Photo Essay

Keratoprosthesis optic and carrier corneal graft "noncontact" as a cause of sterile stromal necrosis in a case of Auro KPro implantation

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Key words: Auro KPro, Boston keratoprosthesis, graft melt, keratoprosthesis, noncontact, stromal necrosis

Case Report

A 50-year-old female with bilateral vascularized corneal opacity and associated limbal stem cell deficiency developed in early childhood following a viral exanthema [Fig. 1a and b] underwent left eye extracapsular cataract extraction with primary implantation of aphakic Auro KPro (Auro KPro cornea; Aurolab, Madurai, Tamil Nadu, India), an Indian version of Boston keratoprosthesis (Boston KPro I; Massachusetts Eye and Ear Infirmary, Boston, MA, USA). There was no clinical evidence of dry eye, trachoma, or cicatricial pemphigoid. Schirmer score was 30 mm. Primary implantation of keratoprosthesis was considered as it was a high-risk case for graft failure because of heavily vascularized corneal opacity with associated limbal stem-cell deficiency. A 14-mm diameter bandage contact lens (BCL) was placed at the end of procedure and was continued in the postoperative period with monthly replacement. During the course, the patient had contact lens losses and BCL was replaced each time. She achieved best-corrected visual acuity of 6/60 with − 5.00 DS. An area of noncontact, however, was noted from 6 to 9 O′ clock hours between the KPro optic and carrier graft in the immediate postoperative period, evidenced by the presence of air bubble underneath the anterior rim of the optic [Fig. 1c]. Anterior segment optical coherence tomography (ASOCT) (Heidelberg Engineering GmbH, Germany) demonstrated a significant gap and entrapped air bubble [Fig. 1d]. After an initial uneventful clinical course, the patient reported with symptoms of discomfort and watering 4 months postoperatively at which time a localized area of ulceration in the carrier corneal graft at 7 O′ clock was detected [Fig. 1e]. The patient had contact lens loss at that time. Infection was ruled out by taking corneal scrapings from the bed and edges of melt which was negative for bacterial or fungal microorganisms. The area of melt progressively increased despite tarsorrhaphy and tenon's flap advancement [Fig. 1f].

Explantation of keratoprosthesis–carrier–graft complex and replacement with a new keratoprosthesis of same design and model and fresh carrier graft was then performed paying meticulous attention to the assembly of all the components. Keratoprosthesis optic was well apposed with no gap visible on slit lamp examination [Fig. 2a]. ASOCT at 3 months, however, still revealed a small gap, which was much less [Fig. 2b]. At 1 year, no gap was seen on ASOCT and keratoprosthesis–carrier graft assembly was well in place [Fig. 2c].

Discussion

A 16%–30% incidence of stromal necrosis with Boston keratoprosthesis type 1 has been reported[1-3] with sterile melts often being located centrally because of the farthest distance from aqueous humour for nutrition while peripheral melts may occur in cases with poor contact lens fitting.[4] In the present case, the large gap between the optic front plate and the carrier corneal graft probably led to inadequate wetting and tear film stasis in the affected area predisposing to desiccation and dellen formation with subsequent tissue necrosis and ulceration. Due to lack of biointegration of the PMMA device with the carrier corneal donor tissue, even in apparently well-apposed keratoprosthesis–corneal tissue assemblies, upto 44% of cases have been reported to have a measurable gap of 8–104 μm on ASOCT.[5] Although the extent of vertical gap likely to result in a significant increase of adverse events requires further studies, any potential space between the optic front plate or stem and carrier graft also remains a conduit for microorganisms or proteolytic enzymes responsible for carrier graft melt.[5] A meticulous attention to assembly of the keratoprosthesis–corneal tissue complex may help in reducing this complication of sterile stromal ulceration and subsequent necrosis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have

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Quick Response Code:

Website: www.ijo.in

DOI: 10.4103/ijo.IJO_1713_18

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Presented in part at: 27th APACRS (Asia Pacific Academy of Cataract and Refractive Surgeons) 13th–16th November, 2014, Jaipur, India.

Manuscript received: 13.10.18; Revision accepted: 08.01.19

Cite this article as: Malhotra C, Dhingra D, Jain AK. Keratoprosthesis optic and carrier corneal graft "noncontact" as a cause of sterile stromal necrosis in a case of Auro KPro implantation. Indian J Ophthalmol 2019;67:685-6.

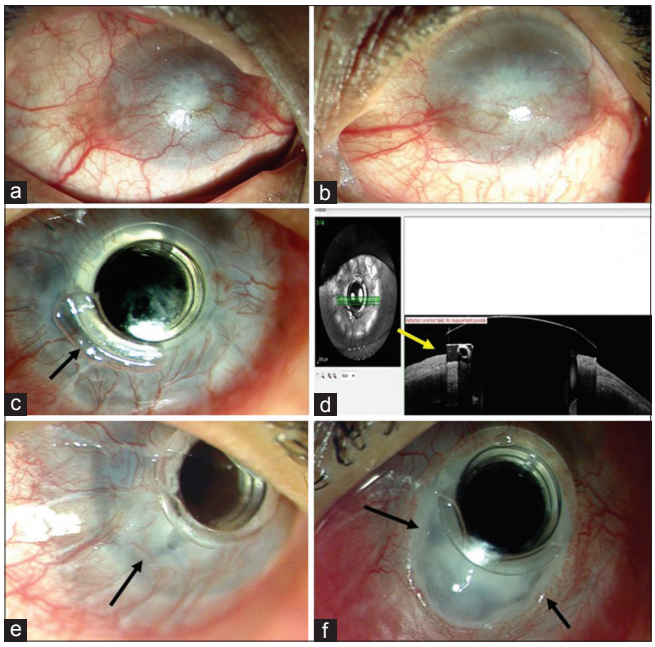

Figure 1: (a and b) Bilateral vascularized corneal opacity. (c) Area of noncontact from 6 to 9 O' clock with presence of air bubble visible beneath the KPro optic (arrow). (d) ASOCT showing potential space between optic and carrier graft with entrapped air bubble (arrow). (e) Area of graft melt at 7 O' clock position (arrow). (f) Area of graft melt from 6 to 9 O' clock hours (arrows)

given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

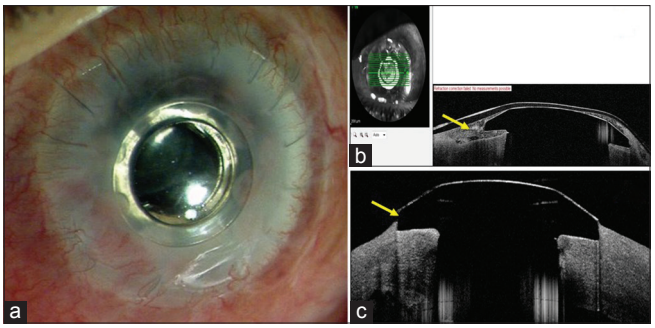

Figure 2: (a) Slit lamp photograph at 3 months showing well-apposed carrier graft and KPro optic after repeat surgery. (b) ASOCT at 3 months showing a small gap (arrow). (c) ASOCT at 1 year with well-apposed carrier graft and optic without any gap (arrow)

Conflicts of interest

There are no conflicts of interest.

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