



Central Toxic Keratopathy after Photorefractive Keratectomy: A Four-Year Case Report

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Abstract

Introduction: Central toxic keratopathy (CTK) is an uncommon, non-inflammatory opacification of the central cornea after excimer laser refractive surgery, usually within the first postoperative week. The cornea loses central stromal tissue and flattens, and the eye becomes hyperopic. Why it happens is not known; both surgical contaminants and an immune-mediated process have been proposed.

Methods: We describe a 26-year-old male physician who underwent bilateral photorefractive keratectomy (PRK) for refractive correction before applying for military service.

Results: One week after surgery he had central corneal haze in both eyes, with topographic flattening and a large hyperopic astigmatic shift that was worse in the right eye. He was treated medically: the topical corticosteroid was intensified and topical cyclosporine was added. Over 16 months of follow-up the left eye recovered completely, while the right eye recovered partially and settled at an uncorrected distance visual acuity of 0.8 with faint residual paracentral haze. At four years the corneas remained stable and the visual result unchanged, without further surgical intervention.

Conclusion: CTK is rare, but the visual loss in the first week after PRK can be severe. Recognizing it early matters, because the alternatives are an unnecessary antimicrobial course or an unnecessary operation. With close follow-up the long-term outlook is good, and patients deserve to hear about this complication before they consent to refractive surgery.

Keywords: Central toxic keratopathy, Photorefractive keratectomy, Corneal opacity, Hyperopia, Postoperative complications

Introduction

Central toxic keratopathy (CTK) is an uncommon complication of excimer laser refractive surgery, reported after laser-assisted in situ keratomileusis (LASIK) and after photorefractive keratectomy (PRK).^{1,2} It was first taken for an aggressive form of diffuse lamellar keratitis. It is now treated as a separate entity, and it declares itself early, usually between the third and the ninth postoperative day.^{2,3} The picture is a dense central or paracentral opacity with loss of stromal tissue, corneal striae, and a hyperopic shift produced by flattening of the central cornea.⁴ What causes it is still unknown. Contamination from surgical gloves, meibomian gland secretions, antiseptics such as povidone-iodine, and excessive laser energy during ablation have all been proposed, none of them proven.^{5,6}

The cornea in CTK thins and flattens without an inflammatory infiltrate, and the current explanation is keratocyte apoptosis with enzymatic degradation of the stromal matrix.⁷ The loss of curvature and thickness can be considerable, sometimes more than 100 micrometers.³ Patients report blurred vision, sensitivity to light, halos, or non-specific visual discomfort. No laboratory test or imaging study confirms the condition, so the diagnosis rests

on the clinical picture: a non-inflammatory central corneal opacity, measurable stromal thinning, and a hyperopic refractive shift.^{6,8}

Treatment is conservative. In most eyes the opacity clears over several months and the refraction settles.^{2,6} Topical corticosteroids, cyclosporine eye drops, and preservative-free lubricants are commonly used to keep the patient comfortable, although none of them has been shown to alter the course of the disease.^{4,9} Whether to lift the flap and irrigate the interface is still argued over, and mechanical debridement is generally avoided, because handling an already thinned stroma risks further tissue loss.

CTK is infrequent, but it matters out of proportion to its incidence. It mimics other postoperative conditions and it can leave a patient with months of poor vision.^{2,10} Long-term reports after PRK in particular remain scarce. We describe a patient followed for four years with serial corneal tomography, which documents how the cornea remodels once the acute phase has passed.

Case Report

Case Presentation

A 26-year-old male physician was seen for refractive

correction before applying for military service. Uncorrected distance visual acuity (UCVA) was 0.1 in each eye. Manifest refraction was -1.50 diopters sphere with -1.00 diopters cylinder at 92° in the right eye and -1.50 diopters sphere with -0.50 diopters cylinder at 73° in the left eye, both correcting to 1.0. Corneal curvature was 41.75 diopters bilaterally. The anterior and posterior segments were unremarkable, and corneal topography confirmed normal morphology in both eyes (Figure 1).

Management

Both eyes were treated in the same operative session on 4 November 2021. Ablation was performed with a WaveLight EX500 argon fluoride excimer laser (Alcon Laboratories, Inc., 6201 South Freeway, Fort Worth, TX 76134, USA) at a wavelength of 193 nm and a pulse repetition rate of 500 Hz, using the topography-guided Contoura Vision treatment profile, which tailors the ablation to the patient's own corneal surface maps. The optical zone was 6.5 mm with a 1.5-mm transition zone. Ablation speed was approximately 1.4 seconds per diopter, and ablation depth was approximately 15 µm per diopter of myopic correction.

