Late onset post-keratoplasty astigmatism in patients with keratoconus

L Lim, K Pesudovs, M Goggin, D J Coster

Aim: 10 eyes of 10 patients are reported where progression of keratoconus in the host cornea occurred more than 10 years after penetrating keratoplasty with resultant increase in astigmatism. The technique and results of graft refractive surgery in seven eyes are presented.

Methods: The clinical features and management of these patients were retrospectively analysed. Graft refractive surgery involved an incision at the graft-host junction adjacent to the host thinning with compressive resutting. Astigmatic changes were calculated using vector analysis.

Results: There were seven men and three women with a mean age of 41.2 years. The average age when undergoing penetrating keratoplasty in the affected eye was 28.4 years and the average time after penetrating keratoplasty until keratoconus appeared in the host cornea defined by host thinning was 13.5 years. The mean cylinder power before host thinning was noted was 5.07D (SD 2.19) and the mean time after host thinning was 11.0D (2.53). The mean vector calculated disease induced astigmatism magnitude was 7.59D (3.09). Graft refractive surgery was performed in seven eyes. The mean cylinder power before and after graft refractive surgery was 11.28D (2.15) and 7.09D (5.53) respectively. The surgically induced astigmatism vector magnitude was 7.36D (4.88).

Conclusion: Progression of keratoconus in the host cornea late after penetrating keratoplasty is characterised by a large astigmatic change where the flat axis of astigmatism passes through an area of host thinning visible on slit lamp examination. Compressive resutting performed in the area of host thinning resulted in satisfactory reduction of astigmatism.

Patients and methods
We analysed retrospectively the clinical features and the surgical outcome of 10 eyes in 10 patients who sought treatment from one surgeon (DJC) for progression of keratoconus in the host cornea after PK. The sources of information were the hospital records and, where applicable, the records of ophthalmologists and optometrists who referred these patients. The data collected included patient demographic information, area of the host cornea where progression of keratoconus occurred as confirmed by slit lamp examination, duration after grafting when host thinning occurred, keratometry, refraction, corneal topography, and corrected visual acuity before and after progression of keratoconus in the host cornea and after graft refractive surgery, type, and location of graft refractive surgery, duration before suture removal, and number of sutures remaining after suture removal.

Graft refractive surgery
Preoperative refraction was difficult to perform in some patients owing to the high astigmatism involved. Although it would be ideal to have preoperative refractions on all these patients, we relied primarily on keratometry in the planning of the surgery. Those who required surgery could not achieve satisfactory vision with contact lenses or spectacles. Compressive suturing was performed in the quadrant of host thinning which corresponded to the area with the flattest meridian on keratometry. This involved making an incision of approximately 90° depth at the graft-host junction spanning 2–3 clock hours, on either side of the flattest meridian. This is followed by compressive suturing of the incision with 3–6 interrupted 10–0 Nylon sutures to achieve an overcorrection, which was verified in the operating theatre with the use of an operative keratometer. Selective suture removal was performed a few months later to give the optimum cylindrical correction.
Vector analysis of astigmatism

Vector analysis was carried out using the Alpins method, with minor modifications to allow for the analysis of disease induced astigmatism. Vector values for disease induced astigmatism, targeted induced astigmatism (TIA), and surgically induced astigmatism (SIA) were derived and analysed using keratometric data.

To analyse the astigmatic change brought about by disease progression in the host cornea, the change in keratometric readings was used to calculate a vector value for this change, the disease induced astigmatism. This may be defined as the degree of corneal flattening induced by the disease. However, since we use the plus cylinder convention and analyse astigmatism using the steepest axis, the figure derived is the steepening of the meridian at right angles to the meridian affected by the disease. Otherwise the vector analysis of disease induced astigmatism is analogous to surgically induced astigmatism. The target of surgery was to reduce the cylinder power to that which can be more easily managed by contact lenses or spectacles, approximately 4D. All procedures were carried out “on axis”; thus, no attempt was made to alter the cylinder axis from that found immediately preoperatively.

