Effects of lamellar keratotomy on postkeratoplasty astigmatism

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Aim: To determine the changes in postkeratoplasty astigmatism induced by lamellar keratotomy.

Methods: A prospective, non-randomised comparative trial of patients undergoing a hinged lamellar corneal flap treatment for significant astigmatism after penetrating keratoplasty. Uncorrected visual acuity, best corrected visual acuity, refraction, and corneal topography were assessed at 1 and 3 months after the lamellar keratotomy.

Results: 17 eyes in 16 patients (13 M, 3 F) were included in the study (mean age 48.2 years; range 20–86 years). Six of 17 eyes (35.3%) changed more than 1 dioptre (D) in spherical equivalent by 3 months. Nine of 17 eyes (52.9%) changed more than 1 D in sphere by 3 months. 12 of 17 eyes (70.6%) changed more than 1 D in refractive cylinder. Seven patients of 15 (46.7%) changed more than 1 D in corneal power as measured topographically. 5 of 17 eyes (29.4%) changed in refractive cylinder axis more than 15 degrees and this was similar to the change measured topographically of 15 eyes (26.7%). Vector analysis showed 60% of eyes had a surgically induced astigmatism (SIA) vector of more than 1 D, including a net corneal astigmatism decrease of more than 1 D in four eyes and increase of more than 1 D in two eyes at 3 months after surgery. Complications of the lamellar keratotomy included two partial buttonholes and one partial wound dehiscence.

Conclusions: The creation of a lamellar flap alone can have significant effects on the astigmatism following penetrating keratoplasty. LASIK for correction of postkeratoplasty astigmatism may be more accurately performed as a two stage procedure rather than a single stage, after the corneal effects of the lamellar keratotomy have stabilised.
examination, corneal topography (Alcon Eye Map EH-290), Software Version 5.00, Fort Worth, TX, USA), and indirect ophthalmoscopy. Patients at the end of 3 months were assessed as to whether to proceed with second stage ablation according to their visual outcomes.

Statistical analyses were performed with the SPSS program (SPSS Inc, Chicago, IL, USA). Group differences for continuous variables were tested using the paired Student’s t test for normally distributed data and the Wilcoxon signed ranks test for non-normally distributed data. Differences were considered statistically significant when the p value was lower than 0.05.

Alpins vector analysis was used to evaluate the corneal astigmatic changes induced by surgery. The surgically induced astigmatism (SIA) vector is the amount and direction of corneal steepening that occurs in achieving the postoperative astigmatism from the preoperative astigmatic state. The SIA vector is the astigmatic change induced by any surgical procedure that alters the shape of the cornea (in our case, the cut by the microkeratome). If the SIA vector is zero, there is no change in the corneal astigmatism.

RESULTS

Seventeen eyes in 16 patients (13 men, three women) were included in the study (Table 1). The mean age of the patients at the time of the creation of the lamellar flap was 48.2 years (range 20–86 years). The mean interval between PK and the time of suture removal was 60.0 months (range 9–276, SD 67.1). Six eyes had undergone corneal relaxing incisions for high astigmatism after PK, at least 6 months before the lamellar keratotomy. The indication for PK was keratoconus in 11 eyes, Fuchs’ endothelial dystrophy in five eyes and herpes simplex keratitis in one eye.

Visual results

The mean uncorrected visual acuity pre-operatively was 0.14 (20/145) (range 0.02–0.5, SD 0.14) (Table 1). At 3 months postoperatively, the mean uncorrected visual acuity was 0.16 (20/125) (range 0.02–0.5, SD 0.14). The difference between preoperative and 3 month postoperative values was not statistically significant (p=0.55). Two eyes (7 and 15) had worse uncorrected visual acuity, one the result of a partial wound dehiscence and resulting increase in astigmatism (eye 15). The mean best corrected visual acuity preoperatively was 0.8 (20/25) (range 0.25–1.20, SD 0.27). At 3 months, best corrected visual acuity was 0.76 (20/26) (range 0.5–1.20, SD 0.23). No significant difference was found between preoperative and 3 month postoperative best corrected visual acuity values (p=0.82). Three eyes had improved best corrected visual acuity two lines or more, two eyes worsened two lines or more, and 12 eyes were within one line.

