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CORNEA

Intraocular pressure elevation and post-DSEK glaucoma after Descemet`s stripping endothelial keratoplasty

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Abstract
Background Intraocular pressure (IOP) elevation is a common problem in penetrating keratoplasty (PK), and possibly leads to graft failure. IOP elevation and secondary glaucoma may also be present after Descemet`s stripping endothelial keratoplasty (DSEK). This retrospective study analyzes the risk factors for IOP elevation and the functional outcome in those patients with post-DSEK glaucoma.
Methods A retrospective analysis of case records of 72 DSEKs between 2007 and 2010 was performed. A total of 59 operated eyes were included. The assessment included the pre-operative history of corneal disease and glaucoma. Furthermore, the response to antiglaucoma treatment, the graft failure, the IOP, and visual acuity development were evaluated.
Results The incidence of IOP elevation was 28.8 % and of post-DSEK glaucoma 11.9 %. Steroid-induced IOP elevation was the most frequent cause, with an incidence of 18.6 %. Patients with pre-existing glaucoma showed a significantly higher risk of developing IOP elevation, steroid-induced glaucoma and post-DSEK glaucoma ($p=0.006$, $p=0.023$, $p=0.009$). In all cases, IOP elevation was treated effectively by tapering down steroid medication and initiating or increasing antiglaucoma medication. Visual acuity after 6 and 12 months improved significantly in cases with and without pre-existing glaucoma ($p<0.0001$). After 24 months, clear grafts were achieved in 53 eyes (89.9 %). There was no significant difference in graft failure rates between cases with or without pre-existing glaucoma ($p=0.581$) and with or without post-DSEK glaucoma ($p=0.306$).
Conclusions IOP elevation after DSEK shows a high incidence. Pre-existing glaucoma increased the risk of developing IOP elevation and post-DSEK glaucoma. Although steroid-induced IOP elevation was the most frequent cause and could be treated effectively by tapering down steroid medication; there are other reasons why post-DSEK glaucoma developed. Management by medical treatment results in good visual acuity and graft survival.

Keywords IOP elevation · Post-keratoplasty glaucoma · DSEK · Antiglaucoma therapy

The authors have full control of all primary data, and agree to allow Graefe's Archive for Clinical and Experimental Ophthalmology to review data upon request.

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Introduction

Descemet`s stripping endothelial keratoplasty (DSEK) has been performed with increasing frequency for cases of endothelial dysfunction in recent years [1]. It is a less invasive corneal transplant technique that selectively replaces a damaged corneal endothelium and Descemet`s membrane [2]. In comparison to penetrating keratoplasty (PK), the advantages include rapid healing, more predictable refractive outcomes, better corneal integrity, and a rapid visual recovery [3–6]. Improvements in surgical technique have resulted in better visual outcome and fewer postoperative complications [4, 6–8].

Glaucoma following PK is a frequent problem, with a high incidence and prevalence. The reported incidence of glaucoma after PK ranged from 9 % to 31 % in the early postoperative period [9–12], and from 18 % to 35 % in the late postoperative period [13–15]. Glaucoma monitoring, its refractory treatment, and its devastating outcome for the

patient with irreversible visual loss [13, 15] are challenging [9, 12]. In addition, pre-existing glaucoma and uncontrolled rises in intraocular pressure (IOP) have been described as major risk factors for poor visual outcome, endothelial cell loss, and subsequent graft failure after PK. Other causes of post PK elevated IOP include response to steroids, use of viscoelastics, damage to outflow mechanisms, loss of angle support, and angle closure due to synechiae [10, 14–16].

In contrast to PK, there are fewer reports of IOP elevation and glaucoma after DSEK [2, 3]. First reports hypothesized that DSEK induces less IOP changes compared to PK [17]. Nevertheless, the factors that generate high IOP after PK may also apply to DSEK. Of these, Vajaranant et al. identified steroid-related ocular IOP elevation as the major cause [3].

To analyse the potential reasons for the development of elevated intraocular pressure in a selected cohort of DSEK patients, we retrospectively evaluated IOP rise, associated risk factors, surgical techniques, visual outcome, and the treatment in patients with glaucoma.