RESULTS

There were seven men and three women with a mean age of 41.2 years (range 34–45 years). The average age at undergoing PK in the affected eye was 28.4 years (range 16–44 years). The average time after PK during which keratoconus progressed in the host cornea was 13.5 years (range 10–18 years). These patients presented with a decrease in visual acuity associated with increased astigmatism after having previously had more than 10 years of stable refraction after PK. The duration of symptoms was relatively short with a mean of 10.2 months (range 1–24 months). We were unable to obtain accurate data on graft size; we estimate a mean of 10.2 months (range 1–24 months). We were after PK. The duration of symptoms was relatively short with a mean age at under-

<table>
<thead>
<tr>
<th>Patient</th>
<th>Disease induced astigmatism vector magnitude</th>
<th>Astigmatism after GRS (D)</th>
<th>Surgically induced astigmatism vector and orientation</th>
<th>Targeted induced astigmatism vector magnitude</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8.12</td>
<td>7.59 (2.19)</td>
<td>7.36 (4.88)</td>
<td>5.75</td>
</tr>
<tr>
<td>2</td>
<td>10.38</td>
<td>2.13 (160)</td>
<td>7.75 (180)</td>
<td>8.88</td>
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<tr>
<td>3</td>
<td>12.50</td>
<td>14.13 (38)</td>
<td>14.06 (6)</td>
<td>10.47</td>
</tr>
<tr>
<td>4</td>
<td>12.09</td>
<td>4.13 (20)</td>
<td>10.72 (3)</td>
<td>6.13</td>
</tr>
<tr>
<td>5</td>
<td>8.63 (93)</td>
<td>6.23 (10)</td>
<td>9.54 (157)</td>
<td>5.00</td>
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<tr>
<td>6</td>
<td>6.63 (10)</td>
<td>6.31 (115)</td>
<td>0.72 (33)</td>
<td>5.63</td>
</tr>
<tr>
<td>7</td>
<td>14.06 (108)</td>
<td>3.31 (115)</td>
<td>3.41 (73)</td>
<td>9.16</td>
</tr>
<tr>
<td>8</td>
<td>6.38</td>
<td>14.108</td>
<td>14.06</td>
<td>9.54</td>
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<tr>
<td>9</td>
<td>4.27</td>
<td>6.63 (10)</td>
<td>6.23 (115)</td>
<td>5.63</td>
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<tr>
<td>10</td>
<td>5.17</td>
<td>14.06 (108)</td>
<td>3.31 (115)</td>
<td>9.16</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>5.07 (2.19)</td>
<td>7.59 (2.15)</td>
<td>7.36 (4.88)</td>
<td>5.75 (2.15)</td>
</tr>
</tbody>
</table>

*Mean of seven eyes with keratometric data before and after graft refractive surgery for comparison.

Figure 1 Slit lamp photography showing progression of keratoconus in the host cornea inferiorly resulting in host tissue thinning.
Late recurrence of keratoconus after host thinning, and after graft refractive surgery (GRS).

Figure 2 Box plots illustrating graft astigmatism before host thinning, after host thinning, and after graft refractive surgery (GRS).

Figure 3 Box plots illustrating visual acuity before host thinning, after host thinning, and after graft refractive surgery (GRS).

magnitude, but is off axis by almost 60 degrees leaving a large cylinder. Patient 3 also developed inferior graft fibrosis following compressive suturing, which may have contributed to this off-axis effect. However, on refraction, 6/9 vision was achieved with $-1.75/-4.00 \times 108^\circ$.

Patient 1 developed endothelial graft rejection 1 month after graft refractive surgery but this resolved with topical steroid treatment over 3 months. Figures 4 and 5 are corneal topography maps of patient 2 before and after graft refractive surgery. Figure 4 is a composite map showing the corneal topography before surgery (top left), after surgery but before suture removal (right), and after selective suture removal with two sutures remaining (bottom left). Figure 5 illustrates the effect of wedge excision and compressive resuturing with deliberate initial overcorrection before suture removal.

