Outcome of Rotational Keratoplasty

Comparison of Endothelial Cell Loss in Autografts vs Allografts

Eckart Bertelmann, MD; Christian Hartmann, MD, PhD; Matthias Scherer, MD; Peter Rieck, MD, PhD

Background: The nature of chronic endothelial cell loss in homologous corneal grafts is still unclear. Possible causes are cell migration to the recipient bed and chronic subclinical immune reaction.

Objectives: To compare endothelial cell loss after autologous rotational keratoplasty and homologous keratoplasty and present the clinical outcome of patients after rotational keratoplasty.

Methods: In this open prospective study, we included 7 consecutive patients who underwent rotational keratoplasty between 1998 and 2000 in our hospital. Patients were examined clinically every 3 months, and visual acuity, astigmatism, and endothelial cell density were evaluated. Endothelial cell densities were compared with endothelial cell counts of 293 homologous keratoplasties.

Results: Mean follow-up for autologous grafts was 39 months. Mean increase in visual acuity was 3.5 lines. Mean astigmatism was 4.75 diopters in the autologous graft group. Mean preoperative endothelial cell density was 2058 (637 cells/mm²). Mean endothelial cell density after 1 year was 1865 (639 cells/mm²), which represents a mean±SD cell loss of 15±7.19%. At the end of follow-up, endothelial cell number after autologous grafting was 1630±622 cells/mm². Endothelial cell loss after 1 year in homologous grafts was 40±21.34%. There was 1 decompensation of autologous graft in the follow-up period.

Main Outcome Measure: Comparison of endothelial cell count at different postoperative time points using nonpaired t test.

Conclusions: Endothelial cell loss in autologous grafts is significantly lower than in homologous grafts, which supports the hypothesis that chronic endothelial cell loss is due to chronic subclinical immune reactions in homologous grafts. Autologous keratoplasties can lead to good functional results and can be superior to homologous corneal grafting in suitable situations.

Arch Ophthalmol. 2004;122:1437-1440

The high rate of endothelial cell loss after penetrating keratoplasty represents an important unsolved problem: chronic endothelial cell loss probably limits transplant survival time to a greater extent than do acute immune reactions.1-4 The nature of cell number reduction is not completely understood. Factors possibly contributing to the endothelial cell loss are surgical trauma, cell exchange between donor and recipient, cell aging, acute immune reactions,5 and chronic subclinical immune reactions.6 In this context, we performed an open prospective study to compare chronic endothelial cell loss in homologous and autologous rotational transplants.

Autologous keratoplasty was described very early in the history of corneal transplantation.7 It has been performed using 3 different approaches: (1) Bilateral autologous keratoplasty: the graft is obtained from the blind contralateral eye, and the 2 corneas are exchanged. (2) Ipsilateral autologous keratoplasty: 2 minigrafts (1 central and 1 peripheral) of the same cornea are exchanged. (3) Ipsilateral autologous rotational keratoplasty.

The first 2 approaches have particularly limited indications. The bilateral autologous keratoplasty requires a blind second eye with a healthy clear cornea. The altered cornea still has to be transplantable; a contraindication might be a painful bullous keratopathy. The ipsilateral keratoplasty with 2 minitransplants requires a small circumscribed central opacity and a sufficient area of clear healthy peripheral cornea. The centrally located sutures can bias the visual results by high astigmatism and subepithelial fibrosis.

The first description of ipsilateral autologous keratoplasty dates from 1914.8

From the Augenklinik Charité Campus Virchow-Klinikum, Humboldt Universität Berlin, Berlin, Germany. The authors have no relevant financial interest in this article.

©2004 American Medical Association. All rights reserved.
Since then, different modifications of this technique have been proposed, for example, the rotation of an 8-shaped graft. Rotation keratoplasty has frequently been assessed as a (worse) replacement for homologous keratoplasty in situations of graft shortage, mainly due to poor visual outcome related to high or irregular astigmatism. For preoperative calculation of graft size and localization, geometric models have been proposed to alleviate preoperative planning. Required size and localization of the rotational transplant depend on the largest circle of clear cornea and on the distance of the circle arc of the optic center. In the present study, we compare endothelial cell loss rates of 7 autologous rotational grafts with those of homologous grafts.

