Complications of Keratolimbal Allograft Surgery

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Purpose: To report postoperative complications of keratolimbal allograft (KLAL) transplantation in patients with bilateral total limbal stem cell deficiency.

Methods: In this retrospective observational case series, medical charts of 45 patients with at least 6 months of follow-up were reviewed. The main outcome measure was postoperative complications including graft-related issues (thickness, position, and alignment) and immunologic rejection.

Results: Sixty-six KLALs were performed on 45 eyes. The mean follow-up period was 26.1 ± 11.8 months (range, 6–48 months). Primary failure occurred in 5 eyes primarily as a result of ocular surface exposure and severe dry eyes. Graft-related complications included misalignment (4 eyes), buttonhole (4), inner-edge tear (4), inadvertent limbal trephination (2), and thick KLAL (2). Postoperatively, regional thinning of the graft was observed in 8 KLALs as a result of exposure, regional ischemia, and after epithelial rejection. Acute rejection was diagnosed 16 times in 8 eyes, whereas chronic rejection was observed in 24 eyes. At last follow-up, 12 cases (26.6%) had failed because of recurrent acute rejection (4), chronic rejection (5), refractory herpetic keratitis (1), exposure (1), and refractory papillomavirus keratitis (1).

Conclusions: KLAL may be complicated by several adverse events. The most important complications are immunologic rejections, chronic ocular surface exposure, and graft-related complications.

Key Words: limbal stem cell deficiency, limbal stem cell transplantation, keratolimbal allograft, KLAL, complication

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A number of techniques for limbal stem cell transplantation have been introduced based on the laterality and severity of limbal stem cell deficiency (LSCD).[1,2] In bilateral total LSCD, keratolimbal allograft (KLAL) is the most commonly preferred surgical treatment.[3] It has been shown that KLAL with or without penetrating keratoplasty (PKP) is an effective procedure for anatomical and visual rehabilitation of eyes with total LSCD.[4] The current literature differs in terms of case selection, surgical procedure, follow-up periods, immunosuppressive regimen, and outcome assessment.[1–6]

KLAL may be accompanied by several complications. The reported literature regarding its complications is limited and sparse. The purpose of this study was to present the postoperative ocular complications of KLAL in a series of patients with bilateral total LSCD.

MATERIALS AND METHODS

Medical charts of consecutive patients with bilateral total LSCD who underwent KLAL between October 2005 and December 2010 with at least 6 months of follow-up were reviewed. The study protocol was based on the tenets of the Declaration of Helsinki, and it was approved by the Institutional Review Board and Ethics Committee of the Ophthalmic Research Center, Shahid Beheshti University of Medical Sciences. Patients with bilateral total conjunctivalized/vascularized corneal opacity, suffering from chronic irritation, redness, tearing, and decreased visual acuity, were considered for surgery. All patients received KLAL from fresh cadaveric eyes. If corneal opacity was deep and severe, and corrected visual acuity was ≤20/200 after stem cell transplantation, subsequent PKP was performed at least 3 months thereafter. All surgeries were performed by 1 surgeon (A.B.-R.). All risks and benefits had been clearly explained to the patients and an informed consent was obtained from all of them.

Diagnosis of total LSCD was based on characteristic clinical findings. Typical clinical manifestations included complete opacification and conjunctivalization/vascularization of the cornea, chronic ocular surface inflammation, and poor epithelial integrity (persistent or recurrent epithelial defects). Clinical diagnosis was confirmed using a standard method of impression cytology.[7] Corneal transparency and conjunctivalization/vascularization of the cornea were evaluated using slit-lamp findings. Integrity of the corneal epithelium was evaluated by fluorescein staining. The findings were documented using digital corneal photography (IMAGEnet, Topcon SL-8Z; Topcon, Tokyo, Japan) in all follow-up visits.

