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Corneal Neuralgia after LASIK

Christos Theophanous*, Deborah S. Jacobs†, and Pedram Hamrah†

ABSTRACT

Purpose. To illustrate that corneal neuralgia may be the basis for refractory dry eye syndrome after laser-assisted *in situ* keratomileusis (LASIK).

Methods. The methodology used is that of a retrospective medical record review of a small case series.

Results. Three male patients, aged 30 to 48 years, referred in 2012 for dry eye syndrome refractory to treatment within 1 year of LASIK or LASIK enhancement are reported. Each patient gave history of eye pain, light sensitivity, and difficulty with visual activities beginning within 2 months of LASIK or LASIK enhancement. Best-corrected visual acuity was 20/15 or 20/20 in each of the six eyes. Tear-centered models and metrics did not explain persistent symptoms, which was consistent with inadequate response to standard dry eye treatments used before referral and reported here. *In vivo* confocal microscopy was abnormal at presentation in each case and was followed over time. Treatments undertaken subsequent to referral included autologous serum tears (three cases), PROSE (Prosthetic Replacement of the Ocular Surface Ecosystem) treatment (two cases), and systemic agents for pain, anxiety, or depression (three cases). By the end of 2013, at a mean of 23 months after LASIK or LASIK enhancement, symptoms improved in all three patients.

Conclusions. Patients with persistent dry eye symptoms out of proportion to clinical signs after LASIK have a syndrome that may best be classified as corneal neuralgia. *In vivo* confocal microscopy can be informative as to the neuropathic basis of this condition. In keeping with current understanding of complex regional pain syndrome, early multimodal treatment directed toward reducing peripheral nociceptive signaling is warranted to avoid subsequent centralization and persistence of pain. Distinguishing this syndrome from typical post-LASIK dry eye remains a challenge.

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Key Words: dry eye, dry eye syndrome, LASIK, corneal neuralgia, neuropathic pain

Although laser-assisted *in situ* keratomileusis (LASIK) is a common and highly effective surgical treatment of myopia,[1–3] dry eye symptoms are reported by nearly half of patients after the procedure.[4–6] Several pathophysiological mechanisms have been suggested for these symptoms, mostly relating to impaired tear production, distribution, and stability.[7] In particular, transection of sensory nerves during the lamellar cut of the procedure is thought to impair the blink reflex and alter corneal sensation.[8–10] Alterations in corneal topography causing uneven tear distribution[8] and postoperative changes in tear composition owing to goblet cell damage[11,12] are also thought to be contributing factors. Typically, these symptoms peak between 1 week and 3 months of surgery[10,13–15] and resolve within 6 months.[10,15] This time course maps closely with the recovery of central corneal sensation[13,15] and a period of rapid corneal nerve density recovery between 6 months and 1 year after surgery.[16,17]

This tear-centered model fails to explain the full spectrum of LASIK-associated dry eye disease. Studies have confirmed a poor correlation between symptoms and clinical signs, such as punctate epithelial keratopathy on corneal staining and abnormal Schirmer tests in LASIK patients,[9,18] and in dry eye patients in general.[19] Patients with normal findings may report severe symptoms,[18] whereas patients with visible epithelial changes after LASIK often do not report discomfort.[4]

An explanation for these inconsistencies is that certain cases of intractable LASIK-associated dry eye disease represent a pathologic process of the nervous system rather than a problem of tear dynamics.[20,21] Neuralgia is defined as pain in the distribution of a nerve or set of nerves with concurrent signs of nerve damage.[22] Intractable dry eye syndrome after LASIK might be considered a variant of complex regional pain syndrome (CRPS) type II[23] and may be better described as post-LASIK neuralgia. A neuropathic

*BA
†MD
Keck School of Medicine, University of Southern California, Los Angeles, California (CT); Boston Foundation for Sight, Needham, Massachusetts (DSJ); and Cornea & Refractive Surgery Service, Massachusetts Eye and Ear, Department of Ophthalmology, Harvard Medical School, Boston, Massachusetts (DSJ, PH).

basis for persistent, intractable, and disabling symptoms out of proportion to clinical signs in a subset of patients after LASIK is plausible given the known nerve injury from the procedure. Because the corneal subbasal nerve plexus (SNP) is not visualized with the slit lamp, *in vivo* confocal microscopy (IVCM) can be useful in identifying signs of nerve damage. A recent report on correlation of symptoms and signs of dry eye disease in patients with meibomian gland disease found that there was only correlation in patients without abnormalities of the SNP.[24] Injury to the SNP of the type that occurs in LASIK may disrupt the correlation between signs and symptoms.