The study included 7 patients (3 women, 4 men) who were consecutively operated on by the same surgeon (C.H.). A signed informed-consent form was obtained from all patients before enrollment into the study.

Preoperative endothelial cell density was examined by noncontact specular microscopy using the NonCon ROBO (Ko-nam Medical Inc, Hyogo, Japan). A central photograph was obtained, as well as additional endothelial images in all 4 quadrants of the periphery.

The rotation procedure was carried out following the technique described by Bourne and Brubaker. In 4 patients, a rotational keratoplasty was performed as a single procedure; in 2 patients, anterior synechiolysis was also performed. In 1 patient, the operation was carried out as a triple procedure with extracapsular cataract extraction and posterior chamber lens implantation.

The trephine size ranged from 7 to 8.5 mm (mean, 7.64 mm). The trephination was performed using the Barron trephination system. Double-running sutures were used in all 7 patients (10-0 and 11-0 nylon). Healon 5 (Pfizer Inc, New York, NY) was injected into the anterior chamber before trephination.

Postoperative treatment consisted of topical steroids 3 times a day for 2 weeks and topical lubricants 12 times a day. To avoid postoperative glaucoma, all patients received systemic carbonic anhydrase inhibitors on the day of surgery.

Postoperative examinations were carried out every 2 weeks during the first 3 months and every 3 months thereafter. Visual acuity, astigmatism, anterior segment, intraocular pressure, and fundus examination were performed at each control. Endothelial cell density was measured by noncontact specular microscopy. Because of temporary postoperative swelling of the transplant, early postoperative endothelial cell counts could not be obtained. The first postoperative endothelial cell density was measured 3 months postoperatively.

The corneal grafts used in homologous keratoplasties were organ cultured in the Cornea Bank, Berlin, following the guidelines of the European Eye Bank Association. The culture medium was minimal essential medium–Earle’s (Biochrom, Berlin, Germany)+2% fetal calf serum (Gibco, Paisley, Scotland). The median culture time was 13 days.

Differences in endothelial cell loss between homologous and autologous grafts after 12 months and 24 months were compared statistically using nonpaired t test. Preoperative and postoperative visual acuities were compared statistically using the Wilcoxon rank sum test.

Preoperative diagnoses of the 7 autograft patients included 4 corneal scars after perforating injury, 1 scar after keratitis due to contact lens–wear damage, 1 scar after corneal ulcer, and 1 scar after perforated corneal ulcer. The mean age of the patients was 48.2 years (range, 20-85 years). Preoperative visual acuity ranged from less than 20/200 to 20/63.

Preoperative keratometric astigmatism ranged from 1.25 diopters to 13 diopters (mean, 5.5 diopters); preoperative refractive astigmatism ranged from 1.25 diopters to 5.5 diopters (mean, 3.3 diopters). Mean±SD preoperative refractive power of the cornea was 40.6 diopters±3.67 diopters (range, 34.75-44.5 diopters). Mean preoperative endothelial cell density was 2063 cells/mm²; cell count ranged from 1200 cells/mm² to 3000 cells/mm².

Four corneas had a central scar and a circular clear periphery. Three patients had complicated corneal scars with anterior synchiae. There were 4 patients who were pseudophakic and had posterior chamber lenses. One patient had secondary glaucoma after perforating injury. Intraocular pressure was regulated preoperatively.

None of the 7 corneas showed neovascularizations. Ocular diagnoses other than the corneal scar were amblyopia in 2 patients (1 with perforating injury as a child and 1 after keratitis with perforated ulcer as a child) and age-related macular degeneration in 1 patient.

Visual acuity increased significantly in homologous and autologous transplant groups (Wilcoxon rank sum test). Mean improvement in visual acuity in autologous transplant group was 3.5 lines. The best postoperative visual acuity after rotational keratopasty was 20/25; the worst was less than 20/200. Mean postoperative astig-
matism was 4.75 diopters ± 1.5 diopters. Suture removal was carried out 1 year postoperatively in cases of persisting astigmatism over 4 diopters. Mean keratometric astigmatism was 4.7 diopters before suture removal and 4.4 diopters after suture removal in homologous keratoplasties. The differences in autologous and homologous astigmatism were not statistically significant.

