combining HSCT with subsequent kidney transplantation using the same HLA-matched donor.[8,9] After HSCT, the immune system reconstituted by the HLA-identical donor's cells should not view other transplanted tissue as foreign because it is from the same donor. In our 3 eyes, long-term successful allogenic OSST without immunosuppression was achieved. Because SI could be avoided, the associated risks for the recipient were theoretically more akin to a conjunctival limbal autograft.

With long-term follow-up (>7 years), treatment with lr-CLAL has maintained a stable ocular surface. By providing conjunctival tissue with additional goblet cells, a lr-CLAL also helps to treat the GVHD-related keratoconjunctivitis sicca. This may be a benefit of using a lr-CLAL in these cases over a procedure such as CLET.

Although GVHD-related LSCD is uncommon, lr-CLAL can successfully restore the ocular surface. In the setting of GVHD-related LSCD after HSCT, there is a unique opportunity to provide a lr-CLAL from the same HLA-identical donor as the HSCT, allowing for true long-term allograft tolerance and avoidance of SI.

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Albert Y. Cheung, MD,[*,†] Brad M. Genereux, OD,[*] Nicholas J. Auteri, BS,[*] Enrica Sarnicola, MD,[*,‡] Amit Govil, MD,[§] Edward J. Holland, MD[*,†]

[*]Cincinnati Eye Institute, Cincinnati, OH; [†]Department of Ophthalmology, University of Cincinnati, Cincinnati, OH; [‡]Department of Medicine Surgery and Neuroscience, University of Siena, Siena, Italy; [§]Division of Nephrology and Hypertension, University of Cincinnati, Cincinnati, OH.

Correspondence to:
Edward J. Holland, MD, 580 South Loop Road, Suite 200, Edgewood, KY 41017; eholland@holprovision.com: eholland@holprovision.com

References

1. Ferrara JL, Levine JE, Reddy P, Holler E. Graft-versus-host disease. *Lancet*. 2009;373:1550-61.
2. Ple-plakon PA, Mian SI. Ocular graft-versus-host disease. In: Holland EJ, Mannis MJ, Lee WB, eds. *Ocular Surface Disease: Cornea, conjunctiva, and Tear Film.* New York: Elsevier; 2013, pp. 178-81.
3. Heinz C, Steuhl K-P, Meller D. Hornhautperforationen bei vitamin a-mangel. *Ophthalmologe*. 2004;101:614-7.
4. Nakamura T, Inatomi T, Sotozono C, et al. Transplantation of autologous serum-derived cultivated corneal epithelial equivalents for the treatment of severe ocular surface disease. *Ophthalmology*. 2006;113:1765-72.
5. Meller D, Fuchsluger T, Pauklin M, Steuhl KP. Ocular surface reconstruction in graft-versus-host disease with HLA-identical living-related allogeneic cultivated limbal epithelium after hematopoietic stem cell transplantation from the same donor. *Cornea*. 2009;28:233-6.
6. Sivaraman KR, Jivrajka RV, Soin K, et al. Superior limbic keratoconjunctivitis-like inflammation in patients with chronic graft-versus-host disease. *Ocul Surf*. 2016;14:393-400.
7. Holland EJ, Schwartz GS. The Paton lecture: ocular surface transplantation: 10 years' experience. *Cornea*. 2004;23:425-31.
8. Spitzer TR, Delmonico F, Tolkoff-Rubin N, et al. Combined histocompatibility leukocyte antigenmatched donor bone marrow and renal transplantation for multiple myeloma with end stage renal disease: the induction of allograft tolerance through mixed lympho-hematopoietic chimerism. *Transplantation*. 1999;68:480-4.
9. Scandling JD, Busque S, Dejbakhsh-Jones S, et al. Tolerance and chimerism after renal and hematopoietic-cell transplantation. *N Engl J Med*. 2008;358:362-8.

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Unusual ocular presentation in a patient with lichen planus

Case Report

Lichen planus is a relatively common mucocutaneous disorder of unknown etiology that is thought to be immunologically mediated. It is a chronic disorder characterized by periods of exacerbation and remission. Lichen planus has variable clinical manifestations, including the skin, oral mucosa, genital mucosa, nails, and scalp. The characteristic lesions have a well-established clinical appearance and histological features that help in establishing the diagnosis.[1] Ocular involvement may occur in patients with lichen planus. Although involvement of the eyelids, lacrimal ducts, conjunctiva, and cornea has been described in patients with lichen planus,[2] scleritis has not been reported in such patients. We report a case of scleritis diagnosed in a patient with lichen planus.

A 46-year-old female presented with redness, discomfort, and pain on ocular movement in the right eye. Review of systems revealed history of pruritic skin lesions of the elbows and knees. Ocular history revealed epiphora, for which she had irrigation of the nasolacrimal ducts, which revealed patent passages. She had been recently diagnosed with lichen planus by her dermatologist, for which she was given local steroid skin creams.