We report the presentation, treatment, and course of disease of three patients referred in 2012 for dry eye syndrome refractory to treatment within 1 year of LASIK or LASIK enhancement. We explain that a subset of patients with severe and persistent ocular symptoms after LASIK may be better understood and treated as corneal neuralgia.

METHODS

Our methodology is that of a retrospective medical record review. This report was approved as exempt from review by the Institutional Review Board/Ethics Committee of Massachusetts Eye and Ear (MEE) and by the New England Institutional Review Board; this study complied with the Health Insurance Portability and Accountability Act and adhered to the tenets of the Declaration of Helsinki. Inclusion criteria for this study included LASIK performed within 1 year of initial consultation in 2012 and persistent ocular symptoms that were not alleviated with conventional dry eye therapy. Data extracted from the medical records included patient demographics, medical history, ocular history, previous treatment, Ocular Surface Disease Index (OSDI) scores,[25] best-corrected visual acuity, clinical findings including vital dye staining and tear metrics, and IVCM imaging using HRT3/Rostock Cornea Module (Heidelberg Engineering GmbH, Heidelberg, Germany), as well as clinical course through December 2013.

RESULTS

The three patients included in this report are male with ages between 30 and 48 years. Demographic and clinical information is summarized in Table 1. Detailed case histories with a mean follow-up of 23 months after LASIK or LASIK enhancement are as follows.

Case 1

A 42-year-old man with a history of hypercholesterolemia underwent LASIK OU. He presented to Boston Foundation for Sight (BFS) 10 months later with a detailed timeline of his own clinical course as follows: Immediately after the procedure, he had a burning sensation in both eyes. Within several weeks, he had noted symptoms of light sensitivity, halo effect, and extreme dry eye. He was initially treated with topical cyclosporine (Restasis, Allergan, Irvine, CA) twice per day, preservative-free lubricating drops throughout the day, and punctal occlusion. These measures substantially reduced his symptoms in the left eye by 2 to 3 months after LASIK; symptoms in the right eye persisted with notation of continued achy, sore pain with tearing that worsened throughout the day, sensitivity to sunlight, blurriness, and dryness. His symptoms in the right eye were subsequently treated with warm compresses twice per day, moisture chamber goggles during the day, ointment thrice per day and nightly, oral cetirizine, trial of bandage soft lenses, and a daytime eye patch, offering limited relief. Doxycycline 100 mg by mouth daily was also prescribed for mild blepharitis. During this treatment period, the patient developed depression and ultimately was hospitalized for suicidal ideation, which he reported was driven by his eye pain.

At presentation to BFS, his best-corrected visual acuity was 20/20 OD and 20/15 OS. His OSDI score was 54.5 (average subgroup scores: A = 1.4, B = 3.0, C = 2.7). Oxford scores[26] were 0 in each eye. Current and prior systemic and local therapies are listed in Table 1. The patient reported relief of symptoms with trial of a Prosthetic Replacement of the Ocular Surface Ecosystem (PROSE) device (BFS, Needham, MA) in the right eye only; hence, the decision was made to proceed with PROSE treatment for that eye alone, with the suggestion that he continues all other modes of treatment of his symptoms. A trial of autologous serum tears (AST) was suggested and consultation at MEE for corneal imaging was recommended.

He was evaluated at MEE 2 weeks later, 4 days after initiation of AST four times per day by his primary eye care provider. Examination was significant for mild superficial punctate keratitis (SPK) OD and large central and inferior SPK OS. Meibomian gland dysfunction OU was noted. Tear breakup time (TBUT) was 2 seconds OU, and tear osmolarity was 296 mOsm/L OD and 289 mOsm/L OS. Schirmer test results with anesthesia were 6 mm OD and 10 mm OS. No pain score was obtained, but he reported total resolution with in-office administration of topical proparacaine. *In vivo* confocal microscopy of his right eye demonstrated significant diminishment of his corneal nerves with very few nerves present, increased tortuosity, beading, and increased presence of immune dendritic cells. *In vivo* confocal microscopy of the left eye showed no nerves in the central cornea and increased presence of immune dendritic cells (Fig. 1). Increased frequency to eight times per day and an increased concentration of AST were prescribed, and loteprednol one drop daily was added to his regimen to suppress inflammation.

