

Case Report

Iatrogenic extreme corneal decompensation treated by sequential Descemet's Stripping Endothelial Keratoplasty surgeries six months apart

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ABSTRACT

Descemet's stripping endothelial keratoplasty (DSEK) is now the most common surgical procedure to treat endothelial dysfunction although it is known that endothelial cell survival is an issue of concern. We present a case whereby severe iatrogenic corneal decompensation caused by Descemet's membrane detachment following premature disconnection of an infusion tube at the end of a trans pars plana vitrectomy and epiretinal membrane peel was successfully treated with two staged DSEK procedures six months apart. The patient was counselled that due to the severity of his extreme corneal oedema more than one DSEK procedure may be needed and the procedure was planned from the outset as a two-stage procedure. There was a measurable decrease in corneal thickness and increase in visual acuity following both the first and second procedures, which may be due to reinvigoration of the endothelial cell count following each procedure. We suggest that repeating the DSEK procedure, even when the first operation has gone well and the graft appears healthy, may be beneficial in obtaining further improvement in cases of severe corneal oedema.

INTRODUCTION

The human cornea consists of three main histological layers; the inner endothelium, the outer epithelium, and the stroma sandwiched between the two. The stroma contains numerous glycosaminoglycans as well as proteoglycans, all of which have a strong osmotic pull and as the clarity of the cornea is dependent of keeping water away from these structures the endothelium has to constantly pump fluid out of the stroma in order to avoid corneal swelling, opacification, breakdown of the overlying epithelium and a blurring of the vision that results from all of these effects. Indeed these are the main signs of corneal decompensation due to endothelial failure.

Descemet's Stripping Endothelial Keratoplasty (DSEK), in which the patient's diseased endothelium is replaced with that of a donor, has become the treatment of choice for endothelial dysfunction and has now become the commonest operation performed in America for this purpose, being performed in 86% of patients with Fuchs' dystrophy, which is the commonest form of endothelial dystrophy^{1,2}. An area of concern with DSEK, as with the preceding treatment of penetrating keratoplasty (PK)³, in which all three layers of the cornea are replaced, is that endothelial cell survival in the

donor tissue is impaired compared with endothelial cells in the normal eye, and can lead to graft failure in up to 3.6% of patients within 5 years⁴. Indeed, the median 5 year endothelial cell loss rate was found to be 53%⁴, with 6 month and one year cell loss rates being greater than those seen with PK². It is thought that increased manipulation of the endothelium may be the reason behind this.

From this data it can be argued that extreme corneal decompensation would possibly best be served by performing a PK, with all the attendant slower recovery and more unpredictable result^{5,6}. We present a case whereby severe corneal decompensation caused by iatrogenically induced Descemet membrane detachment was treated by sequential planned DSEK operations, which we believe to be the first time this has been described in the literature.

CASE REPORT

An 82 year old patient undergoing a trans pars plana vitrectomy and epiretinal membrane peel of the left eye suffered collapse of the eyeball after an infusion line was prematurely cut. On the first post-operative day, severe corneal oedema was noted with a superior Descemet's detachment that had reduced his vision to hand movements from his pre-operative visual acuity of 0.48 LogMAR. On the LogMAR system of visual acuity measurement 0.00 is equal to 6/6 Snellen and 1.00 is equal to 6/60. Pachymetry, a measurement of the thickness of the cornea, measured 1148microns. The average value for corneal pachymetry is 555microns.

After monitoring the patient for three months, during which time no surgical treatment took place, the patient was referred to the corneal clinic where he was counselled that due to the severity of his corneal oedema more than one DSEK procedure may be needed. The first procedure was successfully carried out under a general anaesthetic a month later. This consisted of preparing the donor material on a Katena artificial anterior chamber with an 8.5mm trephine followed by host preparation including removal of

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what remained of the Descemet's membrane. The donor endothelium was mounted on a Busin glide and presented at the inferior incision, pulled to the correct position, centralised and the anterior chamber filled with air. Post operative recovery was uneventful on the usual regime of topical dexamethasone.

After the first DSEK procedure corneal pachymetry revealed improvement in thickness from 1145 microns to 995 microns, the unaffected right eye being 584 microns, with the patient noticing a marked improvement in vision, although objectively this amounted to counting fingers. There were no infections, graft dehiscence or significant change in intraocular pressure noted in the postoperative period. After six months he was seen to be showing only minimal signs of corneal thinning compared to the earlier post-operative period and it was clear that further improvement was very unlikely to occur. Therefore a second DSEK procedure was performed. At this procedure the previously transplanted endothelium was removed with a Reverse Sinsky Hook and a fresh donor Descemet's membrane placed.

This too was a successful operation in which no complications occurred and at three months postoperatively the vision in the left eye was much improved. The vision was noted at 0.80 LogMAR, improving to 0.70 with a pin hole (though no formal refraction was performed), with corneal pachymetry demonstrating thickness of 689microns in the left eye. At six months postoperatively the visual acuity remains stable at 0.78LogMAR, improving to 0.70 with a pin hole, with the pachymetry remaining stable at 681microns. A slight corneal haze persists but centrally the cornea was clear.

DISCUSSION

Here we present the case of a patient with very severe corneal decompensation who obtained some degree of improvement in corneal thickness following primary DSEK but then went on to further improve following repeat DSEK surgery. While it is known that endothelial survival rates are lower at both six months and one year after DSEK compared with after PK³ and that endothelial failure is one of the primary causes of graft failure with this mode of treatment³ the role of planned sequential DSEK's has not been previously explored.

Repeat DSEK surgery has been described by many authors, with one series examining the commonest reasons for this concluding that 24% of these were due to endothelial failure alone.^[6] Other surgeons have quoted higher failure rates due to endothelial failure, but of note is that even among clear grafts the endothelial cell count was noted to have fallen quite significantly, being 1078+/- 507 cells/mm² at one year follow up in one series⁷. Both corneal oedema and corneal thickness have been noted to improve following DSEK up to three months postoperatively before stabilising⁸ and it is possible that this is due to the rapid decline recorded in endothelial cells in the first few months following the procedure.

The vast majority of repeat DSEK operations have been undertaken because of endothelial graft failure⁶ but there have been a few described cases of graft exchange where problems

with the graft interface were thought to be responsible for the suboptimal visual acuity, though endothelial counts were not explored^{9, 10}. To our knowledge there have been no previously published reports of a second DSEK operation being performed in the presence of a functioning graft for the purpose of reinvigorating the endothelial cell count in a severely oedematous cornea.

There were no complications following the first DSEK procedure carried out on our patient with no graft detachment, which is the main factor associated with declining endothelial counts and graft failure⁷, with no other intraoperative or postoperative complications being noted either. It is of great regret that the exact endothelial cell count could not be obtained due to a lack of equipment at our hospital but based on these deductions there is no reason to suppose any unusual or unexpected cause of endothelial cell death was at play. Our deduction in presenting this case is that severely oedematous corneas may need more time to clear than the window offered by one DSEK procedure alone and so a repeat DSEK may be a viable option in obtaining further resolution of oedema. It is acknowledged however that more work needs to be done in this area before a more concrete recommendation is made, with emphasis placed on obtaining clear endothelial cell counts at each stage in the two stage 'double-DSEK' procedure in order to form a more conclusive view of what processes are at play.

The authors have no conflict of interest.

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