Case of late-onset corneal decompensation after iris-fixated phakic intraocular lens implantation

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A 48-year-old myopic patient with bilateral anterior chamber depth of 3.1 mm and endothelial cell density (ECD) of 2525 cells/mm² and 2638 cells/mm² preoperatively had bilateral implantation of an Artisan iris-fixated phakic intraocular lens (pIOL). Five years postoperatively, unilateral corneal stromal edema was seen in a circumscribed area overlying the temporal ridge of the pIOL in the right eye; the ECD was 1631 cells/mm² and the pachymetry, 586 μm. Explantation of the pIOL was refused by the patient. Seven years postoperatively, the ECD was 413 cells/mm² in the right eye and corneal decompensation occurred. The progressive unilateral endothelial loss was explained by excessive rubbing of the eyes because of chronic itching and an anterior shift of the pIOL over the 7 years as demonstrated by anterior optical coherence tomography.


Corneal decompensation is a potential complication of angle-supported intraocular lenses (IOLs). With the introduction of the Artisan iris-fixated anterior chamber IOL (Ophtec BV), this serious long-term complication seemed to be resolved; several studies show stability of the endothelial cell density (ECD) up to 10 years after implantation. Following implantation of a phakic IOL (pIOL) in the anterior chamber, an initial decrease in ECD is expected, with stabilization in the following years.

We present a patient who had a unilateral ECD decrease that led to corneal decompensation occurring 7 years after pIOL implantation. The loss of endothelial cells was attributed to a more anterior position of the pIOL in the anterior chamber of the right eye and forceful rubbing of the eyes.

CASE REPORT

A 48-year-old woman with partial right-side amblyopia was treated with refractive surgery for anisometropic myopia. The best corrected visual acuity (BCVA) was 20/40 with a refraction of −14.00 −1.00 × 5 in the right eye and 20/20 with a refraction of −6.00 −2.50 × 5 in the left eye. The anterior chamber depth (ACD) (endothelium to lens distance) measured with ultrasonography was 3.1 mm bilaterally. A −14.00 diopter (D) concave–convex model Artisan pIOL was implanted in the right eye in 1998 and a −8.00 D Artisan pIOL was implanted in the left eye later that year. Postoperatively, the BCVA was 20/30 with a refraction of −1.0 −0.75 × 145 in the right eye and 20/20 with a refraction of +0.50 −1.50 × 20 in the left eye.

The initial ECD measured 2525 cells/mm² in the right eye and 2638 cells/mm² in the left eye (Noncon Robo, Konan Medical, SP 8000). The central corneal pachymetry was 593 μm and 595 μm, respectively. Annual clinical evaluations with ECD measurements were performed (Table 1). Four years postoperatively, corneal guttata were seen for the first time, with a greater expression in the right eye. A marked decrease in ECD was seen in the right eye; the mean was 1980 cells/mm². The ECD in the left eye had decreased to 2392 cells/mm², which was within the expected range of endothelial cell loss after pIOL implantation. Pachymetry of 615 μm and 616 μm, respectively, showed no corneal thickening. A slow endothelial loss was documented over the following year. No direct contributing factor for the decreased ECD level was established. Five years postoperatively, the first signs of corneal stromal edema were seen in a circumscribed area overlying the temporal ridge of the pIOL. At this point, the ECD was 1631 cells/mm² in the right eye with a central pachymetry of 586 μm. The patient was advised to come for more frequent follow-up visits to estimate the rate of endothelial cell loss and plan removal of the pIOL.
if the ECD loss became worse. Despite the advice to have more frequent follow-up visits, the patient returned one year later, at which point the ECD was 1066 cells/mm² in the right eye. Removal of the pIOL was discussed, but the patient refused this option. She decided not to return for follow-ups indefinitely.