The patient returned for follow-up 2 months later, having been hospitalized again for depression. There had been addition and discontinuation of systemic medications by his other physicians, including introduction of gabapentin and carbamazepine. He was not using loteprednol. Administration of topical proparacaine at this visit significantly diminished but did not eliminate his pain. He was advised to resume suppressive loteprednol. At his next visit 2 months later, he had discontinued wearing the PROSE device and was continuing with AST, and his systemic regimen had been further adjusted. He reported that his pain was 3 of 10, having improved from what he reported retrospectively as 7 of 10. There was evidence of SNP regeneration on IVCM OU.

Eight months from initial consultation, the patient returned for a scheduled 3-month follow-up reporting flare-ups of pain as high as 8 of 10 from a baseline of 4 of 10 with stinging, tearing,

TABLE 1.

Patient demographics and clinical features

Case	Age, y/sex	Medical history before LASIK	Visual acuity	OSDI (A, B, C)*	Vital dye staining (Oxford scale)†	Tear metrics (OD, OS) (mo after surgery)	Prescribed ophthalmologic treatments	Systemic medications at presentation	Additional systemic medications during course
1	42/M	Hypercholesterolemia	OD: 20/20 OS: 20/15	54.5 (1.4, 3.0, 2.7)	OD: 0 OS: 0	TBUT, s: 2, 2 (11) 5, 5 (21) Schirmer, mm: 6, 10 (11) Tear osmolarity, mOsm/L: 296, 289 (11)	Lubricant drops Lubricant ointment Punctal occlusion Moisture goggles Topical antibiotics Topical steroids Topical cyclosporin Bandage lenses Autologous serum‡ PROSE treatment‡	Doxycycline Cetirizine Atorvastatin Aspirin Dexlansoprazole Mirtazapine Risperidone Clonazepam Buproprion Desvenlafaxine Propanolol	Gabapentin§ Carbamazepine§ Eszopiclone§ Tapentadol§ Vilazodone§ Tramadol Oxycodone
2	48/M	Anxiety Allergic rhinitis Gastroesophageal reflux disease	OD: 20/15 OS: 20/15	47.7 (1.2, 3.0, 1.5)	OD: 0 OS: 0	TBUT, s: 3, 3 (8) 8, 8 (19) 4.5, 4.5 (26)	Lubricant drops Lubricant ointment Lacriserts Punctal occlusion Topical steroids Topical cyclosporin Autologous serum‡ PROSE treatment‡	Atorvastatin Aspirin Dexlansoprazole Duloxetine	Escitalopram (replaced Duloxetine) Esomeprazole
3	30/M	(None)	OD: 20/15 OS: 20/15	39.6 (1.8, 2.5, 0)	OD: 1+ OS: 0	TBUT, s: 6.5, 6.5 (14) 5.5, 5.5 (17) Schirmer, s: 7, 10 (5) 6, 7 (9)	Lubricant drops Punctal occlusion Topical steroids Topical cyclosporin Autologous serum‡	(None)	Gabapentin (taken for 1 mo)

*Average response (0 to 4) for each OSDI question subgroup (A, Symptoms; B, Function; C, Environmental Triggers).

†At time of BFS consult.

‡Treatment prescribed after initial consultation.

§Treatment initiated after hospitalization for depression with suicidal ideation.

light sensitivity, blurriness, and pressure OU. He reported being bedbound much of the day because of pain. He had started loteprednol twice per day, which caused mild improvement in his symptoms. He required oral narcotics for breakthrough pain reported as 8 of 10. The situation was similar 3 months later, at which time no SPK was observed OU and TBUT was 5 seconds OU. The patient was advised to continue AST, increase flaxseed oil to 3 tbsp/d, and start hot compresses and lid massage. During the next 3-month interval, he was treated elsewhere with meibomian gland probing and punctal cautery, after which he reported improvement of pain. During this period, the patient remained under the care of ophthalmologists who treated his ocular surface disease, as well as other clinicians who managed his ongoing systemic medical treatment of pain and depression. At the next 3-month follow-up visit and the end of the period reported in this study (27 months after surgery), the patient had returned from disability leave to employment and reported having 0 (of 10) pain in the morning, 1 to 2 (of 10) pain when not working, and 4 to 5 (of 10) pain when working.