On eye examination, her uncorrected visual acuity was 20/25 OU. The Schirmer test without anaesthesia revealed 15 mm of wetting in the right eye and 13 mm of wetting in the left eye after 2 minutes. Slit-lamp biomicroscopy of the right eye revealed normal lids and conjunctiva. The sclera was injected temporally and inferiorly, with marked tenderness; the cornea was clear; the anterior chamber was quiet; the pupil was

round, regular, and reactive; and the lens was clear. Slit-lamp biomicroscopy of the left eye revealed normal findings.

The diagnosis of scleritis was confirmed by anterior segment optical coherence tomography, which was done on the sclera of the right eye. It revealed intrascleral edema and increased sclera thickness consistent with the diagnosis of scleritis. Other causes of scleritis were excluded by extensive questionnaire and negative laboratory work-up. Complete blood count revealed slightly increased neutrophils; erythrocyte sedimentation rate was normal; antinuclear antibodies (ANA) were elevated; *anti–*cyclic citrullinated peptide (*anti-CCP*) antibody testing revealed negative results; antineutrophil cytoplasmic antibodies (c,p ANCA) were negative; tuberculin skin testing was negative; and uric acid levels were normal.

She was treated with topical prednisolone acetate eye drops and oral piroxicam 20 mg tab. After 9 days, she came for follow-up and was found to have markedly improved symptoms and signs of scleritis. The patient was followed up for 3 weeks with complete resolution of scleritis. Oral piroxicam was discontinued, and topical prednisolone acetate eye drops were tapered.

Discussion

Ocular involvement may occur in patients with lichen planus. It may affect the eyelids in the form of pruritic violaceous papules.[2] Ocular inflammatory changes with cellular infiltration, fibrosis, and basement membrane thickening in patients with lichen planus may lead to lacrimal duct stenosis and lacrimal canalicular obstruction.[3] Conjunctival involvement has been described in lichen planus in the form of conjunctival inflammation, cicatrization, and subepithelial scarring.[2]

Lichen planus may be also associated with keratitis, keratoconjunctivitis, and persistent epithelial defects, leading to noninfectious or infectious corneal ulceration.[2] Our patient developed severe scleritis in the right eye shortly after a diagnosis of lichen planus was made by her dermatologist. She gave a history of pruritic skin lesions (initially diagnosed as psoriasis and eczema and later as lichen planus) and epiphora. She reported irrigation of both nasolacrimal ducts, which revealed patent passages. She might have had canalicular or ductal stenosis, which has been previously reported in association with lichen planus. Other serious and more common underlying pathologies were ruled out, particularly rheumatoid arthritis (*negative anti-CCP*) and *Wegener's granulomatosis* and polyarteritis nodosa (negative c,p ANCA). In addition, serum uric acid levels were normal, and there were no clinical features of systemic lupus erythematosus.

The long-term prognosis in patients with lichen planus is variable. Generalized lichen planus tends to heal faster than other variants but has a greater likelihood of relapse. In patients with cutaneous lichen planus, the lesions resolve within 6 months to a year; however, the hypertrophic variant of the disease tends to persist for years if not properly treated. Improperly treated reticular oral lichen planus has a chronic course and may be progressive in nature without complete resolution. Lichen planus may have a recurrent pattern.[2]

Lichen planus is a T-cell-mediated autoimmune disease. Inflammatory cells involved in this process consist of T-helper and T-cytotoxic lymphocytes and other inflammatory cells. T-cell activation plays a central role in the pathogenesis of lichen planus. Normally, the human sclera contains few or no macrophages, Langerhans' cells, neutrophils, or lymphocytes. After scleral inflammation, there is a marked increase in T-helper lymphocytes with a high T-helper: T-suppressor ratio. These findings suggest that T lymphocytes may play a role in some forms of scleritis.[4] On the other hand, ANA may play a role in the pathogenesis of scleritis in patients with lichen planus. Serum ANA were more frequently and significantly observed in patients with some forms of lichen planus.[5,6] Zierhut et al. found that 28% of their patients with scleritis had positive ANA.[7] These findings may suggest an autoimmune disposition related to collagenosis.[7] In our patient, ANA may have contributed to the development of scleritis. The cutaneous lichen planus lesions in our patient responded dramatically to the local steroid creams. Similarly, scleritis responded to anti-inflammatory therapy, suggesting common underlying pathologic mechanisms. However, the exact mechanism of how lichen planus might have affected the sclera rather than the conjunctival or corneal epithelium is yet to be identified.

Lichen planus should be included in the differential diagnosis of cases that may underlie scleritis in the absence of other systemic disease associations. This is particularly important in patients presenting with other ocular features and skin lesions suggestive of lichen planus. It is important to diagnose and treat both ocular and systemic disease efficiently to avoid potentially serious complications.

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Samir S. Shoughy, MD
The Eye Center and The Eye Foundation for Research in Ophthalmology, Riyadh, Saudi Arabia.