Materials and methods

Patients

A retrospective analysis of case records of 72 DSEKs between January 2007 and January 2010 was performed. All DSEKs were performed at the Department of Ophthalmology, Charité–Universitätsmedizin Berlin, Campus Virchow Klinikum by one experienced surgeon in cases of Fuchs' corneal dystrophy or bullous keratopathy (Table 1). For this study, only DSEK cases with at least 3 months of follow-up were included. This retrospective study follows the ethical standards of the Helsinki Declaration.

Definition of postoperative elevated intraocular pressure and pre-existing glaucoma

Post-DSEK elevated IOP was defined as IOP ≥22 mmHg or an increase in IOP from preoperative value ≥10 mmHg at any postoperative examination. At any visit, single IOP measurements that met this criteria would be classified as postoperative IOP elevation. All eyes with postoperative elevated IOP were categorised according to whether the rise correlated with steroid-induced glaucoma, post-DSEK glaucoma, or/and postoperative pupillary block IOP elevation:

- Postoperative pupillary block IOP elevation was defined as IOP elevation in the first 2 days after DSEK.
- Steroid-induced glaucoma was defined as eyes in which the IOP normalized (≤21 mmHg) when the steroid treatment ended.
- Post-DSEK glaucoma, iatrogenic-induced secondary glaucoma, was defined as a lasting elevated IOP (≥22 mmHg) at different time points which required anti-glaucoma medication or surgical intervention [14, 18, 19]. In patients with pre-existing glaucoma, worsening of the IOP control requiring additional medication or surgery was used to diagnose post-DSEK glaucoma [14, 18]. This was independent of associated visual field loss and optic nerve head changes.

The definition of pre-existing glaucoma included any of the following: a documented history of glaucoma, prior glaucoma filtration surgery, preoperative use of antiglaucomatous medications, typical glaucomatous excavation of the optic disc, or a cup/disc ratio of ≥0.6. For cases with suboptimal view of the fundus at preoperative evaluation, the available fundus examination or C/D on the subsequent examination was used.

Table 1 Data of the demographic, surgical and preoperative results

	Without pre-existing glaucoma (A): n=44	With pre-existing glaucoma (B): n=15	Total: n=59
Female:male	28:16	6:9	34:25
Age (years, mean ± SD)	72.1 ± 7.5	72.4 ± 7.4	73.3 ± 7.0
Reason for DSEK			
- % Fuchs` corneal dystrophy	93.1 % (n=41)	86.8 % (n=13)	91.5 % (n=54)
- % Bullous keratopathy	6.8 % (n=3)	13.3 % (n=2)	8.5 % (n=5)
Surgery			
DSEK	45.5 % (n=20)	80.0 % (n=12)	54.2 % (n=32)
Triple DSEK	40.9 % (n=18)	13.3 % (n=2)	33.9 % (n=20)
Re-DSEK	13.6 % (n=6)	6.7 % (n=1)	11.9 % (n=7)
Preoperative VA in LogMAR: median, (range minimum–maximum)	6.0 (2.0–10.0)	4.0 (2.0–7.0)	6.0 (2.0–10.0)
Preoperative IOP in mmHg: mean ± SD	13.08 ± 2.98	14.5 ± 3.09	13.41 ± 3.03
Cup-to-disc ratio: median (range minimum–maximum)	0.2 (0.2–0.4)	0.5 (0.2–0.8)	0.2 (0.2–0.8)

Preoperative and postoperative evaluation

Postoperative examinations were performed after 2 and 6 weeks, and then after 3, 6, 12, and 24 months after DSEK. They included visual acuity, slit-lamp examination, applanation tonometry, and funduscopy.

Distant visual acuity was tested with a Snellen chart, and expressed as a Snellen decimal number. The Snellen decimal number was converted in logMAR by a Visual Acuity Conversion Table [20]. Because refraction was not performed routinely, preoperative and postoperative visual acuity at each visit was analyzed as best-corrected visual acuity with or without refraction or pinhole visual acuity. IOP was measured by Goldmann applanation tonometer (Haag–Streit, Bern, Switzerland). The readings were usually single measurements. Corneal thickness was not considered. In addition, in some cases (<1 %), other tonometers included pneumatic tonometer (CT20D computerized Tonometer, Topcon, Japan) and Perkin applanation tonometer MK2 (Clement Clarke International, Harlow, Essex, UK).

Fundus examination in particular of the optic disc was performed. Where the view of the optic nerve was adequate, the cup-to-disc ratio (C/D) was also documented in the majority of cases.