Decrease in subjective complaints, healing of corneal epithelial defect, and appearance of a smooth and stable epithelial surface with regression of thinning, opacity, and corneal neovascularization were considered as clinical success. Appearance of a peripheral superficial vascularized corneal pannus was considered as corneal conjunctivalization, which was supported by late fluorescein staining and confirmed by

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impression cytology. Primary failure was defined as the absence of primary corneal epithelialization with progressive loss of epithelium over the KLAL within the first week. Any presence of conjunctivalization over the peripheral cornea was considered as partial failure (no central involvement). Total failure was considered when the central 5 mm of the cornea was involved. Late failure (partial or total) in the absence of any other cause (infection, significant exposure, etc) was attributed to chronic rejection. Chronic rejection presented with no visible signs of rejection other than low-grade inflammation with gradual surface failure. There was a subcategory of chronic rejection that manifested by a circumferential perilimbal engorgement, stagnation, and tortuosity of vessels with mild chemosis of KLAL conjunctival mantle. We considered this subgroup as "smoldering" rejection. This subgroup of rejection was occasionally accompanied by mild pain and photophobia. Acute rejection was defined as regional or 360-degree KLAL swelling and hyperemia accompanied by moderate to severe conjunctival injection, pain, and sometimes photophobia. A progressive moving epithelial rejection line may also be visible.

Surgical Technique

Donor Eyes

All cadaveric eyes were received either as a whole globe in a wet chamber or as a corneoscleral ring in a preservation medium (Optisol) from Central Eye Bank. They were harvested and procured with at least a 5-mm, 360-degree conjunctival mantle. They were used on the first or second postmortem day after serological screening tests were negative for human immunodeficiency virus, hepatitis B virus, hepatitis C virus, human T-lymphotropic virus, and cytomegalovirus. Histocompatibility and ABO matching were not done. All harvested corneoscleral grafts were trephined from the endothelial side with a 7- to 8-mm Hessburg–Barron trephine. This donor corneolimbal graft placed over a viscoelastic cushion was trimmed and thinned from the endothelial aspect by dissection with sharp Westcott scissors.

Recipient Eye

Surgery was done under general anesthesia in all cases. After a 360-degree conjunctival peritomy, subconjunctival scar tissues were removed as much as possible. In the case of extensive subconjunctival scar formation, 0.02% mitomycin C was applied to the adjacent conjunctival fornix for 3 to 5 minutes. Then, it was thoroughly irrigated with balanced salt solution. The conjunctiva was recessed 3 to 5 mm away from the limbus. The corneal pannus was removed completely using a corneal scissors with special caution not to perforate the cornea. The exposed ocular surface was thoroughly covered by cryopreserved human amniotic membrane, stroma side down. It was fixed to the ocular surface with 2 running 10-0 nylon sutures at the limbus and 3.0 mm away. In some cases, instead of sutures, fibrin glue was used to fix the human amniotic membrane to the ocular surface. Donor grafts were transferred to the prepared bed and sutured/glued onto the scleral beds with sixteen 10-0 nylon sutures. The KLAL was covered with an overlay amniotic membrane (n = 32) or a bandage contact lens (Kontur Kontact Lens Co, Hercules, CA; n = 34). At the end of surgery, upper and lower punctal occlusion and lateral tarsorrhaphy were done in all cases. If necessary, corneal transplantation was performed using a Hessburg–Barron vacuum trephine and interrupted 10-0 nylon sutures in a standard manner.

Postoperative Management

Topical antibiotic (0.5% chloramphenicol) and steroid (0.1% betamethasone) drops 4 times a day were started after surgery. The former was discontinued when corneal epithelialization was complete, whereas the latter was tapered down according to ocular surface inflammation. Oral prednisolone 1 mg/kg/d was started postoperatively, which was tapered during 8 to 12 weeks after surgery when inflammation subsided. All cases were kept on the maintenance dose of prednisolone (5–10 mg) thereafter. Patients received topical 20% autologous serum drops every 2 hours, prepared from their own blood drawn during surgery, to decrease inflammation and facilitate epithelial healing. It was tapered off after 2 to 3 months. Thereafter, topical preservative-free artificial tears (Artelac; Bausch and Lomb, Rochester, NY) or lubricating gels (Liposic; Bausch and Lomb) were indefinitely continued. All patients used high-DK contact lenses (Kontur Kontact Lens Co) after KLAL and subsequent PKP in the long term.