Case 2

A 48-year-old man with history of anxiety, allergic rhinitis, and gastroesophageal reflux disease underwent LASIK OU with subsequent enhancement procedures 6 months and 9 months later (OS and OD, respectively). Seven months after his last enhancement procedure, he was referred to BFS for consideration of PROSE treatment of dry eye syndrome after LASIK. The patient reported that he began to experience ocular pain and foreign body sensation 1 month after the second enhancement procedure, worse OS. He also described shooting pain when watching TV, achiness, burning, light sensitivity, and difficulty sleeping because of eye pain. He had been initially treated with preservative-free lubricating drops eight times per day, Restasis twice per day, prednisone 1% drops four times per day, hydroxypropyl cellulose ophthalmic insert, punctal occlusion, fish oil/flaxseed oil 1000 mg/845 mg twice per day, and ointment nightly with limited success.

At consultation, he reported continued light sensitivity, painful and sore eyes, and discomfort with sustained visual activity. The

patient also reported a recent flare-up of anxiety and depression symptoms. Best-corrected visual acuity was 20/15 OU. Ocular Surface Disease Index score was 47.7 (average subgroup scores: A = 1.2, B = 3.0, C = 1.5). Vital dye staining was classified as an Oxford score of 0 OU. There was resolution of symptoms OU with trial of PROSE devices; thus, PROSE treatment was undertaken. He was referred to MEE for consideration of treatment with AST and for corneal imaging and was seen 1 month later. At this visit, he reported that his pain symptoms were 7 of 10 and improved to 2 of 10 with administration of topical proparacaine.