Age, gender, pre-DSEK diagnosis, prior history of glaucoma, and the post-DSEK glaucoma treatment were documented.

Graft and surgical techniques

The DSEK surgical technique was performed in a standardized manner described in detail by Price and Price [4, 7, 8, 21]. In all cases clear corneal incisions were used, with an incision size of 3.2 mm. In some cases, a combined procedure (Triple DSEK) with DSEK following standard cataract surgery was performed.

All patients received organ cultured grafts from the Cornea Bank Berlin. For transplantation, grafts had a minimum central endothelial density of 2000/mm^2. The donor graft was dissected using either the Moria ALTK system or the Schwind Carriazo-Pendular microkeratome. Over the study period and with increasing experience, the donor lamella depth was reduced from approximately 200 μm to 100 μm. The maximum diameter of the graft was 8.5 mm.

The standard postoperative treatment included daily application of a topical steroid (three to five times daily) and lubricant eye drops (five times daily) and a combined antibiotic and steroid ointment at night. Postoperatively, pilocarpine eye drops (1 %) were given until the air bubble was absorbed (approx 1 week). Thereafter, the ointment was stopped, and prednisolone acetate 1 % was used topically (3 times daily for the first 3 months) with lubricant eye drops five times daily. The prednisolone acetate 1 % was tapered down over a period of 2–3 months to once or twice daily, and patients remained on this dosage until 1 year postoperatively unless they developed steroid-induced glaucoma.

Treatment of elevated IOP

In patients who developed IOP elevation, the prednisolone acetate 1 % was tapered down. If transient IOP elevation remained after tapering down the steroid eye drops, topical glaucoma medication was used. This was initiated with topical timolol maleate 0.5 % twice daily. If further treatment was necessary, topical carbonic anhydrase inhibitors, prostaglandin derivatives, alpha-2 selective adrenergic agonist, or pilocarpine could be added.

In cases of IOP elevation during the first 2 postoperative days because of pupillary block glaucoma, air bubble removal via a paracentesis and pilocarpine instillation was performed to reduce the IOP.

Statistical methods

The statistical analysis was performed using SPSS Windows (SPSS Software, Munich, Germany). Normality was tested for all outcome measures. None of the measures followed a normal distribution. Therefore, nonparametric tests (Kruskal–Wallis, Wilcoxon signed-rank test) were used for analysis. Descriptive statistics were expressed as median and range between minimum and maximum or mean ± 2 standard deviations (SD). Clear graft survival and timing of postoperative IOP elevation were plotted using the Kaplan–Meier survival analysis. Log-rank test was used to evaluate statistical significance in the groups. To analyze the distribution of IOP elevation, steroid-induced glaucoma and post-DSEK glaucoma, the chi square distribution was used. Differences were considered statistically significant when P values were less than 0.05.

Results

Seventy-two DSEK procedures were undertaken during the study period (2007–2010). Of these, 59 eyes fitted the study criteria. Forty-four eyes did not have pre-existing glaucoma (group A). Fifteen were categorised as having pre-existing glaucoma before DSEK (group B). The mean follow up time was 437 ± 235 days (range 131–1065 days).

Patients with pre-existing glaucoma were treated preoperatively with topical antiglaucomatous medication; none had had previous glaucoma surgery. Only one eye was

treated with an Argon Laser Trabeculoplasty (ALT) prior to DSEK.

Table 1 summarize the data of the demographic, surgical, and preoperative results for both groups.

IOP elevation

For the with and without pre-existing glaucoma groups, the percentage of cases with risk factors that may lead to post-DSEK IOP elevation have been shown in Table 2. Data presented in Fig. 1 show that a greater percentage of patients with pre-existing glaucoma developed elevated IOP than those without, and remained significantly over a 12 month period (Table 3).

There was no significant difference in the mean post-operative IOP between cases developing a post-DSEK glaucoma and those that did not, expected after 1 year (p=0.004) (Fig. 2).

Therapy

All patients with IOP elevation were treated medically to control the IOP. In 11 eyes with steroid-induced glaucoma, tapering down local steroids was sufficient to normalize IOP. To bridge IOP elevation during tapering, glaucoma medication was given. In three of 11 eyes of steroid-induced glaucoma, IOP rose again after tapering down the steroid and normalising the IOP, and developed a post-DSEK glaucoma.