Immunosuppression Regimen

All patients received both 1 g of oral mycophenolate mofetil and 4 mg of tacrolimus twice a day starting 1 week preoperatively. The trough levels of mycophenolate mofetil and tacrolimus were checked every 1 to 3 months, and their doses were adjusted according to ocular surface inflammation and systemic adverse effects in collaboration with a transplant specialist. They were tapered down after 12 months and, based on the level of inflammation, were indefinitely kept on the maintenance dose. They were discontinued if irreversible graft failure or significant adverse effects were noted. An antiherpetic prophylactic regimen of 0.8 to 1.2 g of systemic acyclovir was administered for 4 to 6 months after surgery when the ocular surface was not inflamed.

Acute and smoldering rejections were treated with increasing topical and systemic steroids along with increased systemic immunosuppression. However, very mild cases were managed only by increasing topical steroids. Chronic rejection was treated with an increase in dosage of systemic immunosuppressives.

RESULTS

Sixty-six KLALs were performed on 45 eyes (19 right eyes and 26 left eyes) of 45 patients (41 men and 4 women) with bilateral total LSCD. The etiologies of the LSCD included acidic (n = 21) or alkaline (n = 15) chemical injuries, thermal burn (n = 5), and Stevens–Johnson syndrome (SJS; n = 4). The mean age at the time of surgery was 26.7 ± 8.7 years (range, 12–50 years). The mean interval between the ocular injury and the surgery was 28.0 ± 3.4 months (range,

6–32 months). The mean follow-up period was 26.1 ± 11.8 months (range, 6–48 months).

In cases with primary failure, repeat KLAL was done 2 to 3 weeks later. KLAL was repeated once in 3 eyes and twice in 2 eyes, which had failed primarily. Likewise, repeat KLAL was done in 14 eyes because of late graft failure. At last follow-up, 33 of the 45 eyes (73.4%) had a stable ocular surface and were considered a clinical success. Twelve eyes (26.6%) were considered to have intractable failure because of recurrent acute rejection (4), chronic rejection (5), refractory herpetic keratitis (1), exposure (1), and refractory papillomavirus keratitis (1). The best spectacle-corrected visual acuity (BSCVA) before KLAL was −2.12 ± 0.34 logarithm of the minimum angle of resolution (logMAR; −3.0 to −2.0). Excluding failed surgeries, the BSCVA at the last follow-up was −0.42 ± 0.12 logMAR (−0.68 to −0.23) and −0.28 ± 0.12 logMAR (−0.56 to −0.14) in eyes without and with PKP, respectively. BSCVA had improved at least 2 lines on the Snellen chart in 84.8% of eyes that underwent KLAL alone and in 100% of eyes that had also undergone a subsequent keratoplasty.

Complications

A wave of epithelial healing appeared as 360-degree centripetal progressive epithelium in the first 48 hours after surgery in 40 eyes that completed epithelial healing during 4 weeks. Of them, 28 healed during 2 weeks. Delayed epithelial healing (healing between 2 and 4 weeks) was observed in 12 eyes: inadvertent decentered limbal trephination (2), irregular corneal stromal bed (2), regional thick KLAL (2), mild exposure (3), fair donor tissue quality (1), floppy eyelid syndrome (1), and dry eye in the context of SJS (1). Graft-related complications included inadvertent decentered limbal trephination (2), regionally thick KLAL (2), graft misalignment (4), buttonhole (4), and inner edge tear (4), which in some cases also contributed to the delayed epithelialization. In eyes with inadvertent decentered limbal trephination, epithelialization was delayed and asymmetric, with the adjacent corneal epithelium being the last area to epithelialize. Thick limbal grafts led to dellen formation and corneal thinning in both. They were both trimmed and repositioned back to the surgical site with successful epithelialization. KLAL misalignment was unacceptable in 2 cases that were subsequently resutured and repositioned back during the first week after surgery to prevent chronic graft exposure and/or a probable trephination during future PKP. All other etiologies were conservatively managed until complete epithelialization.

