

Are Post-SLT Keratitis and Central Toxic Keratopathy the Same Condition?

BY STEVEN J. DELL, MD

Central toxic keratopathy (CTK) is the name given by Somnez and Maloney to a rare condition seen after LASIK and PRK that results in central corneal opacification with tissue loss and a significant hyperopic shift.¹ Onset typically occurs on postoperative days 3 to 9, and the condition may last many months.¹ Others have described what appears to be the same condition but have given it different names.²⁻⁴

CTK is often preceded by diffuse lamellar keratitis (DLK),⁵ and some surgeons argue that CTK may simply be a variant of severe, grade 4 DLK. There is no clear evidence regarding the exact etiology of CTK, but it has been suggested that the laser's activation of various substances in the LASIK interface such as meibomian gland secretions, povidone-iodine, or talc may be involved. Of importance, the condition has also been reported in PRK patients where there is no flap interface. To my knowledge, CTK has not been reported in cases without laser application to the cornea. The typical course is significant central corneal opacification extending deeply into the stroma, with a hyperopic shift due to tissue loss, followed by gradual resolution. Corneal thickness eventually increases toward the pre-CTK level due to epithelial hypertrophy. Some experts state that the condition is noninflammatory and does not respond to topical steroids.¹ There is controversy over how these eyes should be managed, as one might expect for a rare condition with an unknown etiology.

The case of post-SLT keratitis that Dr. Marquis presents bears many similarities with CTK—namely, laser application to the anterior segment with subsequent central corneal opacity, loss of corneal stromal tissue, and hyperopic shift,

followed by gradual partial resolution. As Dr. Marquis points out, the laser application in SLT does not occur to the cornea itself. Any effect on the cornea from post-SLT keratitis seems to be an indirect one. Similarly, in the case of CTK, the stromal involvement is not confined to the level of the cornea receiving laser application; rather, it extends throughout much of the thickness of the corneal stroma. It makes sense that the loss of tissue from both of these entities could be due to something similar to the photoactivation of collagenase. In CTK, the degree of irregular astigmatism caused by the tissue loss is typically greater than that seen in this case. This difference may be due to the absence of a corneal flap, or perhaps it is simply a question of the vastly greater magnitude of laser energy delivered to the eye by the excimer laser.

Although both conditions seem to be quite rare, perhaps future study will help us determine whether they are related, the same, or distinct entities.

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