Eye position was tracked at 1050 Hz by active infrared, with a latency of about 2 milliseconds. The beam path was sealed and nitrogen-flushed, and energy consistency was checked repeatedly through the treatment (Table 1). The operation gave no trouble, and a soft bandage contact lens was placed on each eye. He went home on fluorometholone, moxifloxacin, and preservative-free artificial tears.

Outcome

On 10 November 2021, the seventh postoperative day, the epithelium had healed and the bandage contact lenses came out. At the slit lamp the right cornea carried a dense central opacity, and the left carried a similar opacity sitting slightly off center. His vision had dropped sharply. The right eye could only count fingers at 4 meters, and the left read 0.3. Neither eye would give an autorefractive reading through the opacity.

Two weeks after surgery, on 17 November 2021, topography showed both corneas flattened and thinned. Keratometry (K) in the right eye read 38.0 D and 26.0 D; in the left, 39.0 D and 37.0 D. An autorefractive could now be obtained, and it showed a large hyperopic shift with

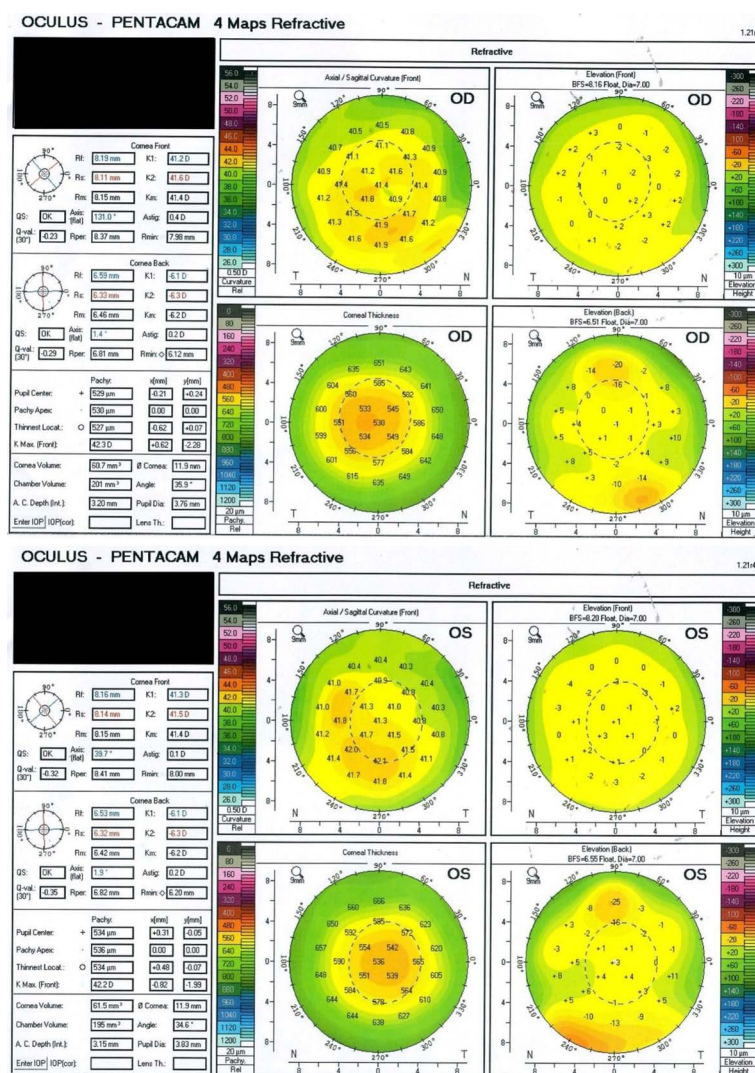


Figure 1. Preoperative Pentacam tomography of the right eye (OD) and left eye (OS). Both corneas show normal morphology with a curvature of 41.75 D bilaterally

Table 1. Laser parameters for the topography-guided photorefractive keratectomy procedure

Parameter	Value
Type of laser	Argon fluoride excimer, WaveLight EX500 (Alcon Laboratories, Inc., Fort Worth, TX, USA)
Wavelength	193 nm
Emission mode	Pulsed
Pulse repetition rate	500 Hz
Ablation speed	~1.4 s per diopter (6.5-mm optical zone)
Treatment profile	Topography-guided (Contoura Vision)
Delivery system	Nitrogen-flushed sealed beam path with repeated energy-consistency verification
Eye tracking	1050 Hz active infrared tracker, ~2 ms latency
Pulse duration	6 ± 2 ns
Fluence (energy density)	Approximately 400 mJ/cm ² at the cornea
Spot diameter at the cornea	0.68 mm FWHM (Gaussian flying spot); ~0.95 mm effective ablation diameter
Optical zone and transition zone	6.5 mm and 1.5 mm
Ablation depth	Approximately 15 µm per diopter of myopic correction

high astigmatism: +13.00 -7.00 × 160 on the right and +8.00 -3.00 × 150 on the left. A myopic young man had been made hyperopic in two weeks. There was still no sign of infection. Flattening, thinning, and a hyperopic shift in a quiet eye: the pattern belonged to CTK (Figure 2).