Seven eyes had keratometric data available before and after refractive surgery for astigmatism. The mean cylinder, preoperatively, was 11.28D (2.15) and postoperatively was 7.09D (5.53). To achieve 4D on-axis of remaining astigmatism, the mean TIA vector magnitude was 7.28D (2.15). Vector analysis of the final keratometric readings demonstrates the SIA vector magnitude was 7.36D (4.88). The mean magnitude of error (that is, the difference between the SIA and TIA) shows a small “overcorrection” of 0.07D (4.3). However, since any astigmatism less than 4D was deemed acceptable, this is only an overcorrection in terms of vector analysis where, to calculate how well the surgery achieved its aim, the target has to be given a specific value. To obtain a residual cylinder value of zero would still be desirable although difficult to achieve.

The angle of error is the difference between the SIA vector angle and the TIA vector angle. It is a measure of the accuracy of the meridional placement of the surgery and its predictability with regard to axis. The mean was $-7.34^\circ$ (64.05*) (clockwise).

DISCUSSION

Astigmatism after corneal transplantation is common. A subgroup of patients develops excessive astigmatism years after successful surgery. This group of patients has progression of ectasia in the recipient cornea, which is usually eccentric and creates attenuation of the wound in the affected area. This has a flattening effect on the donor cornea as the thinned host cornea weakens in its support for the donor cornea at the graft-host junction. This induced a steepening effect in the opposite meridian of the donor cornea with an average vector value of 7.59D in our series. The schematic diagram in figure 6 illustrates inferior host thinning with resultant weakening of the graft-host junction.

It is important to recognise patients in which this mechanism is present. Conventional approaches to post-keratoplasty astigmatism are unlikely to be effective. Incisional surgery in the steeper axis or LASIK (laser in situ keratomileusis) would be weakening procedures in an already ectatic eye. A “strengthening” procedure at the site of the problem, incision and suturing, or repeat keratoplasty would be more appropriate. Therefore, clinicians need to be aware of the pattern of disease resulting from progressive ectasia and be able to offer a rational corrective procedure based on an understanding of the pathophysiology.

These patients presented with decreasing visual acuity over a relatively short period of time (mean 10.2 months). This was coincident with increasing refractive and keratometric astigmatism. An area of host thinning was evident on slit lamp examination (fig 1) in the area of host cornea with the flat meridian on keratometry. The mean disease induced astigmatism vector value was 7.59D. The direction of axis change did not show any recognisable trend. By giving the direction a sign based on the direction of the change and assuming random distribution of direction and magnitude of change, one would expect the derivation of a mean axis change to yield a value of zero. Here the value is 21* in the anticlockwise direction but the numbers of eyes are small and the standard deviation is large (55.98).

Table 2 Improvement of visual acuity (VA) after graft refractive surgery

<table>
<thead>
<tr>
<th>Case</th>
<th>VA before GRS</th>
<th>VA after GRS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6/12 (glasses)</td>
<td>6/9 (glasses)</td>
</tr>
<tr>
<td>2</td>
<td>6/18 (glasses)</td>
<td>6/9 (glasses)</td>
</tr>
<tr>
<td>3</td>
<td>CF (unaided)*</td>
<td>6/9 (glasses)</td>
</tr>
<tr>
<td>4</td>
<td>6/60 (unaided)*</td>
<td>6/12 (glasses)</td>
</tr>
<tr>
<td>5</td>
<td>6/24 (glasses)</td>
<td>6/12 (glasses)</td>
</tr>
<tr>
<td>6</td>
<td>6/36 (unaided)*</td>
<td>6/9 (glasses)</td>
</tr>
<tr>
<td>7</td>
<td>CF (unaided)*</td>
<td>6/9 (glasses)</td>
</tr>
</tbody>
</table>

GRS = graft refractive surgery; VA before GRS = acuity obtained with spectacle refraction as contact lens fitting was difficult because of the high cylinder.

