Microbial Keratitis Identified During Eye Bank Screen of Corneoscleral Tissue Harvested from Patients With Laser In Situ Keratomileusis History

Laser-assisted in situ keratomileusis (LASIK) has become a well-accepted and effective procedure for the treatment of a wide range of refractive errors. Microbial keratitis is a potential complication of LASIK with causative organisms, including gram-positive bacteria, atypical mycobacteria, fungi, and viral pathogens. Direct ocular trauma and exposure keratopathy in severely ill patients in intensive care units are risk factors for microbial keratitis. We describe the first cases, to our knowledge, of infectious keratitis in donor corneas harvested from cadavers with a past history of LASIK. Slitlamp, specular microscopy, and histopathologic findings are presented. These donors would likely have suffered vision loss if they had survived their illnesses. This article illustrates the importance of eye care by intensive care personnel managing patients with a past history of LASIK.

Report of Cases  Case 1. In December 2002, a healthy 22-year-old man underwent uncomplicated myopic LASIK in both eyes. Two days later, he was involved in a high-speed motor vehicle crash; he sustained diffuse brain injury. He was maintained on life support for 1 week and was evaluated for organ donation. During his hospitalization, the patient received tobramycin and dexamethasone drops. Death-to-preservation time for in situ corneal excision was 6 hours 48 minutes. The donor corneas were placed in Optisol-GS cornea storage media (Chiron Ophthalmics, Irvine, Calif) and sent to the New Mexico Lions Eye Bank (Albuquerque), using protocols established by the Eye Bank Association of America.

On slitlamp examination, the right donor cornea had an obvious LASIK flap with localized flap dehiscence and folds (Figure 1A). This cornea had evidence of an infiltrate with feathery edges (Figure 1B). The left donor cornea also had a poorly healed LASIK flap edge and a more extensive corneal infiltrate (Figure 2). Specular microscopy performed 4 days after death revealed central endothelial cell counts of 2008/mm² in the right cornea and 2159/mm² in the left cornea. Highly reflective particles were seen in both donor corneas when the specular microscope was focused in the region of the stroma corresponding to the LASIK interface.

The donor corneas were then placed in 10% neutral buffered formalin and processed for permanent sections. Sections were stained with hematoxylin-eosin, periodic acid–Schiff, Brown and Brenn, and Gomori methenamine silver. Histo-
logic examination of the right donor cornea revealed missing epithelium over the inferior lamellar interface wound with mild chronic nongranulomatous inflammatory cells in the anterior stroma. No incriminating organism was found. Histologic examination of the left eye revealed similar findings, with the noted difference of a moderate anterior stromal infiltrate. Additionally, the Gomori methenamine silver stain showed the presence of multiple yeast organisms on the affected corneal surface, with some invading the anterior stroma (Figures 3A and 3B).

Case 2. A 61-year-old man with a medical history remarkable for metastatic colon cancer had undergone LASIK in both eyes at an unknown date. He was placed in hospice care in October 2001. Ten days later, the patient died. The patient’s body was refrigerated for 2 hours postmortem. Death-to-preservation time for in situ corneal excision was 12.5 hours. The donor corneas were placed in Optimol-GS cornea storage media and sent to the New Mexico Lions Eye Bank, using established Eye Bank Association of America protocols.

Histologic examination of the right donor cornea revealed the presence of a LASIK lamellar flap with missing epithelium over the entire flap and the cornea inferior to it. The latter region of the cornea had an ulcer, inferior to the LASIK flap wound, with a moderate chronic nongranulomatous inflammatory cell infiltrate in the anterior stroma (Figure 5A). Brown and Brenn stain revealed a small focus of gram-positive cocci on the surface of the ulcer (Figure 5B).

Comment. These case reports represent the first description of infectious keratitis identified during eye bank screening of corneoscleral tissue from donors with LASIK history. Microbial keratitis is considered a rare complication following LASIK, varying from 0.1% to 1.2%.3-5 Although some of the reported infections after LASIK are interface infections, these cases seem to represent surface corneal ulceration. Laser-assisted in situ keratomileusis severs the branches of the trigeminal nerves that enter from the limbus centripetally to enervate the stroma and epithelium of the central cornea.6 Regeneration of corneal nerves usually occurs within 1 year after LASIK.7 However, 1 study reported that LASIK results in decreased corneal and conjunctival sensitivity even at 18 months after the procedure.8 This study also reported that the decreased ocular sensitivity is accompanied by a decrease in aqueous tear production, punctate epithelial erosions, and a decrease in tear fluorescein clearance probably due to a decreased
Blink rate.8 Intubated or sedated patients are at risk for lagophthalmos and exposure-related keratitis. The post-LASIK neurotrophic status of these patients may further predispose them to infectious keratitis.

The first case represents, to our knowledge, the earliest post-LASIK human histopathologic finding (9 days post-LASIK). The neurotrophic cornea would certainly be a risk factor in the immediate postoperative period. The first patient had evidence of probable traumatic displacement of the LASIK flaps, which may have contributed to the development of keratitis (Figures 1A and 3A). Late traumatic displacement of LASIK flaps occurring more than 2 years after the procedure, complicated by diffuse lamellar keratitis, has been reported.9 Late post-LASIK fungal keratitis related to trauma has also been reported.10 Based on the well-healed scar of the second case, the neurotrophic status of the cornea may predispose the cornea to infection even months after the LASIK procedure. Postmortem microbial contamination of the donor corneas is unlikely given the chronic inflammatory cell response documented by histopathologic examination.

These cases suggest that intensive care unit personnel add a refractive surgery query to the eye history taken from family members of trauma patients and other obtunded patients. Intubated and sedated patients who have had LASIK surgery require close monitoring for exposure keratopathy and prompt diagnosis of keratitis. These patients would probably benefit from aggressive lubrication and prophylactic antibiotic ointment.

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The authors have no relevant financial interest in this article.

This study was presented in part at the 41st annual meeting of the Eye Bank Association of America, Ponte Vedra Beach, Fl., June 22, 2002.

The study was supported in part by the Dedicated Health Research Funds of the University of New Mexico School of Medicine, Albuquerque.

We thank Tissue Banks International (Baltimore, Md) for coordinating the collection of donor corneas. We also thank Edward Otero, BA, and Chris Miguel-Nathan, BA, for their assistance with manuscript preparation.

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Pigmented Conjunctival Lesions as Initial Manifestation of Ochronosis

Alkaptonuria (ochronosis) is an inherited aminoacidopathy of the phenylalanine/tyrosine metabolism (Figure 1). Phenylalanine is an essential amino acid that is irreversibly hydroxylated to tyrosine by homogentistic acid (HGA) oxidase, which is found in the liver and kidneys. In alkaptonuria, the enzyme is absent, and HGA accumulates in collagenous tissues such as cartilage and tendon, especially in the ear, nose, cheeks, conjunctiva, cornea, and sclera. Although conjunctival involvement in ochronosis is rare, it should be considered in the differential diagnosis of pigmented lesions and deposits of the ocular sur-