Primary failure occurred as a result of ocular surface exposure (3), dry eye in the context of SJS (1), and poor quality of the donor (1). In these 5 cases, the corneal surface was never reepithelialized and persistent epithelial defect (PED) extended over the KLAL. All grafts were replaced by a fresh KLAL after 2 to 3 weeks. Ocular surface exposure was surgically corrected in these 3 cases. In one eye with remaining exposure and one with SJS, repeat KLAL was performed because of another failure. PED occurred in these eyes again, and the ocular surface was covered with mucous membrane graft to prevent corneal perforation. Repeat KLAL was performed in 14 eyes with late graft failure as a result of floppy eyelid syndrome (1), mild chronic exposure (1), progressive pyogenic granuloma in the interface (2), insufficient immunosuppression or chronic rejection (8), and recurrent acute epithelial rejections (2).

Acute rejection, which started from the superior part of the KLAL in all cases, was evident as a progressive epithelial rejection line with/without corneal epithelial defect behind it. It occurred 16 times in 8 eyes (once in 2 eyes, 2 times in 4 eyes, and 3 times in 2 eyes). Clinically, it was associated with dry eye (1), trichiasis (1), lagophthalmos (1), loose sutures (1; Fig. 1), and a history of smoldering rejection (4). The cornea behind the leading edge of epithelial rejection line was invaded by conjunctiva with superficial neovascularization and mild to moderate haze formation. Four of the 6 eyes that experienced recurrent graft rejections ultimately failed, including both of the eyes that had 3 acute rejection episodes. KLAL thinning occurred in 4 eyes after acute rejection (2 eyes in recurrent cases). Chronic rejection was observed in 24 eyes that were treated by increasing the dosage of immunosuppressives. Despite that, 8 eyes failed and were later regrafted. Four of the 8 regrafted eyes failed again because of chronic rejection refractory to medical therapy. Of the 24 eyes with chronic rejection, smoldering rejection was observed in 8 eyes. In this subgroup, engorgement, tortuosity, stagnation of vessels, and edema reduced during 6 to 8 weeks after treatment. Three to 4 months after smoldering rejection, acute rejection occurred in 4 of them, which led to sectoral conjunctivalization. Of these, 2 eyes failed because of recurrent acute rejection (Fig. 2).

Regional KLAL thinning was observed in 8 KLALs. It was attributed to chronic mild to moderate exposure (2; Fig. 3), regional ischemia (2; Fig. 4), and after acute epithelial rejection (4). In 4 cases, thinning of the graft led to progressive conjunctivalization and vascularization threatening the visual axis. Sectoral KLAL was performed in these 4 cases successfully.

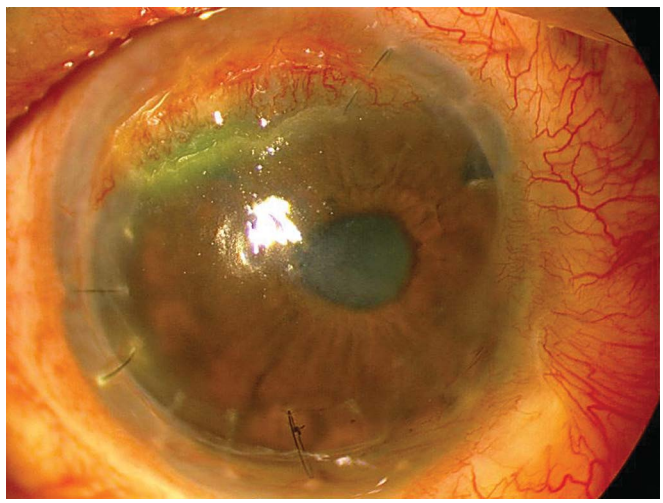

FIGURE 1. Acute rejection because of a loose suture. Note the epithelial rejection line, local inflammation, PED, and invading vascular tissue behind it.