Complications occurred in 2 patients during follow-up after autologous grafting. In 1 patient, a decompensation of the transplant occurred 6 weeks postoperatively, requiring a homologous transplant. Indication for rotation in this patient was a corneal scar due to a prior perforated ulcer. The rotation had been carried out as triple procedure in this patient, and preoperative endothelial cell density was the lowest of all 7 patients (1200 cells/mm²).

In another patient who had a perforating injury, a complicated retinal detachment occurred several months after the keratoplasty, requiring vitrectomy and silicone oil tamponade. In this patient, several complicated operations had been performed prior to the rotation, including lens extraction, secondary posterior lens implantation, and posterior chamber lens reposition.

Mean endothelial cell loss in all autologous keratoplasty patients 1 year after the operation was 15% ± 7.19%, compared with 35% ± 15.16% in homologous normal-risk keratoplasties and 40% ± 21.34% in all 293 keratoplasties. Endothelial cell loss after 2 years was 44% ± 16.5% in normal-risk keratoplasties and 49% ± 21.98% in all homologous keratoplasties. In autologous grafts, endothelial cell loss after 24 months was 19.3% ± 7.32%.

Endothelial cell loss rates of 293 homologous keratoplasties are shown in Figure 1. Figure 1 also shows endothelial cell loss of 29 normal-risk keratoplasties performed in patients with keratoconus. Differences in endothelial cell loss (keratoconus compared with all homologous keratoplasties) were not statistically significant. Figure 2 shows the comparison of endothelial cell numbers after homologous and autologous grafting.

Differences in endothelial cell loss between homologous and autologous grafts are statistically significant after 12, 24, and 36 months (P < .05) (autologous grafts vs homologous grafts [all indications] and autologous grafts vs homologous grafts [normal risk]) (Figure 3).

In this study, we demonstrate the outcome of a relatively small number of patients after autologous rotational keratoplasty. Indications for this special type of keratoplasty are rare; thus, studies have included comparably small numbers of patients.11,13-15 All patients in our study had a complicated preoperative situation, as shown in Figure 4B. Visual outcome is comparable with results after homologous keratoplasty in complicated situations, and postoperative astigmatism is in the same range as astigmatism after homologous keratoplasty. This is in contrast to the results of Jonas et al,11 who reported a significantly higher astig-
matism in autologous transplants. The use of double-running sutures in our study, instead of interrupted sutures in all cases, may explain partly this result.

Endothelial cell loss rate after autologous keratoplasty was significantly lower than endothelial cell loss rate after homologous keratoplasty. Late endothelial failure is known as the most important reason for late-graft decompensation after homologous keratoplasty.16

Comparison of preoperative endothelial cell densities between homologous and autologous transplants is difficult because homologous grafts undergo additional endothelial cell stress by preparation and organ culture.17 However, it has been shown that there are no statistically significant differences between endothelial cell loss rates in corneas stored in tissue culture medium (eg, Optisol, Chiron Ophthalmics, Irvine, Calif), the most widely used storage technique in the United States, and endothelial cell loss rates in organ-cultured corneas in the first 2 years.18

Furthermore, early postoperative endothelial cell counting is often not possible after homologous transplantation owing to early postoperative reduction of stromal transparency. However, despite these restrictions, differences in endothelial cell densities between homologous and autologous transplants increase over at least 3 years (Figure 2), which cannot be explained by initial endothelial cell damage during organ culture. There is only a minor additional endothelial cell loss in autologous transplants after the first year.

Immune reactions naturally cannot occur in autologous transplants. Therefore, only short-term postoperative steroid treatment, if any, is necessary after autologous grafting. For this reason as well, steroid-induced glaucoma cannot occur.

The significantly reduced endothelial cell loss rate after autologous corneal grafting supports the hypothesis that there is an immunogenic reason for chronic endothelial cell loss in homologous corneal grafts. Owing to the limited indications, autologous keratoplasties will be restricted to rare situations. However, in suitable situations, autologous keratoplasty may be superior to homologous grafting as far as long-term graft survival is concerned.

Submitted for publication March 25, 2003; final revision received February 25, 2004; accepted February 25, 2004.
Correspondence: Eckart Bertelmann, MD, Augenklinik Charité Campus Virchow-Klinikum, Humboldt University Berlin, Augustenburger Platz 1, 13353 Berlin, Germany (eckart.bertelmann@charite.de).

REFERENCES