Retention of iris supported intraocular lenses at the time of penetrating keratoplasty for pseudophakic corneal oedema

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SUMMARY The long-term follow-up of patients undergoing penetrating keratoplasty for pseudophakic corneal oedema is reported. The cause and frequency of corneal decompensation following intracapsular cataract extraction with insertion of an iris supported lens was assessed and found to be unrelated to the implant in the majority of cases. The most common cause of decompensation was endothelial touch during the cataract extraction. In all eyes the intraocular lens was retained at the time of penetrating keratoplasty. The hazards of removal of the lens are discussed and the recommendations made that lenses be retained when penetrating keratoplasty is undertaken for pseudophakic corneal oedema, other than in exceptional cases.

Pseudophakic corneal oedema is one of the leading indications for penetrating keratoplasty. Its incidence may be higher in intracapsular cataract extraction with insertion of an iris clip lens than in extracapsular surgery with a posterior chamber lens.

This has led to the false assumption that the continued presence of an iris supported lens in the eye after penetrating keratoplasty will necessarily prejudice the survival of a corneal graft. Some authors have recommended that all iris supported lenses be removed at the time of penetrating keratoplasty for pseudophakic corneal oedema, though this remains a controversial issue.

We present the results of follow-up of all the patients under the care of the senior author and who required penetrating keratoplasty for pseudophakic corneal oedema over the period 1971–84. Particular attention is drawn to the original cause of endothelial decompensation necessitating the graft and of the subsequent survival of the graft in patients retaining the iris supported lens.

The results lead to the conclusion that removal of the intraocular lens is contraindicated except when conditions exist causing a shallow anterior chamber, with the resulting risk of endothelial touch.

Materials and methods

The records of 675 cases of intracapsular cataract extraction with the placement of a Binkhorst four-loop lens performed during the period 1971–84 were studied. The incidence of postoperative endothelial decompensation was noted as well as that of preoperative endothelial touch. The patients who subsequently underwent penetrating keratoplasty for corneal oedema from whatever cause were reviewed and the cause of their endothelial decompensation recorded.

All cataract surgery was performed by a standard technique, described by the senior author, with the use of chymotrypsin prior to cryoextraction of the lens. The intraocular lens was placed obliquely and, for maximum stability, prevented from rotating by a single iris suture round the upper, anterior loop of the lens. Two peripheral iridotomies were made.

The subsequent corneal grafting was undertaken by a number of surgeons, using either 16 interrupted 10/0 sutures or a combination of eight interrupted and one continuous. In all patients the intraocular lens was retained at the time of keratoplasty. In one patient an adhesion between the lens loop suture and the cornea was divided, and in another the anterior loops of the lens were removed owing to the shallowness of the anterior chamber and the suspicion of touch of the corneal endothelium. A further two patients had an extra 10/0 Prolene suture inserted to fix the inferior lens loop to the iris, for increased stability. One patient required a limited anterior vitrectomy owing to herniation of vitreous through the peripheral iridotomies at the time of
keratoplasty. No vitreous was present in the anterior chamber in the other cases.

The patients and their notes were reviewed between 6 and 70 months after keratoplasty (mean 24.5 months). Details of follow-up period are shown in Fig. 1. In all cases the endothelial status as assessed prior to cataract extraction was noted, the time following surgery at which endothelial decompensation was first noted, and the suspected factors contributing to it. The per- and postoperative complications of cataract surgery were studied and the current state of the corneal graft and visual acuity. The presence or absence of clinical cystoid macular oedema was recorded, as defined by the presence of visible macular oedema in association with reduction of visual acuity to 6/12 or below.

Results

Of 675 cataract extractions, endothelial decompensation causing a reduction of visual acuity to less than 6/12, and hence requiring penetrating keratoplasty, developed in 18. The reasons for the corneal decompensation following cataract surgery are listed in Table 1. The commonest cause was peroperative endothelial touch, the occurrence of which was always specifically recorded in the operation notes. Although endothelial touch was recorded in 35 of the 675 operations, it led to endothelial decompensation affecting vision in only six of the eyes, and the other 29 cases in which touch was recorded have not subsequently decompensated.

It is interesting to note that all six eyes had been operated on prior to the widespread use of sodium hyaluronate. It is also of importance that peroperative endothelial trauma is not usually immediately manifest: indeed the mean time between surgery and the first signs of decompensation was 35 months. Study of all intracapsular cataract extractions with insertion of Binkhorst lenses and followed up for more than five years showed some endothelial corneal decompensation in 2-1% of eyes at two years and 6-6% at five years. It was often peripheral only, and did not affect visual acuity. Clinically significant corneal decompensation, defined as that causing a reduction in visual acuity to less than 6/12, occurred in only 18 patients and was treated with penetrating keratoplasty.

The corneal graft remained clear and without further problems in 14 of the 18 patients. The reasons for graft failure in the other four are shown in Table 2. The two patients whose grafts opacified owing to raised intraocular pressure together with, respectively, herpes simplex infection and graft rejection, had successful repeat keratoplasty. The former achieved 6/60+1 acuity only, as a result of extensive

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Reasons for failure of four corneal grafts</th>
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<tbody>
<tr>
<td>Patient</td>
<td>Reason for failure</td>
</tr>
<tr>
<td>1</td>
<td>Endothelial failure secondary to raised intraocular pressure; herpes simplex infection</td>
</tr>
<tr>
<td>2</td>
<td>Immunological graft rejection</td>
</tr>
<tr>
<td>3</td>
<td>Peripheral anterior synchiae pulling lens loop to corneal endothelium</td>
</tr>
<tr>
<td>4</td>
<td>Lens touch due to postkeratoplasty wound leak and shallowing of the anterior chamber</td>
</tr>
</tbody>
</table>

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Table 3  Reasons for visual acuity of less than 6/12 after keratoplasty

<table>
<thead>
<tr>
<th>Pathology</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cystoid macular oedema</td>
<td>2</td>
</tr>
<tr>
<td>Glaucomatous nerve damage</td>
<td>2</td>
</tr>
<tr>
<td>Age related macular degeneration</td>
<td>2</td>
</tr>
<tr>
<td>Diabetic maculopathy</td>
<td>1</td>
</tr>
<tr>
<td>Opacuare corneal graft</td>
<td>2</td>
</tr>
</tbody>
</table>
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