In five of seven eyes with post-DSEK glaucoma, two different drugs were necessary to control the IOP. In the other two eyes, additional medication was required. No eyes required surgical intervention.

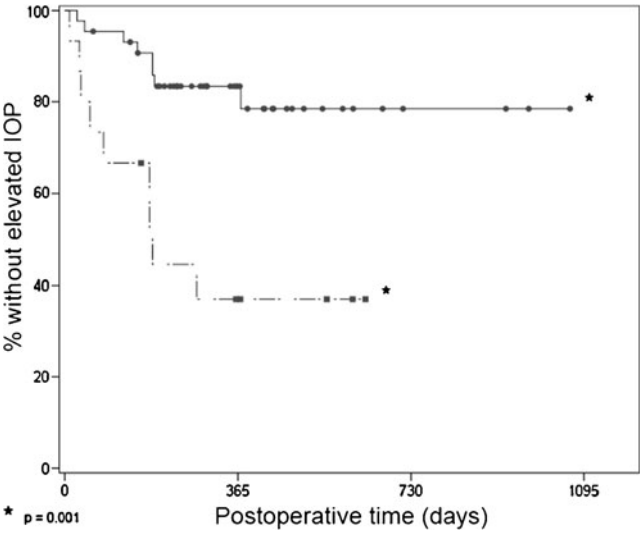

Fig. 1 Kaplan–Meier survival plot shows the percentage of eyes without elevated IOP after DSEK with and without pre-existing glaucoma. Endpoint is elevation of IOP excluding cases which developed a pupillary block glaucoma after DSEK in the first 2 days after surgery. After 1 year, 83.4 % of grafts without pre-existing glaucoma demonstrated no elevation of IOP (*continuous line*), and 37.0 % of grafts with pre-existing glaucoma (*dotted line*) (p=0.001)

Outcome

Visual acuity

Visual acuity data for the eyes with and without pre-existing glaucoma are presented in Table 4. Wilcoxon signed-rank test indicated a statistically significant improvement in visual acuity at 6 and 12 months when compared with preoperative visual acuity (p<0.0001). There was no significant difference in the visual acuity between eyes with or without

Table 2 Incidence of postoperative intraocular pressure changes

	Without pre-existing glaucoma (A): n=44	With pre-existing glaucoma (B): n=15	Total: n=59	P values
IOP elevation after DSEK (study criteria ≥22 mmHg or ≥10 mmHg from preoperative IOP)	31.8 % (n=14)	60.0 % (n=9)	39.0 % (n=23)	
- without cases with postoperative pupillary block IOP elevation	13.6 % (n=8)	60.0 % (n=9)	28.8 % (n=17)	p=0.006 (df1, χ^2=9.54)
- postoperative pupillary block IOP elevation (first 2 days after DSEK)	13.6 % (n=6)	6.7 % (n=1)	11.9 % (n=7)	
- steroid-induced glaucoma	11.4 % (n=5)	40.0 % (n=6)	18.6 % (n=11)	p=0.023 (df1, χ^2=6.05)
- post-DSEK glaucoma	4.5 % (n=2)	33.3 % (n=5)	11.9 % (n=7)	p=0.009 (df1, χ^2 =8.87)
IOP elevation: (IOP elevation without postoperative pupillary glaucoma)				
≥30 mmHg	6.8 % (n=3)	20 % (n=3)	10.2 % (n=6)	
≥24 mmHg	4.5 % (n=2)	33.3 % (n=5)	11.9 % (n= 7)	
≥22 mmHg	6.8 % (n=3)	6.7 % (n=1)	6.8 % (n=4)	

Table 3 Development of intraocular pressure changes

IOP in mmHg: mean ± 2 SD	Without pre-existing glaucoma (A): n=44	With pre-existing glaucoma (B): n=15	P values
Preoperative	13.08 ± 5.96	14.5 ± 6.18	p=0.148
After 2 weeks	12.43 ± 5.80	16.42 ± 10.94	p=0.022
After 4 weeks	14.19 ± 7.28	17.25 ± 12.68	p=0.124
After 3 months	14.14 ± 8.76	17.54 ± 9.92	p=0.014
After 6 months	14.03 ± 8.66	18.67 ± 11.60	p=0.007
After 12 months	13.85 ± 8.18	19.5 ± 15.50	p=0.007
6 months compared with preoperative IOP	p=0.066		
12 months compared with preoperative IOP	p=0.043		

pre-existing glaucoma, and between eyes with or without post-DSEK glaucoma (Fig. 3).