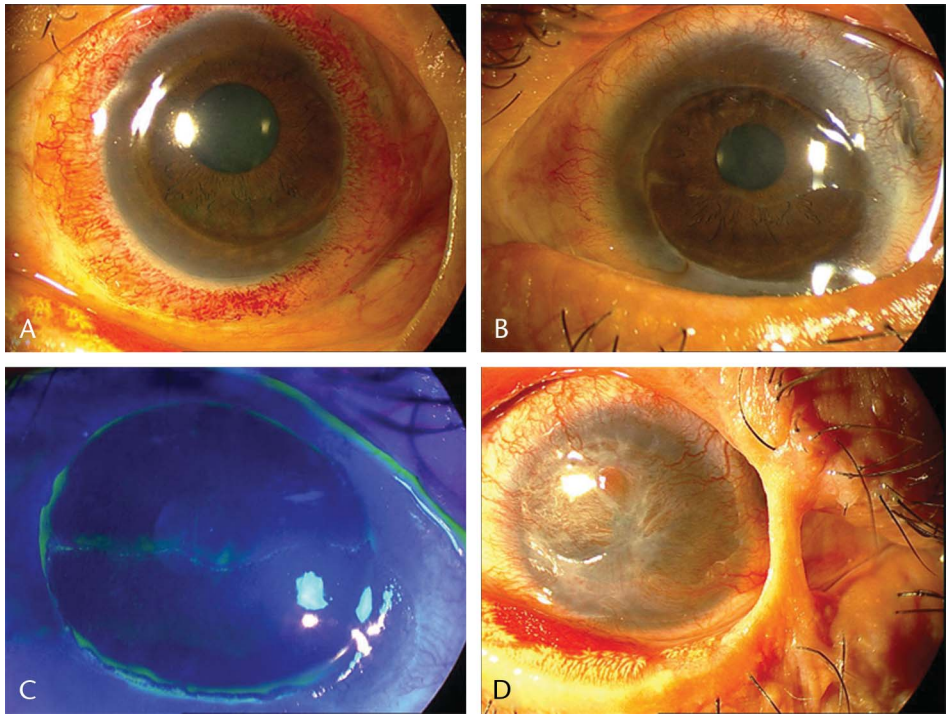

FIGURE 2. A, Smoldering rejection as a subgroup of chronic rejection. Note the vascular engorgement and stagnation of blood vessels in an inflamed and edematous conjunctival mantle of KLAL. B, Acute rejection episode occurred in the same eye 4 months after resolution of smoldering rejection. C, Fluorescein staining of the same cornea with acute rejection demonstrating epithelial rejection line. D, Late KLAL failure because of chronic and several acute rejections in the same eye.

Progressive interface granulation tissue was observed in 2 eyes. It was associated with intrastromal corneal hemorrhage in one eye and partial vascularization in another. Bacterial corneal ulcer occurred in 4 eyes, 2 of which had chronic rejection. In one case with chronic rejection, this led to corneal perforation, which was managed by tectonic keratoplasty to save the eye. High intraocular pressure occurred in 8 eyes. In 6 eyes refractory to medical therapy, shunt procedure (4), cyclophotocoagulation (1), and cyclocryotherapy (1) were performed. Phacoemulsification and posterior chamber intraocular lens implantation were performed in 6 eyes because of cataract formation. Scleral thinning and staphyloma formation were observed in 2 eyes behind the KLAL, which were assumed to be a result of mitomycin C application to ischemic sclera (5 minutes) during fornix reconstruction. There were 4 cases with herpetic keratitis and 2 cases with papillomavirus keratitis, which were confirmed by polymerase chain reaction. Herpetic ocular surface disease was controlled with systemic acyclovir and by reducing the dosage of immunosuppressive medication. In one eye, it was refractory to treatment, which required discontinuation of systemic immunosuppression leading to failure of the graft. Both cases with papillomavirus keratitis were treated with topical interferon-α, which ultimately led to

