Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/mjafi

Case Report

Post traumatic neovascularisation of a cataractous crystalline lens

Brig Sagarika Patyal, ^{SM^{a,*}}, Brig Sunita Kakkar, ^{vsm^b}, Brig V.S. Gurunadh ^c,
P.G. Pradeep ^d

^a Consultant (Ophthalmology), Base Hospital, Delhi Cantt 10, India

^b Brig Med, Western Command, C/o 56 APO, India

^c Professor & Head, Dept of Ophthalmology, Armed Forces Medical College, Pune 411040, India

^d Ex-Resident, Dept of Ophthalmology, Armed Forces Medical College, Pune 411040, India

ARTICLE INFO

Article history:

Received 23 June 2013

Accepted 2 September 2014

Available online 25 November 2014

Keywords:

Neovascularisation

Lens

Cataract

Introduction

The crystalline lens is an avascular structure and its neovascularisation is rarely seen and reported in literature. We report a case of a mature cataract with neovascularisation in a 69 years old male patient.

Case report

A 69 years old male patient reported with painless progressively increasing diminution of vision of the left eye of three months duration. There was no ocular history of redness,

lacrimation, chronic topical drug usage, trauma or previous ocular surgery. There was no systemic history of diabetes mellitus, hypertension or coronary artery disease.

On examination, he was found to have vision of 6/9 in the right eye with normal anterior and posterior segments and normal intraocular pressure. In the left eye, vision was only perception of light with accurate projection of rays. He had a 2–3 mm raised, brownish pigmented nodular lesion about 3 mm superior to the limbus at 11 o'clock position surrounded by vascularisation (Fig. 1). The crystalline lens was a mature cataract with no signs of subluxation. No neovascularisation of the iris was visible. On instillation of mydriatics the pupil dilated fully except for posterior synechiae at 5 and 7 o'clock positions (Fig. 2). A sheaf of fine blood vessels was seen on the anterior lens capsule at 11 o'clock and the anterior lens capsule was also opacified in that area, in comparison to the rest of the capsule. There was no circumcorneal ciliary congestion, keratic precipitates or pigments on the endothelium. Gonioscopy did not reveal abnormal findings. Intraocular pressure was normal by Applanation tonometry. There was no fundal glow and B-Scan of the left eye showed an attached retina or with no abnormalities in the vitreous.

A scraping was taken from the nodular lesion on the sclera for histopathological examination as the patient was elderly and malignancy had to be ruled out, but no malignant cells were found and only a few benign epithelial cells and inflammatory cells were found. Routine investigations consisting of haemogram, Blood sugar, both fasting and post prandial, ECG, were found to be normal.

* Corresponding author. Tel.: +91 9643437024 (mobile).

E-mail address: sagarikapatyal@gmail.com (S. Patyal).

<http://dx.doi.org/10.1016/j.mjafi.2014.09.001>

0377-1237/© 2014, Armed Forces Medical Services (AFMS). All rights reserved.

Fig. 1 – Curved arrow shows raised brownish pigmented lesion on sclera. Block arrow shows vascularisation of anterior lens capsule.

Patient was taken up for phacoemulsification and posterior chamber Intraocular lens implantation on 14 Oct 2008 but the capsulorhexis could not be done at 11 o'clock position due to capsular thickening and the capsulotomy had to be completed by the can opener technique. Due to the ragged edges of the capsulotomy and intraoperative bleeding from the cut blood vessels on the anterior lens capsule, phacoemulsification was

Fig. 2 – Long arrow shows anterior lens opacification, notched arrow shows posterior synechia.

Fig. 3 – Arrow shows deposits on the posterior lens capsule.

abandoned and the surgery was converted into conventional extracapsular cataract extraction. The anterior capsule was sent for histopathological examination. Histopathological examination revealed small proliferating capillary vessels with scattered chronic inflammatory cells (Fig. 4).

The lens nucleus was extracted in toto. The posterior capsule was found to be thickened with whitish granular deposits (Fig. 3). A posterior capsulotomy was done as Yag Laser capsulotomy would have required more energy to break such a thickened capsule and a rigid posterior chamber intraocular lens was implanted in the bag. The limbal wound was closed by interrupted 10/0 monofilament nylon sutures.