Seven years after surgery, the patient presented with a BCVA of 20/200 and bullous keratopathy of the temporal side of the cornea in the right eye (Figure 1). The detailed history indicated that she had been chronically and forcefully rubbing her eyes because of itchiness. The ECD in the right eye was 413 cells/mm² and had also decreased in the left eye to 1748 cells/mm²; the central corneal pachymetry was 708 µm and 618 µm, respectively. The endothelium–optic distance and ACD were measured with anterior segment optical coherence tomography (OCT) (Visante, Carl Zeiss Meditec AG) before and after topical instillation of cyclopentolate 1%. The ACD (endothelium to natural lens distance) was 2.26 mm in the right eye and 2.49 mm in the left eye (Figure 2). The central endothelium to optic distance was 1.59 mm and 1.75 mm, respectively. The endothelium–optic distance measured without cyclopentolate at the edges of the optic was 1.36 mm temporally and 1.16 mm nasally in the right eye (Figure 3) and 1.48 mm and 1.40 mm, respectively, in the left eye.

Table 1. Annual ECD measurements after pIOL implantation in the right and left eyes.

<table>
<thead>
<tr>
<th>Time of Examination</th>
<th>Right Eye</th>
<th>Left Eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preoperative</td>
<td>2525</td>
<td>2638</td>
</tr>
<tr>
<td>Postoperative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 year</td>
<td>2364</td>
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<td>2358</td>
</tr>
<tr>
<td>4 years</td>
<td>1980</td>
<td>2392</td>
</tr>
<tr>
<td>5 years</td>
<td>1631</td>
<td>2341</td>
</tr>
<tr>
<td>7 years</td>
<td>413</td>
<td>1748</td>
</tr>
<tr>
<td>8 years</td>
<td>—</td>
<td>1695</td>
</tr>
</tbody>
</table>

ECD = endothelial cell density

![Figure 1](image1.png)
Figure 1. Localized corneal edema that developed over the temporal pIOL rim 7 years after implantation.

![Figure 2](image2.png)
Figure 2. A: Decrease in ACD from 3.1 mm to 2.26 mm in the right eye shown by OCT over a period of 7 years. B: Decrease in ACD from 3.1 mm to 2.49 mm in the left eye.

The corneal thickness directly above the nasal optic edge was 930 µm in the right eye. After instillation of cyclopentolate, the endothelium–optic distance was 1.54 mm temporally and 1.33 mm nasally in the right eye and 1.67 mm and 1.89 mm in the left eye, showing an increase in clearance after instillation of cycloplegic eyedrops.

A penetrating keratoplasty was performed in the right eye combined with removal of the pIOL. One year later, the ECD in the left eye was 1695 cells/mm². Although this endothelial cell loss of 3% within a single year was excessive 7 years post-operatively, it was less than the 25% loss in the previous

![Figure 3](image3.png)
Figure 3. Proximity of the pIOL rim to the corneal endothelium in the right eye—1.16 mm nasally and 1.36 mm temporally—shown by OCT.
2 years. The patient is being monitored at 3-month intervals to detect further endothelial cell loss, which would warrant pIOL removal.

**DISCUSSION**

Anterior chamber IOLs are divided into angle-supported IOLs and iris-fixated IOLs, which now exist in a rigid model and a flexible model. In the past, the angle-supported IOLs have been the only option for anterior chamber IOL placement, especially in phakic patients, but this has proven to have major drawbacks. Corneal decompensation has been a frequent complication with angle-supported AC IOLs, with an ECD loss of up to 20% in the first year. It was so common that when the Artisan iris-fixated pIOL was introduced, numerous studies of the endothelial safety of the pIOL were performed. The ECD changes after pIOL implantation range from severe endothelial cell loss to an increase in ECD. Most studies show an initial endothelial cell loss in the first 12 months, which does not significantly decrease more up to 24 months after implantation. Several short-term and a few recent long-term studies evaluating corneal ECD loss after Artisan pIOL implantation show a mean ECD loss of 0.7% to 11.7% over 3 years. In the only 10-year, long-term follow-up study of endothelial density loss after implantation, an ECD decrease of 0.6% was shown, confirming the safety of implantation of the Artisan pIOL. Most recent studies confirm the initial endothelial loss and the long-term stability of ECD, with a 8.3% to 9% endothelial cell loss after 5 years. A higher endothelial cell loss correlates with a shallower ACD before pIOL implantation.

The initial loss of endothelial cells after pIOL implantation can be explained by intraoperative trauma to the endothelium by operating instruments or the optic itself. An initial postoperative elevation in intraocular pressure or chronic anterior chamber inflammation can also account for the initial loss of endothelial cells. In the early development phase of the Artisan pIOL, the endothelium–optic touch that was seen in the biconvex models for myopic pIOL placement was a concern, but this was resolved by creating the current concave–convex model.