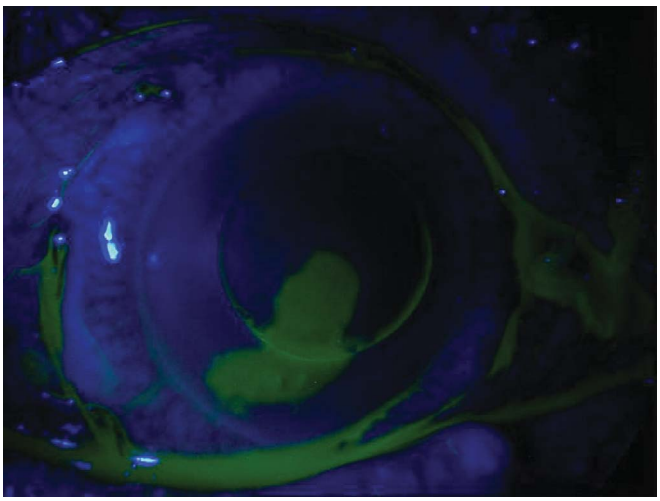

FIGURE 3. Fluorescein staining of a corneal epithelial defect extending over KLAL secondary to moderate exposure.

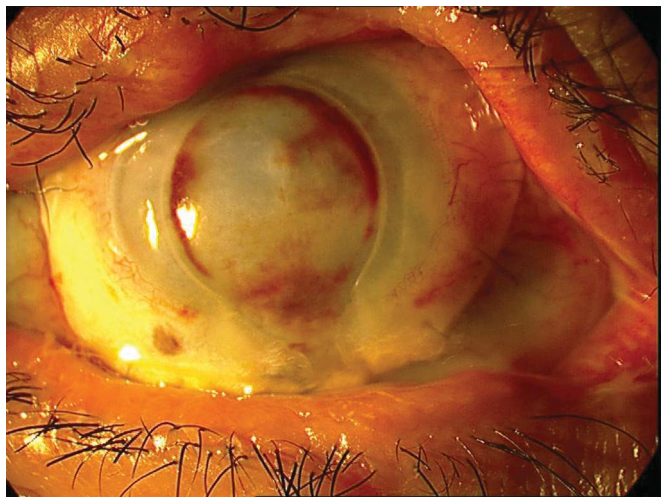

FIGURE 4. Sectoral KLAL thinning in an eye with regional severe ischemia.

failure of the graft in one (Fig. 5). Other complications included microperforation, corneoscleral dellen formation, epithelial cysts, and graft hematoma.

Thirty-four optical PKPs were performed on 26 eyes. The mean interval between KLAL and first PKP was 7.3 ± 3.4 months (range, 3–18 months). Corneal endothelial graft rejection was observed in 9 cases. Recurrent endothelial rejection led to repeat PKP once in 2 eyes and twice in 1 eye. Occasional epithelial breakdown was observed in 12 postkeratoplasty eyes. This was attributed to dry eye, surface failure, and exposure and was managed in all cases by punctal occlusion, bandage lens, or tarsorrhaphy.

DISCUSSION

In this study, it was shown that KLAL surgery is an effective procedure for anatomical and visual rehabilitation of eyes with bilateral total LSCD. However, it may be accompanied by complications. The most significant complications are related to immunologic rejections, chronic ocular surface exposure, and graft-related complications (thickness, position, and alignment). The final outcome of these complications is mainly ocular surface epithelial breakdown including PED, thinning, and progressive corneal conjunctivalization.