At this point we increased fluorometholone to hourly dosing and reviewed him every two weeks. Moxifloxacin was continued until two weeks after the bandage contact lenses had been removed and was then stopped, as there had been no sign of infection at any visit. Four weeks after surgery topical cyclosporine was added twice daily, to settle the inflammatory component and to support remodeling of the stroma.

Follow-up

Over the following months the haze regressed gradually and keratometry improved, more quickly in the left eye than in the right.

At 16 months postoperatively (16 February 2023), UCVA had improved to 0.8 in the right eye and 1.0 in the left eye. Slit lamp examination of the right eye showed a mild, faint, off-center corneal opacity; the left cornea was clear. K-readings at that visit were 40.5 D and 38.75 D for the right eye and 39.75 D and 39.5 D for the left eye.

At the most recent evaluation, four years postoperatively (11 June 2025), Pentacam tomography showed further improvement in corneal curvature and thickness bilaterally. Only a faint paracentral opacity remained in the right eye, and the left eye was completely clear. Visual acuity was stable at 0.8 in the right eye and 1.0 in the left eye, confirming a long-term stable recovery (Figure 3).

Discussion

CTK appears early after excimer laser refractive procedures, after PRK as well as LASIK, and it is not an inflammatory keratitis.^{1,2} Our patient showed the classical triad of central

corneal opacification, stromal thinning, and hyperopic shift within the first postoperative week, which fits the reported window of the third to ninth day.^{2,3}

What makes the case instructive is that nothing went wrong in the operating room. There were no epithelial defects, no intraoperative difficulties, and no signs of postoperative inflammation or infection. The epithelium had closed by day 7 and there was no anterior chamber reaction, which supported a diagnosis of CTK rather than infectious keratitis or diffuse lamellar keratitis. Findings of this kind reinforce the theory that CTK arises from biochemical or immune-mediated mechanisms rather than mechanical trauma.⁶

The diagnosis rested on the deliberate exclusion of the principal differentials. Infectious keratitis was unlikely, given the absence of pain, conjunctival injection, an epithelial defect, a stromal infiltrate, hypopyon, or anterior chamber reaction, and a clinical course inconsistent with active infection. Diffuse lamellar keratitis was excluded because a surface ablation leaves no lamellar interface and no interface inflammatory cells were seen; the stromal thinning and the hyperopic shift observed here are not features of that condition.¹¹ Ordinary post-PRK inflammatory haze was separated by timing and by the direction of the refractive change, since conventional haze produces myopic regression with preserved or increased curvature, whereas this patient flattened centrally, lost stroma, and became hyperopic. Taken with the non-inflammatory picture, the tomographic pattern, and the self-limiting course, the diagnosis was CTK.^{2,6}

The hyperopic shift was profound, +13.00 diopters in the right eye, and it went with marked corneal flattening and thinning on topography. That combination is what keratocyte apoptosis and stromal matrix degradation would be expected to produce.⁷ Conventional post-PRK haze tends to drive the refraction the other way, toward myopic regression; CTK flattens the center and leaves the eye hyperopic.^{3,5}

Treatment stayed medical. The corticosteroid ran on because haze was overlapping the picture, and cyclosporine went in once the antibiotic came out, to quiet the surface and temper the healing response. After femtosecond LASIK there is an argument for lifting the flap and irrigating the interface, and better visual and anatomic results have been reported in some of those eyes.⁶ A surface ablation offers nothing to lift. Debridement we rejected on principle: in a cornea that has already given up stroma, taking more tissue is a poor way to treat tissue loss.