Refractive results

The mean preoperative spherical equivalent, sphere, and cylinder were, respectively, −5.28 D (range −16.25 to +1.50, SD 4.79), −1.82 D (range −13.00 to +5.50, SD 4.87), and −6.79 D (range −11.00 to −3.00, SD 2.15) (Table 2). At 3 months postoperatively, the spherical equivalent, sphere, and cylinder were respectively −4.79 D (range −16.00 to +1.25, SD 5.10), −1.74 D (range −11.50 to +5.00, SD 5.15), and −6.11 D (range −17.00 to −0.05, SD 3.78). Six of 17 eyes (35.3%) changed more than 1 D in spherical equivalent by 3 months. Nine of 17 eyes (52.9%) changed more than 1 D in sphere by 3 months. Twelve of 17 eyes (70.6%) changed more than 1 D in refractive cylinder. The refractive cylinder axis changed postoperatively by more than 15 degrees in five of 17 eyes (29.4%). This was the only change in refractive parameter that reached statistical significance (p=0.02).

Topographic changes

The mean preoperative topographic corneal power and keratometric astigmatism were respectively 47.26 D (range 42.16–51.00, SD 2.9) and 6.02 D (range 1.48–9.66, SD 2.10). At 3 months postoperatively, the mean topographic corneal power and keratometric astigmatism were respectively 47.03 D (range 42.16–51.00, SD 2.59) and 6.01 D (range 0.8–12.84, SD 2.10) (Table 2). The postoperative topographic data were missing in two eyes. The differences between preoperative and 3 month postoperative data for topographic corneal power and keratometric astigmatism were not significant (p=0.74 for corneal power; p=0.62 for keratometric astigmatism). Seven of 15 eyes (46.7%) changed more than 1 D in corneal power as measured topographically and four of 15 eyes (26.7%) changed more than 1 D in keratometric astigmatism. Four of 15 eyes (26.7%) changed in keratometric cylinder axis more...
than 15 degrees and this was similar to the change measured by refraction (5/17, 29.4%). There was a significant correlation between the change in corneal power and the change in spherical equivalent (Pearson correlation coefficient \( r = -0.55 \), \( r^2 = 0.30 \), \( p = 0.03 \)), in such a way that 30% of the 30% change in refractive spherical equivalent can be explained by the change in keratometric corneal power.

### Vector analysis results

The mean surgically induced astigmatism (SIA) by vector analysis was 1.82 D (range 0.01–5.01, SD 1.45) and the mean SIA axis was 47.3° (range 22–110°, SD 42.4°). Nine of 15 eyes (60.0%) had a SIA of more than 1 D at 3 months after surgery. Of these eyes, four had a decrease and two had an increase of the preoperative keratometric astigmatism. In three eyes there was a change of preoperative astigmatism as a result of a torque force (SIA at 45° in relation to the preoperative astigmatism axis).

### Complications

Complications of the lamellar keratotomy included two partial buttonholes (eyes 8 and 17) and one partial wound dehiscence (eye 15). The partial buttonholes although centrally located, healed without visual compromise. The partial wound dehiscence resulted in a 6 D increase from –11.0 D to –17.0 D in the keratometric astigmatism after 3 months. There were no episodes of epithelial ingrowth or graft rejections.

### DISCUSSION

Significant anisometropia with or without high degree astigmatism resulting from PK surgery is relatively common. To date, various surgical techniques have been used to correct postkeratoplasty anisometropia and high astigmatism, with a variable rate of success. Both relaxing incisions and wedge resections can significantly reduce postkeratoplasty astigmatism, but their effect on spherical refractive errors is minimal. More recently, both PRK and LASIK have proved to significantly reduce postkeratoplasty refractive errors; however, LASIK has become preferred over PRK owing to the higher rate of complications with PRK, including haze and loss of best corrected visual acuity. LASIK has been effective in reducing postkeratoplasty refractive errors and its safety has been documented in previous studies, although there are limitations. Owing to the high vacuum applied during the microkeratome cut, the wound must be healed adequately in order to avoid wound dehiscence. The lamellar cut is different from the normal cornea as postkeratoplasty corneas have steep keratomy and are at risk of buttonholes and thin flaps. The results of LASIK performed on postkeratoplasty eyes have shown good results in correcting the spherical component of the refraction, although the predictability in correcting postkeratoplasty astigmatism has been poor.