Rejection can be categorized into acute and chronic in these cases. Acute rejection can lead to partial LSCD manifesting as sectoral corneal conjunctivalization. It has been reported in 0% to >50% of published studies.[8–15] In our study, acute epithelial rejection was observed in 8 eyes (17%). There is a subgroup of chronic rejection that we named smoldering rejection. It is manifested by limbal engorgement, vascular stagnation, and tortuosity of conjunctival mantle; subconjunctival hemorrhage; and edema of KLAL. In our study, there were 8 eyes with smoldering rejection, of which 4 eyes eventually developed acute rejection. Smoldering rejections should be aggressively treated with topical, subconjunctival, and systemic steroids[10]; however, it disappears slowly.

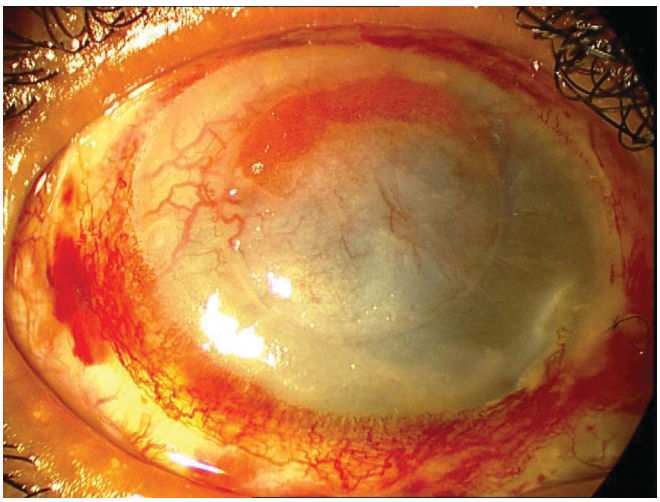

FIGURE 5. Papillomavirus keratitis in an eye with successful KLAL 2 months after surgery.

The most common reason for late graft failure is chronic low-grade rejection, which may be prevented or delayed by adequate immunosuppression.[16,17] Chronic rejection, which may develop after acute and smoldering rejections, is observed as a progressive asymmetric 360-degree conjunctivalization/vascularization of the cornea. It is usually associated with thinning of KLAL. Because limbal tissue is highly vascularized and antigenic and rich in Langerhans cells, KLALs carry a high risk of immune rejection.[9] KLAL transplantation is best done in close collaboration with an organ transplant team experienced with immunosuppressive therapy. It has been shown that KLAL survival can be prolonged with effective and adequate immunosuppression.[3,9,10]

Intraoperative graft-related complications are thick graft, buttonhole, decentered trephination, inner-edge tear, and misalignment. A thick limbal graft can create a step off and lead to improper distribution of the tear film with subsequent dellen formation.[2,18] Amniotic membrane transplantation may sometimes help reduce the step off between KLAL and the ocular surface. Gentle trimming of the graft by a sharp scissors or a scalpel blade prevents buttonholing or tearing of the inner edge of the graft. Recently, other techniques for creating thin KLALs have been described, which may facilitate dissection of thinner KLALs.[19] Eccentric trephination can be prevented by drying the epithelial surface and meticulous technique. Limbal alignment is important to prevent exposure and probable cut during trephination in future keratoplasty. Choosing a proper size trephine with good centration is important. Excessive edema of conjunctival mantle can be caused by excessive surgical manipulation, inadequate Tenon excision, poor graft orientation, young age, and graft hematoma.[2,18]

Corneal transplantation may be needed for visual rehabilitation after KLAL. However, patients who require subsequent keratoplasty may be at higher risk for immunologic failure.[3,12] Epithelial problems including punctate epithelial erosions and breakdown can be exacerbated after corneal transplantation in a KLAL patient. This may be because of an additional neurotrophic component and surface irregularities in eyes with borderline stem cell reserve. Using fresh donor corneal grafts with an intact epithelium is important in patients receiving corneal transplantation after KLAL.

