Corneal Perforation After Conductive Keratoplasty in a Patient With Previously Undiagnosed Sjögren Syndrome

Conductive keratoplasty (CK) was approved by the US Food and Drug Administration in March 2004 for the correction of hyperopia ranging from +0.75 to +3.00 diopters (D) and 0.75 D or less of cylinder in patients older than 40 years. With CK, hyperopic correction is achieved through the use of a nonablative radiofrequency that shrinks corneal stromal collagen and steepens the central cornea. A keratoplast is used to apply 8 to 32 treatment spots at the 6-, 7-, or 8-mm optical zone.

Patients chosen for CK are carefully screened to avert ocular complications. We report the case of a patient with previously undiagnosed Sjögren syndrome who developed corneal perforation after CK.

Report of a Case. A 52-year-old Chinese woman with uncorrected visual acuity of 20/25 OU and dry eyes with normal meibomian gland status underwent uncomplicated CK of the right eye because of hyperopia (16 spots at 7- and 8-mm optical zones) for planned monovision on March 14, 2005, in Hong Kong. One week later, her manifest refraction was −0.25 + 0.25 × 90, uncorrected vision was 20/20−2, and near vision was Jaeger 3. Postoperative inferior corneal punctate staining was treated with artificial tears.

On March 31, 2005, 2 areas of full-thickness corneal perforation at the nasal and temporal CK-treated spots with iris plugging had developed. Treatment with oral acetazolamide and ascorbic acid, as well as 1% atropine sulfate, was given and the eye was patched. On April 4, 2005, the patient was taken to the operating room and the temporal iris plug was not removed to enable vascularization of the cornea. Glue was applied and a bandage contact lens placed. Serologic testing results revealed that the patient was positive for antinuclear antibody (1:320; positive, >160) and had antibodies to SS-A and SS-B (immune status ratio, 4.6 and 4.7, respectively; positive, >1.2). The diagnosis of Sjögren syndrome was made; the patient was treated systemically with celecoxib and prednisone.

The patient came to the Stanford University Eye Center, Stanford, California, for consultation on June 24, 2005. On examination, her visual acuity was counting fingers OD and 20/20 OS. Slitlamp examination revealed a large plug of glue over the right central cornea with an overlying bandage contact lens. The infranasal cornea appeared thinned and vascularized at the area of previous perforation. Sixteen CK spots were seen. Penetrating keratoplasty was recommended (Figure).

Comment. To our knowledge, this is the first reported case of corneal perforation in a patient who underwent CK having undiagnosed Sjögren syndrome. Sjögren syndrome, a chronic autoimmune inflammatory disease of the exocrine glands, including the lacrimal gland, predisposes to corneal stromal degradation and thinning, ulceration, and perforation as a result of diminished tear secretion, corneal epithelial breakdown, and enzymatic degradation of collagen by inflammatory cells. Corneal stromal degradation and thinning can be the initial sign of primary Sjögren syndrome.

In patients with chronic dry eye before CK, it is important to evaluate for Sjögren syndrome. Correct diagnosis before refractive surgery may identify patients who may not be appropriate candidates for CK and prevent unnecessary postoperative complications.

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Acute Optical Coherence Tomographic Findings in Cancer-Associated Retinopathy

Cancer-associated retinopathy (CAR) is a rare paraneoplastic condition characterized by painless subacute visual loss in the setting of a distant neoplastic process. The symptoms are often asymmetrical, initial fundus examination results can be normal, and in 50% of cases, CAR is seen before the primary tumor is diagnosed, making initial misdiagnosis common. We describe the acute optical coherence tomographic (OCT) findings in a case of CAR.

Report of a Case. A 58-year-old woman was first seen by us with 1 week of visual obscuration and photopsia. There was no relevant ocular, family, or medical history. She was systemically healthy. Visual acuity at first examination was 6/6 corrected OU and rapidly deteriorated to 6/9 OD and counting fingers OS over the course of 3 days. There was no relative afferent pupillary defect or color vision abnormality. The anterior segment was normal. Fundus examination revealed a few central macular drusen, healthy optic discs, and subtle generalized arteriolar narrowing (Figure 1). There was bilateral peripheral field loss on Goldmann perimetry. Fluorescein angiography and autofluorescence imaging results were normal. The photopic full-field electroretinogram was extinguished and scotopic responses were subnormal with markedly reduced amplitude and delayed implicit times. The OCT 3.0 (Carl Zeiss Meditec, Dublin, California) showed dramatic thinning of the retina with loss of the inner-highly reflective layer (Figure 2).

Figure 1. A, Fundus photographs showing a few central macular drusen, healthy optic discs, and subtle generalized arteriolar narrowing. The arrows indicate the orientation and location of the corresponding optical coherence tomographic scan. B, High-resolution cross-sectional optical coherence tomographic scans showing loss of the inner, highly reflective layer of the retina (more marked in the left eye).

Figure 2. Optical coherence tomographic macula thickness map demonstrating marked retinal thinning in both eyes. Six optical coherence tomographic scans were obtained in a radial spoke pattern centered on the fovea. The mean retinal thickness in each area was calculated automatically from multiple measurements and displayed numerically and geographically as a false-color topographic map in 9 standardized Early Treatment Diabetic Retinopathy Study areas with a central zone that was 1000 µm in diameter and 2 outer rings with diameters of 3000 µm and 6000 µm. (The normal reported retinal thickness on OCT 3.0 [Carl Zeiss Meditec, Dublin, California] is 212 µm [SD, 20 µm] in the central zone and 242 µm [SD, 32 µm] in the surrounding macula regions.)

Computed tomography of the chest, abdomen, and pelvis revealed a uterine mass. Endometrial biopsy led to the diagnosis of endometrial adenocar-