Graft status

After 24 months, clear grafts were achieved in 53 eyes (89.9 %); only in six eyes did graft failure occur. Of these, one had a repeat DSEK, and the other five penetrating keratoplasty. In one case of graft failure, the reason given was a questionable graft rejection (1.7 %), in all other cases loss of endothelial cells. There was no significant difference with regard to graft failure between eyes with or without pre-existing glaucoma (Fig. 4, p=0.581) and with or without post-DSEK glaucoma (p=0.306).

One patient developed a retinal detachment after 2 years, and was operated successfully.

Discussion

To date, there exists one report concerned with the incidence of elevated IOP after DSEK [3]. In our study, we analyzed not only the incidence of elevated IOP after DSEK but also the incidence of post-DSEK glaucoma. Vajaranant et al. reported that the steroid response is the most likely major cause of postoperative IOP elevation [3]. We investigated this assumption, and analyzed the rate of steroid-induced IOP elevation in patients who underwent DSEK surgery between 2007 and 2010 in the Department of Ophthalmology, Charité–Universitätsmedizin Berlin, Campus Virchow Klinikum .

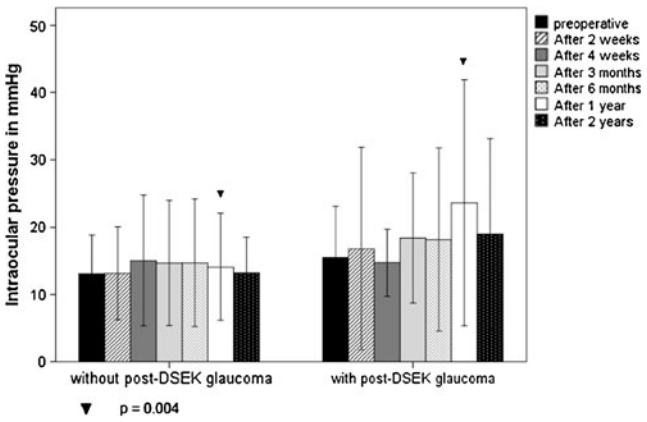

Fig. 2 *Box plot* showing the changes in IOP following DSEK from a preoperative mean value and at postoperative 2 weeks, 6 weeks, 3, 6, 12, and 24 months in the group without post-DSEK glaucoma and with post-DSEK glaucoma

Incidence

Elevated IOP and glaucoma are frequent problems after penetrating keratoplasty (PK), with an incidence of IOP elevation between 9 % and 35 % [3, 9–15, 22–25]. In agreement with the findings of Espana et al. and Vajaranant et al., who reported a substantial incidence of IOP elevation of 35 % after DSEK during the first postoperative year, we also found a high percentage of IOP elevation after DSEK, with a total incidence of 39 % (excluding cases with postoperative pupillary block IOP elevation: 28.8 %) [2] [3]. This incidence of IOP elevation is also comparable to the reported results after PK (9 %–35 %) [3, 9–15, 22–25]

The significant higher incidence of IOP elevation of 60 % in patients with pre-existing glaucoma supports data of Espana et al. and Vajaranant et al. (43–45 %) and results of studies after penetrating keratoplasty (29–80 %) (p=0.006) [2, 3, 9, 18, 22–27]. Additionally, patients with pre-existing glaucoma demonstrated a higher mean IOP, and developed significantly more often a post-DSEK glaucoma (p=0.009). The progression of a pre-existing glaucoma probably plays a more important role in the development of a post-DSEK glaucoma than the iatrogenic-induced worsening of the glaucoma; however, making a clear distinction is not possible.

Reasons for IOP elevation

The response to steroids, use of viscoelastics, damage to outflow mechanisms, loss of angle support, and angle closure due to synechiae [10, 14, 16] may all generate a high post-PK IOP.