In summary, anatomical results and visual outcomes of KLAL surgery for ocular surface reconstruction are acceptable. However, several complications may happen. The most important complications are immunologic rejections, ocular surface exposure, and graft-related complications (thickness, position, and alignment). Adequate immunosuppression, full correction of adnexal abnormalities, a good tear film status, and proper handling and dissection of limbal grafts are the most important issues to prevent complications. Immediate diagnosis and prompt management of complications will prevent subsequent adverse events including graft failure.

REFERENCES

1. Liang L, Sheha H, Li J, et al. Limbal stem cell transplantation: new progresses and challenges. *Eye (Lond)*. 2009;23:1946–1953.
2. Fernandes M, Sangwan VS, Rao SK, et al. Limbal stem cell transplantation. *Indian J Ophthalmol*. 2004;52:5–22.

3. Espana EM, Di Pascuale M, Grueterich M, et al. Keratolimbal allograft in corneal reconstruction. *Eye (Lond)*. 2004;18:406–417.
4. Cauchi PA, Ang GS, Azuara-Blanco A, et al. A systematic literature review of surgical interventions for limbal stem cell deficiency in humans. *Am J Ophthalmol*. 2008;146:251–259.
5. Djalilian AR, Mahesh SP, Koch CA, et al. Survival of donor epithelial cells after limbal stem cell transplantation. *Invest Ophthalmol Vis Sci*. 2005;46:803–807.
6. Kim JY, Djalilian AR, Schwartz GS, et al. Ocular surface reconstruction: limbal stem cell transplantation. *Ophthalmol Clin North Am*. 2003;16: 67–77.
7. Tseng SC. Staging of conjunctival squamous metaplasia by impression cytology. *Ophthalmology*. 1985;92:728–733.
8. Henderson TR, Coster DJ, Williams KA. The long term outcome of limbal allografts: the search for surviving cells. *Br J Ophthalmol*. 2001;85:604–609.
9. Ilari L, Daya SM. Long-term outcomes of keratolimbal allograft for the treatment of severe ocular surface disorders. *Ophthalmology*. 2002;109: 1278–1284.
10. Liang L, Sheha H, Tseng SC. Long-term outcomes of keratolimbal allograft for total limbal stem cell deficiency using combined immunosuppressive agents and correction of ocular surface deficits. *Arch Ophthalmol*. 2009;127:1428–1434.
11. Maruyama-Hosoi F, Shimazaki J, Shimmura S, et al. Changes observed in keratolimbal allograft. *Cornea*. 2006;25:377–382.
12. Solomon A, Ellies P, Anderson DF, et al. Long-term outcome of keratolimbal allograft with or without penetrating keratoplasty for total limbal stem cell deficiency. *Ophthalmology*. 2002;109: 1159–1166.
13. Tsai RJ, Tseng SC. Human allograft limbal transplantation for corneal surface reconstruction. *Cornea*. 1994;13:389–400.
14. Han ES, Wee WR, Lee JH, et al. Long-term outcome and prognostic factor analysis for keratolimbal allografts. *Graefes Arch Clin Exp Ophthalmol*. 2011;249:1697–1704.
15. Wylegala E, Dobrowolski D, Tarnawska D, et al. Limbal stem cells transplantation in the reconstruction of the ocular surface: 6 years experience. *Eur J Ophthalmol*. 2008;18:886–890.
16. Daya SM, Bell RW, Habib NE, et al. Clinical and pathologic findings in human keratolimbal allograft rejection. *Cornea*. 2000;19:443–450.
17. Holland EJ, Schwartz GS. Epithelial stem-cell transplantation for severe ocular-surface disease. *N Engl J Med*. 1999;340:1752–1753.
18. Eslani M, Baradaran-Rafii A, Djallilian A. Conjunctival-limbal autograft. In: Thomson W, ed. *Advances in Eye Research*. Hauppauge, NY: Nova Publishers; 2011. pp 1–46.
19. Nassiri N, Pandya HK, Djalilian AR. Limbal allograft transplantation using fibrin glue. *Arch Ophthalmol*. 2011;129:218–222.