*Refraction was difficult to perform because of the high cylinder.
These patients have an average of 11.00D of astigmatism, which makes correction with glasses or contact lenses difficult. Various surgical methods of correcting post-keratoplasty astigmatism (all sutures out astigmatism) have been described. These include relaxing incisions, compressive resuturing, relaxing incisions with counterquadrant augmenting compressive sutures (known as augmented relaxing incisions), wedge resections, trapezoidal keratectomy, and excimer laser refractive surgery. Trapezoidal keratectomies are technically difficult and the results are unpredictable. In our series of patients with high astigmatism resulting from host thinning we believe that the logical and best technique to employ would be compressive resuturing over the area of host thinning, that is, over the graft-host junction with the flat meridian. Although relaxing incisions along the steep meridian alone are appropriate for many cases of post-keratoplasty astigmatism, they will not improve the strength of the graft-host junction in the flat meridian, which is the cause of the astigmatism in host thinning (fig 6). It follows that it is important in cases of increased astigmatism in an old graft to examine the host cornea adjacent to the junction for any sign of thinning. For seven eyes, compressive resuturing was performed in the area with host thinning. The schematic diagram in figure 6 illustrates

![Figure 4](http://bjo.bmj.com/)

**Figure 4.** Corneal topography maps. Upper left: recurrence of keratoconus nasally and temporally resulting in a flat horizontal meridian. Right: wedge excision and compressive resuturing nasally and temporally resulting in overcorrection before suture removal. Lower left: reduction of astigmatism after suture removal.

![Figure 5](http://bjo.bmj.com/)

**Figure 5.** Corneal topography maps. Upper left: recurrence of keratoconus nasally and temporally resulting in a flat horizontal meridian. Right: wedge excision and compressive resuturing nasally and temporally resulting in overcorrection before suture removal. Lower left: difference map showing the surgical correction of astigmatism.
the theory behind our technique. If the host thinning results in more than 10D of astigmatism, wedge excision could be used in addition to compressive resuturing for more effect (patient 2).

This technique has resulted in the postoperative mean astigmatism of 7.09D, which is a significant improvement from the preoperative mean astigmatism of 11.28D with a mean reduction of 4.2D of astigmatism by cylinder subtraction. This is comparable to previous reported studies on graft refractive surgery which reported a mean reduction in cylinder of 3.6–5D and 5.5D (for the group with preoperative astigmatism between 4–10D).15 16 Although there appears to be little difference in the keratometric astigmatism before and after graft refractive surgery for patients 3, 5, and 7, these patients were able to tolerate corrective glasses after surgery. Perhaps the treatment of host thinning with compressive resuturing corrected some irregular astigmatism with resultant better spectacle tolerance.

The mean surgically induced astigmatism was just over 7D. It is notable that this is close to the mean disease induced vector value for the seven eyes undergoing graft refractive surgery (8.42D (3.32)). Taking a target value of 4D of remaining astigmatism, this was achieved, in vector terms, with a mean magnitude of error (SIA – TIA) of only 0.07D, with the SIA exceeding the TIA very slightly. Although there appears to be little difference in the keratometric astigmatism before and after graft refractive surgery for patients 3, 5, and 7, these patients were able to tolerate corrective glasses after surgery. Perhaps the treatment of host thinning with compressive resuturing corrected some irregular astigmatism with resultant better spectacle tolerance.

In conclusion, patients presenting with increased astigmatism after PK should be examined for signs of host thinning in the flat meridian. Such cases can be satisfactorily managed.
with incision and compressive resuturing in the graft-host junction and, where astigmatism is high, additional wedge excisions.

ACKNOWLEDGEMENTS

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Authors’ affiliations

L Lim, K Pesudovs, M Goggin, D J Coster, Department of Ophthalmology, Flinders Medical Centre, Flinders University of South Australia, Bedford Park 5042, South Australia

REFERENCES

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