Our patient exhibited no clinical signs of extensive endothelial cell loss in the initial 4 years and had none of the mentioned complications that should have led to corneal decompensation. The late surge in ECD loss in this patient demonstrates the vulnerability of the corneal endothelium to an anterior chamber pIOL when changes in the ACD occur in combination with eye rubbing. For safety reasons, a preoperative ACD of 3.0 mm and a minimum ECD of 2000 cells/mm² have been recommended. A reduction of 28% to 34% in ACD has, however, been seen in patients receiving Artisan pIOLs for myopia. In our case, the ACD (endothelium–natural lens distance) decreased from 3.1 mm bilaterally to 2.26 mm in the right eye and 2.49 mm in the left eye. The endothelium–optic distance was 1.59 mm and 1.75 mm, respectively. This indicates that even with safe ACD parameters before implantation of an Artisan pIOL, the danger of endothelium–optic touch with subsequent endothelial damage is always present and should be taken into account for a life-long period.

Kim et al. recently reported a case of corneal decompensation in a patient with Artisan pIOLs in which incorrect enclavation of the pIOL resulted in movement of the IOL toward the posterior corneal surface. In our case, there were no signs of increased mobility of the pIOL with eye movements, which could explain the localized decrease in ECD. In theory, one could argue that in the current case, the decrease in the ACD was at least partially caused by the induced corneal edema. However, since the central corneal thickness increased by only 115 μm from preoperatively to 7 years postoperatively, the decrease in ACD of 840 μm cannot be explained by the increased corneal thickness. As mentioned, the ACD was measured by 2 different methods, ultrasonography and Visante OCT. Although these methods are highly correlated, ACD values cannot be regarded as interchangeable. However, a difference of almost 1.0 mm is unlikely to be induced by measurement bias. The variability in the endothelium–optic distance due to the accommodative state of the eye and the annual growth of the lens increases the risk to the endothelium over time. External factors such as forceful rubbing of the eye may enhance this potential risk for endothelial cell damage.

The IOL manufacturer advises a minimum endothelium–optic rim distance of 1.5 mm, similar to previous recommendations by Baikoff. In this case, the recommended distance was not maintained at the nasal aspect of the IOL in both eyes. Therefore, one would expect the corneal decompensation to begin at the nasal aspect of the cornea. The forceful rubbing of the right eye in a temporal to nasal movement could explain the edema starting on the temporal side of the right eye. The slowdown in the ECD decline in the left eye also reinforces the idea that chronic eye rubbing was a major factor in the ECD decrease.

Manual damage to the corneal endothelium by eye rubbing after Artisan pIOL implantation has been considered. Our case clearly illustrates this relationship through the location of the first signs of corneal edema (over the temporal rim of the pIOL) and the cessation of the endothelial cell loss after the patient realized the damage the rubbing had caused. Patients should be reminded that an IOL has been placed in the eye and that forceful eye rubbing can cause irreparable damage to
the corneal endothelium. In addition, we believe that conditions that lead to chronic irritation of the eye (seasonal allergic conjunctivitis or eyelid pathology) should be identified preoperatively and seen as a risk factor for Artisan pIOL implantation or managed aggressively if they develop postoperatively. The decrease in endothelial cell loss in our patient’s left eye suggests that with the right motivation and correct treatment of the cause of the irritation, patients can be persuaded to refrain from rubbing their eyes. Since specular microscopy endothelial cell counting is inherently inaccurate (an intermeasurement variability of 7% is reported24), we will carefully monitor the endothelial cell count in our patient to assess whether the rate of endothelial cell loss has truly slowed. Should the endothelial cell count drop below 1500 cells/mm², the pIOL will be explanted.

Artisan pIOL implantation remains a safe refractive surgery option for highly myopic patients. The course of this case should be seen as a reminder that long-term, yearly follow-up examinations are essential after pIOL implantation, not only to follow the ECD but also to measure changes in the anterior segment morphometric parameters using modern anterior segment imaging techniques.

REFERENCES

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