The risk of developing a postoperative pupillary block glaucoma is also increased because of the need of an air

Table 4 Development of visual acuity

Visual acuity in logMAR: median (range minimum–maximum)	Without pre-existing glaucoma (A): n=44	With pre-existing glaucoma (B): n=15	P values
Preoperative	6.0 (2.0 – 10.0)	4.0 (2.0 – 7.0)	p=0.73
After 2 weeks	6.0 (2.0 – 10.0)	6.0 (3.0 – 7.0)	p=0.213
After 4 weeks	8.0 (3.0 – 11.0)	7.0 (4.0 – 11.0)	p=0.654
After 3 months	8.5 (4.0 – 13.0)	7.0 (4.0 – 12.0)	p=0.022
After 6 months	9.0 (1.0 – 13.0)	7.5 (4.0 – 13.0)	p=0.156
After 12 months	10.0 (4.0 – 14.0)	8.0 (4.0 – 13.0)	p=0.138
6 months compared with preoperative VA	p<0.0001		
12 months compared with preoperative VA	p<0.0001		

bubble to fixate the posterior lamella when performing the DSEK. Although Vajaranant et al. reported no cases of this in their study, and Koenig et al. and Cheng et al. reported 3–5 %, temporary pupillary block glaucoma occurred in 11.9 % of our cases. This may have been due to the lack of an iridectomy [28, 29]. However, only one of seven cases developed post-DSEK glaucoma, and this one case had pre-existing glaucoma. Thus it is unlikely that a pupillary block glaucoma induced by the air bubble may contribute to the formation of peripheral anterior synechiae and if left untreated result in chronic angle closure [30, 31].

In accordance with other studies that provided evidence of a high incidence of steroid-induced IOP elevation following DSEK [3, 31], the rate was also high in our study (18.6 %). However, in contrast to Vajaranant et al., who attributed IOP elevation after DSEK entirely to the use of corticosteroids [3], we found cases in which post-DSEK glaucoma developed (11.9 %) despite tapering down steroids. In these cases, other reasons including angle closure due to crowding of the angle, peripheral anterior synechiae, or progressing of pre-existing glaucoma should be taken into account. Although DSEK performed with small incision and without suturing minimizes the risk of postoperative angle distorsion, there are exceptions such as decentred graft, patients with history of narrow angles, or those having undergone laser iridotomy [31]. In our study, no decentred grafts were apparent, and the maximum diameter of donor grafts was 8.5 mm. Therefore, there would have been no risk of angle closure by the graft. Additionally, our results supported the results of Fingert et al. that patients with glaucoma are much more likely to develop a steroid response than unaffected individuals [31, 32] (p=0.023).

With respect to the high incidence of steroid-induced glaucoma and the low risk of graft rejection after DSEK (1.7 % in our study, 7.5 % after 2 years: see Allan et al. [33]), an early tapering of steroids to prevent steroid induced glaucoma optic nerve damage should be considered [34, 35]. Steroid-induced IOP elevation could normally be controlled by glaucoma medication, tapering steroids, or

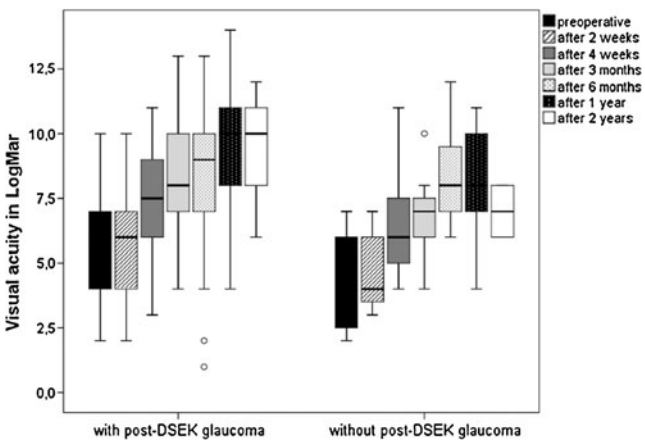

Fig. 3 *Box plot* showing the changes in visual acuity following DSEK from a preoperative mean value and at postoperative 2 weeks, 6 weeks, 3, 6, 12, and 24 months in the group with and without post-DSEK glaucoma