Correspondence to:
Samir S. Shoughy, MD; samir.shawki@hotmail.com.: samir.shawki@hotmail.com

References

1. Krupaa RJ, Sankari SL, Masthan KMK, Rajesh E. Oral lichen planus: an overview. *J Pharm Bioallied Sci*. 2015;7:S158-61.

2. Gorouhi F, Davari P, Fazel N. Cutaneous and mucosal lichen planus: a comprehensive review of clinical subtypes, risk factors, diagnosis, and prognosis. *Sci World J*. 2014;2014:742826.
3. Webber NK, Setterfield JF, Lewis FM, Neill SM. Lacrimal canalicular duct scarring in patients with lichen planus. *Arch Dermatol*. 2012; 148:224-7.
4. Fong LP, Sainz de la Maza M, Rice BA, Kupferman AE, Foster CS. Immunopathology of scleritis. *Ophthalmology*. 1991;98:472-9.
5. Lin HP, Wang YP, Chia JS, Sun A. Modulation of serum antinuclear antibody levels by levamisole treatment in patients with oral lichen planus. *J Formos Med Assoc*. 2011;110:316-21.
6. Carrizosa AM, Elorza FL, Camacho FM. Antinuclear antibodies in patients with lichen planus. *Exp Dermatol*. 1997;6:54-6.
7. Zierhut M, Denk PO, Klein R, Berg PA, Erb C, Thiel HJ. [Autoantibody pattern in scleritis and episcleritis]. *Ophthalmologe*. 1997;94:157-60.

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Small-pupil cataract surgery with/without hooks using femtosecond laser with fluid interface

Cataract surgery is one of the most commonly performed surgical procedures in the world. The classic surgical method was advanced in 2001 after the introduction of femtosecond laser technology.[1] Before 2001, the surgeon performed all of the steps in the procedure manually using an ultrasound probe to break down the cataract. Currently, many of these steps can be automated with the use of femtosecond laser–assisted cataract surgery (FLACS), which emits ultra-short optical pulses.[2] A study investigating the physiological effects of FLACS on the eye highlighted a disadvantage: Miosis occurs during the gap in time between procedures—that is, cataract breakdown during laser application and the ultrasound phacoemulsification remainder of the operation. Therefore, the smaller size of capsulotomy (<4.6 mm), along with pre-existing small pupil, was thought to be more challenging.[1]

Case Reports

We had 2 identical patients with cataracts and small pupils (3.5 mm each) scheduled for FLACS under local anaesthesia. In this video report (Video 1) we demonstrate the ease and feasibility of using FLACS with and without the pupil-expanding device like iris hooks.

In the first case (Video 1; Fig. 1), with 3.5 mm pupil and a cataract, no iris hooks were used. A desired capsulotomy diameter was measured using surgical callipers. A 3.4 mm anterior capsulotomy was performed with fragmentation of the nucleus using a mobile Femto LDV Z8 (Ziemer, Port, Switzerland), which was housed in the same operating theatre. After this, hydrodissection and phacoemulsification was performed gently, as was the insertion of the lens. In the case of the latter, we recommend the insertion of the lens in such a way that the leading haptic goes into the capsule bag through the small capsulotomy opening and then gently tucking the tailing haptic inside, avoiding undue stress to the anterior capsular margins (Video 1). The case was completed after gentle irrigation and aspiration and injection of intracameral cefuroxime.

In the second case (Video 1; Fig. 2), with 3.5 mm pupil and a cataract, a pupil-expanding device, iris hooks, was used. Four paracentesis incisions were created using 15-degree blade. Iris hooks were inserted without inflating the anterior chamber with viscoelastic, and the capsulotomy diameter was measured using a surgical callipers. A suction ring was placed over the hooks to identify and measure the amount of the hook that needed to be trimmed. These were then cut flush to the eye to ensure that they did not touch the docking interface of the femtosecond laser when the laser was docked onto the suction ring. The suction ring was placed over the eye while ensuring that the hook ends were placed within the inner rim of the suction ring, and the suction cup was filled with balanced salt solution (BSS; Alcon, Fort Worth, Tex.) after attaining appropriate vacuum. The iris hooks were removed after implantation of the intraocular lens and before irrigation and aspiration. The remainder of the surgery followed a routine cataract surgery methodology (Video 1) as described in the first case.

In both cases, the mobile Femto LDV Z8 (Ziemer) was placed next to the patient's bed. After painting and draping, the femtosecond laser was docked and laser procedure was completed (Video 1). After the application of femtosecond laser, the assistant moved the laser away and pulled the phacoemulsification machine nearer to the patient's bed. This ensured that no time was wasted between the laser application and phacoemulsification (Video 1).

Outcomes and Follow-up

The patients were followed up at 1 and 3 months after the surgery. The best-corrected visual acuity (BCVA) improved from 0.2 logMAR preoperatively to 0 logMAR postoperatively at 1 month in the first case and 0.3 logMAR preoperatively to 0 logMAR in the second case. The