A possible explanation for the poor predictability of LASIK on high astigmatism postkeratoplasty eyes may be found in the biomechanics of the cornea. Robert has shown that simply performing a hinged lamellar keratotomy induces a biomechanical response in the cornea, resulting in substantial changes in its shape. On corneas with previous PK, where the natural biomechanical state is altered, this effect could be greater. The graft-host wound has different stress forces and biodynamics compared with the normal limbus. Postkeratoplasty astigmatism may be caused by a combination of size differences between the donor and host, as well as irregular scarring at the graft-host junction, creating asymmetric contractile forces that deform the cornea. The cutting of a lamellar corneal flap could influence these contractile forces resulting in a change of the existing astigmatism. Another surgical variable that may also influence astigmatism outcome is the effect of the position of the flap hinge and the axis of astigmatism. All hinges in this study were created in the superior position; however, with the use of later generation microkeratomies that are able fashion the hinge in varying positions, this variable will be able to be investigated further.

A study by Dada et al reported, in a young patient, a 5 D reduction in postpenetrating keratoplasty astigmatism occurring at the creation of a lamellar corneal flap. Another study by Busin et al specifically addressed the effect of lamellar keratotomy on postkeratoplasty astigmatism. They reported nine eyes in nine patients and found on average the spherical equivalent to flatten by 1 D and the refractive cylinder to reduce by 1.5 D. No topographic or vector analysis were performed. Although these studies are mainly based on refractive data, they show a change in astigmatism after the creation of a lamellar corneal flap alone. Therefore, it is unlikely for lamellar keratotomy alone to fully correct the astigmatism; however, it does alter the degree of laser ablation at the second stage.
Manifest refraction is a subjective measurement. It may be difficult to obtain an accurate preoperative manifest refraction especially in postkeratoplasty patients with severe refractive errors. The use of corneal topography (simulated keratometry values) provides an objective analysis of the corneal astigmatism and may reflect more accurately the change in corneal shape, especially if processed by vector analysis (Alpins), which takes into account power and axis. Most reports on LASIK procedures for the correction of high astigmatism after PK have been performed as a single stage surgery—that is, creating a lamellar keratotomy and immediately afterwards applying laser ablation to the corneal stromal bed. Based on our results, the actual cylinder and/or the axis may differ from the existing astigmatism in more than half of the patients after making the corneal flap with a microkeratome. It is likely that using the preflap data will result in less precise treatment parameters and thereby limit the success of postkeratoplasty astigmatism correction with LASIK when performed as a single treatment procedure. In this study, the induced changes at 1 month compared with 3 months were not statistically different; however, in individual cases there may be wider variation. This suggests the second stage LASIK may be performed as soon as 1 month, but in cases where there has been a significant change at 1 month, 3 months may be a better time for the second stage. It was not possible in this study to conclude whether larger cylinder astigmatism requires a longer period for the flap to stabilise. The worsening of the astigmatism as occurred in one patient could be indicative of a subclinical area of partial wound dehiscence. The graft-host interface must be carefully examined by slit lamp microscopy for evidence of healing before automated lamellar keratotomy, regardless of the duration postpenetrating keratoplasty.

In conclusion, the creation of a lamellar corneal flap with a microkeratome modifies the existing postkeratoplasty astigmatism. The mechanisms for the microkeratome induced change in astigmatism are relief of the irregular forces from the graft-host interface and altered replacement of the lamellar corneal flap into the stromal bed. The results for LASIK correction of postkeratoplasty astigmatism may be improved by performing a two stage procedure, allowing the lamellar flap to stabilise and increase the reliability of refraction and topography. Further studies are indicated to determine the optimum period required between the two stages and whether the long term visual outcomes are improved by a two stage procedure or whether the risks of a flap lift in this group of patients outweighs the benefits.

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