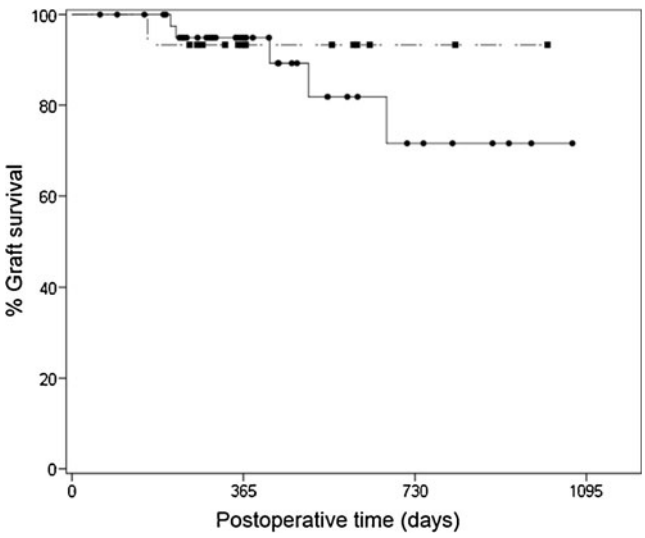

Fig. 4 Graft failure. Kaplan–Meier survival plot of clear graft survival. Endpoint is graft failure from any cause. After 1 year, 94.9 % of grafts without pre-existing glaucoma survived clear (*continuous line*), and 93.3 % of grafts with pre-existing glaucoma (*dotted line*) (p=0.581)

changing steroids. Further studies are necessary to investigate the rate of graft rejections when steroids are tapered earlier than the accepted norm [34].

Outcome

Uncontrolled IOP is associated with increased risk of poor visual outcome and graft failure after PK [33]. Wagoner et al. noted significant glaucoma worsening as a consequence of graft failure following PK. The graft survival was 75.8 % in the glaucoma worsening group versus 88.1 % in the normal group after 3 years [36, 37]. In comparison to these studies, our rate of graft survival after DSEK was higher (89.9 % after 2 years). There was no significant difference in graft failure rates between cases with or without pre-existing glaucoma (p=0.581) and with or without post-DSEK glaucoma (p=0.306).

In addition to the high rate of clear graft survival, the visual acuity improvement within the first 2 years after DSEK was statistically significant. In agreement with the findings of Vajaranant et al., there was no statistically significant difference in visual acuity improvement between cases with and without pre-existing glaucoma and between cases with and without post-DSEK glaucoma [3].

The graft failure and visual acuity results support the notion that pre-existing glaucoma and postoperative IOP elevation are not associated with a poor outcome for patients enlisted in this study [3]. The good functional outcome in this study could be due to the fact that DSEK was performed on those with Fuchs' endothelial dystrophy and with bullous keratopathy who have no other pathologies that would counteract visual acuity improvement such as graft rejection and ocular surface problems. Additionally, these patients were effectively treated by medical treatment, and no surgical procedures were necessary to control the IOP elevation [38].

Study limitation

Since this is a retrospective study rather than a designed study, the patient groupings and questions posed were framed around existent information. As an example, patients were grouped according to whether they had or had not glaucoma prior to surgery. Since the diagnosis for glaucoma was based on whether a patient had a documented history of glaucoma, prior glaucoma filtration surgery, or was using antiglaucomatous medications rather than the primary diagnostic tests for glaucoma [3], it is possible that some patients with undiagnosed glaucoma were enrolled in the non-glaucoma group. Alternatively, although it is true that patients with ocular hypertension have a higher glaucoma conversion risk [39, 40], these patients may have been erroneously included in the group of patients with pre-existing glaucoma.

Although IOP measurements are critical for monitoring the development of post-keratoplasty glaucoma [17, 19, 41, 42], conventional techniques lack precision because of corneal irregularity and increases in corneal thickness. Because the IOPs of patients enrolled in this study were measured by Goldmann tonometry in most, but not all cases, the data lacks precision. However, apart from the eyes where high IOP was noted, most eyes (42/59) after DSEK showed no IOP rise, despite comparable differences in the thickness and rigidity of the cornea pre- and postoperative (Table 3, Fig. 3). Additionally, pre- and postoperative IOP variations rarely occurred in individual patients.

In conclusion, IOP elevation after DSEK is a common problem, with a high incidence of approximatively 29 %. Pre-existing glaucoma doubled the risk of developing IOP elevation, and increased the risk of a post-DSEK glaucoma by about seven times. Steroid-induced IOP elevation was the most frequent cause, and was treated effectively by tapering down the medication. Although there were other reasons for the development of post-DSEK glaucoma, in all cases successful management by medical treatment was possible and resulted in a good visual acuity. Prospective clinical trials are required to further define the risk factors in development of post-DSEK glaucoma, and to assess the efficacy of various treatment regiments.

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Competing interest All authors, none declared.

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