factors for pterygium in Ilam Province, Iran. *J Ophthalmic Vis Res.* 2017;12(3):270–274.

18. Roka N, Shrestha SP, Joshi ND. Assessment of tear secretion and tear film instability in cases with pterygium and normal subjects. *Nepal J Ophthalmol.* 2013;5(1):16–23.

19. Forsius H, Maertens K, Fellman J. Changes of the eye caused by the climate in Rwanda, Africa. *Ophthalmic Epidemiol.* 1995;2(2):107–113.

20. Hill JC, Maske R. Pathogenesis of pterygium. *Eye (Lond).* 1989;3(Pt 2):218–226.

21. Koli K, Saharinen J, Hyytiainen M, et al. Latency, activation, and binding proteins of TGF-beta. *Microsc Res Tech.* 2001;52(4):354–362.

22. Khalil N, Xu YD, O'Connor R, et al. Proliferation of pulmonary interstitial fibrosis is mediated by transforming growth factor-beta1-induced release of extracellular fibroblast growth factor-2 and phosphorylation of p38 MAPK and JNK. *J Biol Chem.* 2005;280(52):43000–43009.

23. Ma C, Nasser C. Regulation of matrix metalloproteinases (MMPs) and their tissue inhibitors in human myometrial smooth muscle cells by TGF-β1. *Mol Hum Reprod.* 1999;5(10):950–954.

24. Guo P, Zhang SZ, He H, et al. PTX3 controls activation of matrix metalloproteinase-1 and apoptosis in conjunctivochalasis fibroblasts. *Invest Ophthalmol Vis Sci.* 2012;53(7):3414–3423.

25. Nagineni CN, Samuel W, Nagineni S, et al. Transforming growth factor-beta induces expression of vascular endothelial growth factor in human retinal pigment epithelial cells: involvement of mitogen-activated protein kinases. *J Cell Physiol.* 2003;197(3):453–462.

26. Tsai YY, Bai DT, Chiang CC, et al. Pterygium and genetic polymorphism of DNA double-strand break repair gene Ku70. *Mol Vis.* 2007;13:1436–1440.

27. Carmichael TR. Genetic factors in pterygium in South Africans. *S Afr Med J.* 2001;91(4):322.

28. Defazio G, Martino D, Aniello MS, et al. A family study on primary blepharospasm. *J Neurol Neurosurg Psychiatry.* 2006;77(2):252–254.

29. Fukumura T, Kose H, Takeda C, et al. Genetic interaction between hyperglycaemic QTL is manifested under a high-calorie diet in OLETF-derived congenic rats. *Exp Anim.* 2011;60(2):125–132.

30. Savage DB, Agostini M, Barroso I, et al. Digenic inheritance of severe insulin resistance in a human pedigree. *Nat Genet.* 2002;31(4):379–384.

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