The two eyes did not recover at the same pace. The left was back to normal at 16 months; the right was still at 0.8 uncorrected, with a little paracentral haze left and -1.75 D of irregular astigmatism. Four years on, both corneas had remodeled, and the vision had not shifted again since. A course of 12 to 18 months is what others report.^{2,12}

Reported incidence runs from 0.05% to 0.77% of refractive procedures.⁵ We could find no culprit in this one. No povidone-iodine had touched the surface, the gloves carried no powder, the laser energy was nothing

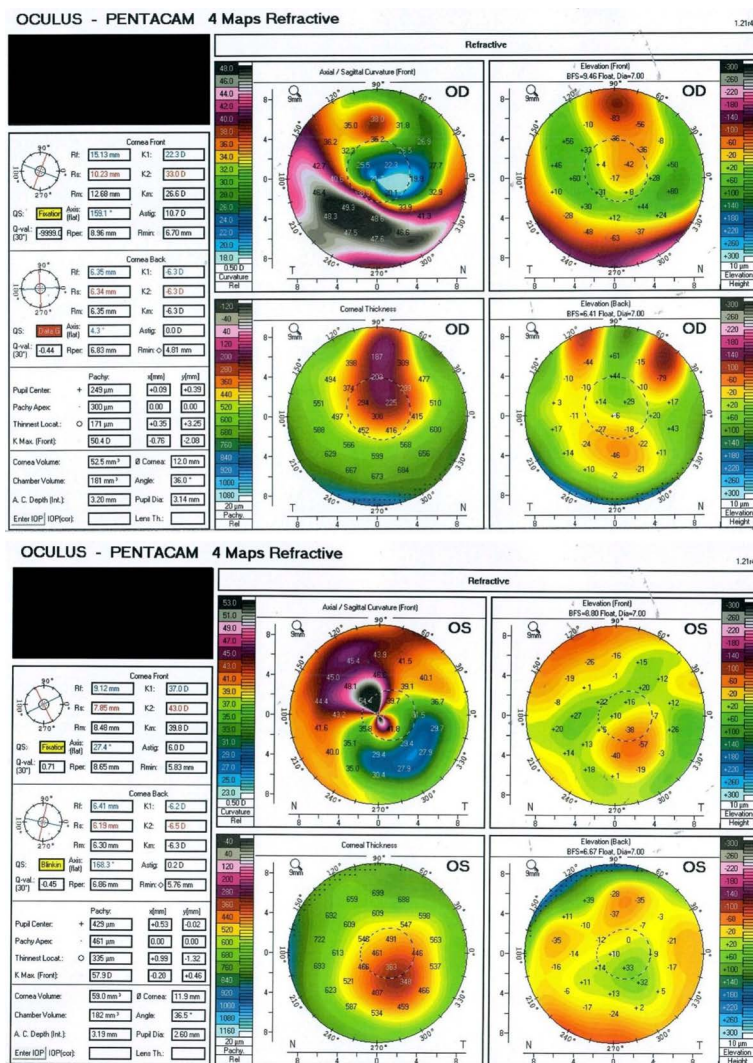


Figure 2. Pentacam tomography of the right eye (OD) and left eye (OS) during the acute phase, two weeks after surgery. Both eyes show central corneal flattening, stromal thinning, and a marked hyperopic shift, more pronounced in the right eye, consistent with central toxic keratopathy

out of the ordinary, and the eye developed CTK all the same.⁹ The diagnosis stayed clinical, as it has to: the triad, and an inflammatory keratitis ruled out. Anterior segment OCT and serial topography help document the thinning and flattening, but neither confirms the condition on its own.¹⁰ Rather than attributing this case to any single mechanism proposed in the literature, we emphasize that CTK developed after an entirely uneventful PRK with no identifiable intraoperative trigger. None of the previously described etiologies can be invoked here, which indicates that the condition can arise independently of recognized risk factors.

This is a single patient, and a single patient cannot establish why CTK occurred or how often it behaves this way. The diagnosis rested on the clinical and tomographic picture, without histological confirmation, as it must in a condition that has no confirmatory test.

Early recognition is what protects the patient here, because the reflex when a cornea turns white in the first postoperative week is to treat for infection or to intervene surgically, and in CTK both are wrong. Recovery is slow, but with support and without interference the outcome is good. That is worth saying to the patient before the

operation rather than after it: the complication is rare, it can take a year or more of poor vision to resolve, and it usually does resolve. When a refractive result departs from what was planned, follow the eye until the departure is explained. Four years of serial Pentacam tomography are, as far as we know, among the longest reported for CTK after PRK, and they show the remodeling holding.

Conclusion

A healthy young man came for a routine PRK and lost most of his sight within the week. Centrally the cornea was hazy, the stroma had thinned, the refraction had swung deep into hyperopia, and the eye stayed quiet throughout: no inflammation, no infection. Topical corticosteroids, cyclosporine, close review, and patience were the whole of the treatment. His sight came back slowly, and it stayed. CTK settles on its own. What it asks of the surgeon is recognition, an honest explanation to the patient, and the discipline to leave the cornea alone while it repairs itself.

Authors' Contribution

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Data Curation: Abdallah M. Aljazzazi

Formal Analysis: Raed Shatnawi, Abdallah M. Aljazzazi

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