The patient had an uneventful post operative recovery having been treated with topical antibiotic steroid combination – Ciprofloxacin with dexamethasone 8 times left eye X2 days, followed by tapering doses, after surgery. There was no uveitis or bleeding in the anterior chamber

Fig. 4 – HPE (H&E stain) of anterior lens capsule showing small proliferating capillary vessels with scattered chronic inflammatory cells.

postoperatively. The patient regained 6/9 vision after removal of limbal sutures after eight weeks. Examination of the retina postoperatively did not reveal any angiogenic source.

Discussion

The normal adult crystalline lens is an avascular structure primarily to ensure lens clarity and therefore a clear refractive medium for optimum vision. During the early stages of lenticular development, it is opaque due to the presence of blood vessels but as the blood vessels regress, the lens becomes transparent.

There are very few reports in literature describing neovascularisation of the crystalline lens. Review of literature through search engines Yahoo, Google, Pubmed has revealed only three such reports-three case reports^{1–3} and an analysis of four case reports.⁴

Our patient reported with progressively increasing diminution of vision of only the left eye with good vision of the right eye. No history of ocular trauma was elicitable. He was found to have four clinical findings which suggested that the mature cataract of the left eye could have been complicated one: presence of two synechiae inferiorly, sheaf of blood vessels on the anterior lens capsule in the superior half of the lens, localised thickened area of anterior lens capsule and a raised pigmented lesion on the sclera near the limbus in line with the blood vessels. Penetrating injury to the lens anterior or posterior capsule causes hydration resulting in cataracts.⁵ Sometimes the capsular break is so small that fibrosis seals it leaving a small visually insignificant opacity which possibly occurred in this patient. The lenticular break may also release lens proteins causing phacoanaphylactic uveitis which probably did not occur in this case as there was fibrosis of the capsule.⁶ Uveitis occurred in this patient evident by the presence of synechiae. There was no sign of active uveitis. Fundus could not be seen but the B-scan did not reveal any vitreous or retinal abnormality.

The cause of neovascularisation of the lens capsule was possibly due to ocular inflammation^{7,8} due to injury and due to

subsequent uveitis.⁹ Angiogenesis occurs when the balance between angiogenic and nonangiogenic factors favours the former.¹

The pathogenesis of the posterior capsule thickening with whitish deposits is difficult to explain. Such deposits are seldom seen. Possible explanation for such a condition could be inflammation or proliferation of epithelial cells from the anterior lens capsule which, over a prolonged period, caused localised deposits on the posterior lens capsule. Such a lesion has not been described in literature nor does its appearance resemble any type of posterior capsular thickening.

Conflicts of interest

All authors have none to declare.

REFERENCES

1. Hwang Yih-Shiou, lai Chi-Chun, Lee Jiahn-Shing. Posttraumatic neovascularisation in a cataractous crystalline lens. *J Cataract Refract Surg.* 2002;28:715–716.
2. Pangtey SM, Khokhar S, Sony P, Panda A, Sethi HS, Chandra P. Posttraumatic neovascularisation in a cataractous crystalline lens. *J Cataract Refract Surg.* 2003;29:859–860.
3. Patel P, Rodman J. Intralenticular neovascularisation in a cataractous lens. *Optometry.* 2012;30(4):125–126.
4. Kabat AG. Lenticular neovascularisation subsequent to traumatic cataract formation. *Optom Vis Sci.* 2011;88(9):1127–1132.
5. Hargis-Greenshield L, Sims L. *Emergencies in Eye Care.* Slack Incorporated; 1999:49 [Chapter 5].
6. Lee David, Hogginsbotham Eve. In: Lee David, Hogginsbotham Eve, eds. *Clinical Guide to Comprehensive Ophthalmology.* Thieme Medical Publishers; 1999:314.
7. Brown GC, Margargal LE, Schachat A, Shah H. Neovascular glaucoma: etiologic considerations. *Ophthalmology.* 1984;284.
8. Tombran-Tink J, Barnstable CJ. In: Tombran-Tink J, Barnstable CJ, eds. *Ocular Angiogenesis: Diseases, Mechanisms and Therapeutics.* Humana Press; 2006 [Chapter 17].
9. Henkind P. Ocular neovascularisation. *Am J Ophthalmol.* 1978;85:287–307.