Intra-ocular lens extrusion in a patient with corneal graft melting

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ABSTRACT

Purpose: Postoperative extrusion of an intraocular lens at a site unrelated to the surgical incision is a very rare complication. We report the case of a posterior chamber intraocular lens extrusion through the cornea eight years after a penetrating keratoplasty in a patient with spontaneous aseptic stromal melting.

Observation: A 77-year-old man was treated for pseudophakic bullous keratopathy with a penetrating keratoplasty complicated by chronic graft rejection and severe glaucoma. He referred to our emergency department eight years after the surgery. The examination showed that the pseudophakic lens optic had eroded completely through the donor cornea. The globe remained intact during the extrusion process. The patient underwent excision of the pre-intraocular lens tissue, removal of intraocular lens implant and capsular bag, liberation of synechia, anterior vitrectomy and corneal graft under general anesthesia. There was no complication during or after the surgery. Histologic study suggests that the intraocular lens optic was covered by conjunctival epithelium with malpighian metaplasia.

Conclusion: To our knowledge, it’s the first case of posterior chamber intraocular extrusion through ocular tissues following penetrating keratoplasty. This case emphasizes the importance of early identification of risk factors, strong postoperative follow up and good patient compliance and the need to minimize drug and surgery-induced iatrogenic effects. If the complication does ensue, early surgical intervention may prevent or minimize serious damage.

1. Introduction

Postoperative extrusion of an intraocular lens (IOL) through the surgical wound has been well documented. Most reported cases are related to ocular trauma and facial injuries.\textsuperscript{1} However, spontaneous transcorneal IOL extrusion at a site unrelated to the surgical incision is a very rare complication. In 1983 Cameron and Doughman described erosion of an iris-fixated IOL through a corneal graft.\textsuperscript{2} Anterior chamber IOL extrusion in the presence of intercurrent illness such as rheumatoid arthritis, glaucoma, or herpes zoster ophthalmicus\textsuperscript{3} and posterior chamber IOL extrusion following an episode of corneal ulcer\textsuperscript{4} have been described.

We report the case of a posterior chamber IOL extrusion through the cornea eight years after a penetrating keratoplasty (PK) in a patient with spontaneous aseptic stromal melting.

2. Case report

A 77-year-old man was referred to our emergency department complaining of tearing and decreased vision in his right eye (RE) for 3 months, without redness or pain. The patient underwent PK in the RE in 2012 for a pseudophakic bullous keratopathy (PBK). The surgery itself was uneventful but postoperative period was complicated by a chronic graft rejection and severe glaucoma. The patient was diabetic and hypertensive with no medical history of rheumatism or other autoimmune disease. He did not report any traumatic history. Best-corrected visual acuity was light perception in RE and 20/30 in LE. Slit-lamp exam of the RE revealed almost complete corneal graft melting and intra-ocular lens implant exposure (Fig. 1). Anterior segment optical coherence tomography (OCT) showed the exposure and anterior protrusion of the implant, covered by a thin tissue layer (Fig. 2). Schirmer test without anaesthetic was abnormal at 3 mm. Fundus was not visible. The LE was quiet, with posterior chamber pseudophakia.

The patient underwent excision of the preIOL tissue, removal of
intraocular lens implant and capsular bag, liberation of synechia, anterior vitrectomy and corneal graft under general anesthesia (Fig. 3). There was no complication during or after the surgery. The post-operative treatment consisted in topical corticosteroid tapered and ciclosporin 2% three times a day.

Microscopic examination of the tissue revealed that the anterior surface of the implant was covered by conjunctival epithelium with malpighian metaplasia. There were chronic inflammatory reaction signs. (Fig. 4).

Postoperatively, a slow process of re-epithelialization occurred proceeding from the donor limbal zone centripetally, until complete epithelialization was accomplished within 2 weeks. The fundus examination showed atrophic optic nerve probably explaining that visual acuity was still light perception.

Fig. 1. Photography of the right eye. We observed an exposure of the intra-ocular lens closing the anterior segment of the eye (1A).

Fig. 2. Anterior segment optical coherence tomography of the right eye. The exam confirmed the exposure and anterior protrusion of the implant, covered by a thin tissue layer of 44 μm.
3. Discussion

To our knowledge, only one case of implantable IOL extrusion through ocular tissues following PK has been reported. A series of events probably occurred, slowly leading to the corneal melting and implant extrusion while maintaining an intact, relatively uninfamed eye.

First, PK may have caused some degree of corneal denervation and complications might have occurred during the surgery creating corneal endothelial damage.

In addition, the chronic graft rejection and inadequate control of intra-ocular pressure (IOP) after the PK should lead to graft failure and severe corneal damage. Multiple factors could explain the intraocular hypertension (IOH) in this case. The PK itself can generate IOH by anatomical changes and inflammatory processes associated with the surgery and by the chronic use of topical steroid required. Then, it has been reported that the incidence of IOH after surgery is associated with the indications for PK and patients with PBK were shown to be at high risk for IOH. The IOL material and the effects of pseudophakia on the angle structures are also possible explanations for the IOH.

Moreover, the chronic use of antibiotic, steroid and glaucoma eye drops without strong follow-up, should participate to corneal decompensation by creating ocular surface toxicity and pro-inflammatory conditions.

Intraocular lenses should be designed to provide maximum stability with minimum tissue contact. However, in this case maybe there was mechanical compression or disruption of the tissue by the synthetic material during a prolonged period leading to local necrosis and release of inflammatory mediators, such as prostaglandins and oxidizing agents, that can be directly toxic to the corneal endothelium.

All of these factors probably contribute to chronic degenerative corneal disorder and neurotrophic keratopathy, responsible for the impairment in corneal healing and for the changes on the ocular surface that lead to the corneal melting, the migration of the implant through the tissue and finally the total transcorneal extrusion.

The anterior ocular surface was closed by the lens itself and by the formation of a thin layer of conjunctival epithelium in such way that the globe remained intact during the progress of extrusion. The slow rate of the process allowed the anterior re-epithelialization up to the emerging optic, limiting the risk of infection.

4. Conclusion

IOL extrusion is a rare condition with potentially severe complications. To prevent its occurrence, early identification of risk factors, strong postoperative follow up and good patient compliance are important. However, if the complication does ensue, early surgical intervention may prevent or minimize serious damage.

Patient consent

The patient consented to publication of the case.

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All authors attest that they meet the current ICMJE criteria for authorship.

The optic of the lens was resting on the anterior ocular surface so that the border of the lens optic coincided with the border of the receiver.

Fig. 3. Photography of the right eye just before surgery (A), during the lens extraction (B, C), and at the end of the procedure after corneal graft (D).

Fig. 4. Masson’s trichrome stain histological section of the pre-IOL tissue (magnification ×10). Microscopic examination showed malpighian metaplasia of conjunctival epithelium (arrow) and connective tissue (arrow head) with fibrosis and chronic inflammation.
The lens surface seemed to be keratinised (arrow). The receiver peripheral cornea was opaque and neovascularized. The anterior chamber was flat (1B). There was no obvious inflammatory signs.

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**Declaration of competing interest**

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