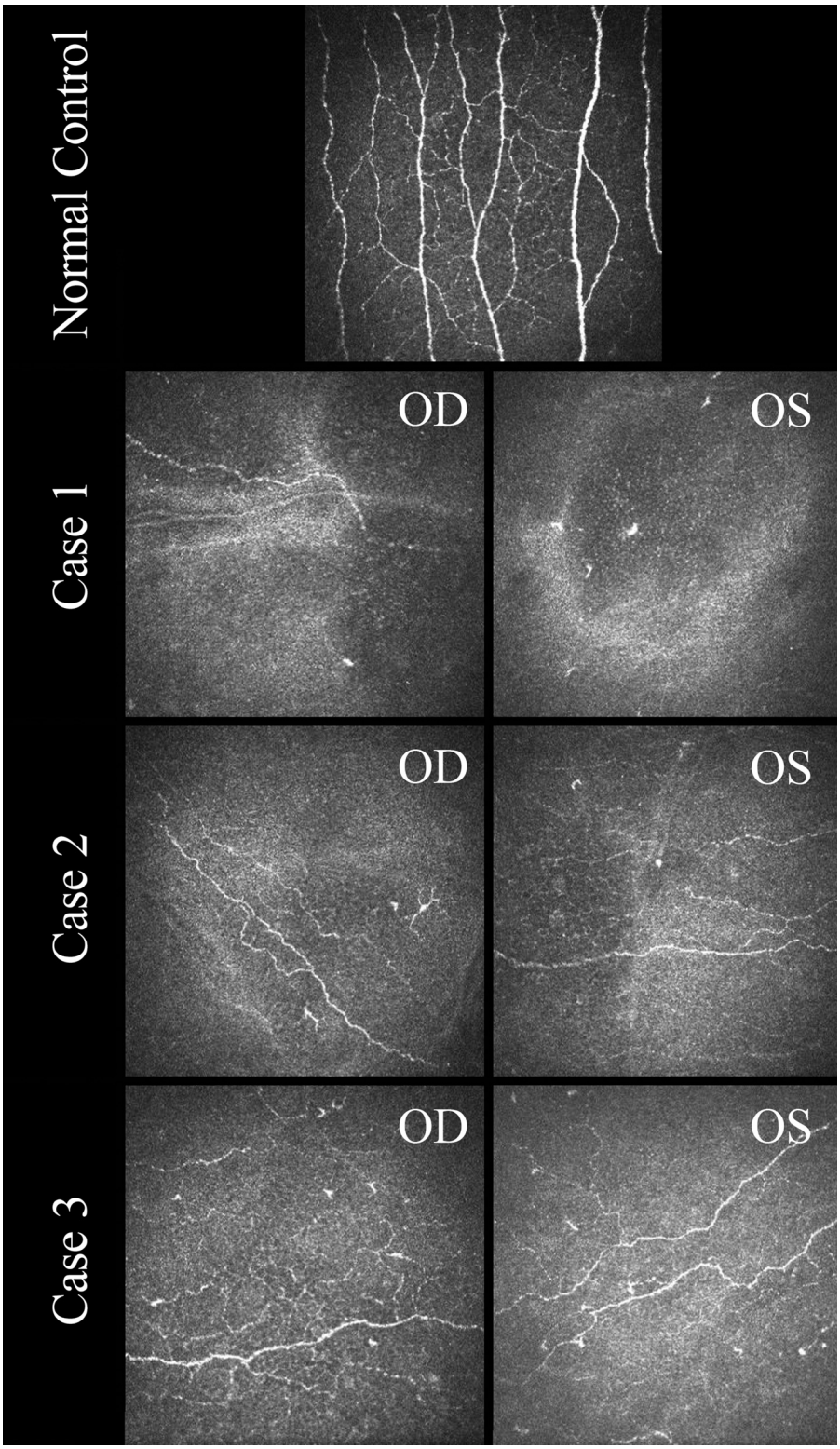

FIGURE 1.

In vivo confocal microscopy images showing corneal SNP taken at the time of initial MEE consultation. An image from a normal control is provided for reference. Specific findings are detailed in the report of each case.

In vivo confocal microscopy revealed significant diminishment of his corneal nerves, high tortuosity of subbasal corneal nerves, beading, and increased presence of immune dendritic cells in both eyes (Fig. 1). Increased flaxseed oil was recommended and AST eight times per day was added to his regimen, and fluorometholone eye drops were tapered. When he returned 2 months later, he reported wearing PROSE devices in the early part of the day and administering serum tears later in the day. He reported that his baseline symptoms were 4 of 10 and improved to 2 of 10 with administration of proparacaine. He returned for subsequent evaluation 2 months later, reporting that he had discontinued the PROSE devices because he was feeling better. He rated his pain that day as 6 of 10 improving to 2 of 10 with proparacaine. A tapering course of loteprednol was added to his regimen. He returned 1 month later, reporting his general baseline as 4 of 10 but his symptoms with proparacaine trial went from 2 of 10 to 0 of 10.

Six months later, 12 months after initial consultation, the patient reported 1 of 10 pain OS only that improved to 0 of 10 with proparacaine challenge. Tear breakup time was 8 seconds OU. Examination showed 1+ inferior SPK OS. *In vivo* confocal microscopy showed increased corneal nerves with reduced inflammatory cells OS. The situation was similar at his next 3-month follow-up on the same medication regimen. Four months later, at the end of the study period (26 months after last surgery), the patient's pain was still 1 of 10 OS with no flare-ups. Tear breakup time was 4.5 seconds OU. Examination showed trace superficial punctate epithelial erosions inferiorly OU. *In vivo* confocal microscopy showed increased and more normal-appearing nerves OU, with no inflammatory cells. At that time, the patient was instructed to reduce AST to four times per day and to discontinue topical methylprednisone, which had been substituted for loteprednol.

Case 3

A 30-year-old man was referred by a cornea specialist for consideration of PROSE treatment. His symptoms were eyestrain worse with reading and watching TV, light sensitivity, and morning dryness that started 2 months after LASIK that had been performed OU 7 months previously by another cornea specialist. He was initially treated by the LASIK surgeon with topical prednisolone and lubricant drops, which, by his own report, ameliorated his symptoms at first, but the effect waned. Schirmer tests with anesthesia performed by the referring doctor were 7 mm OD and 10 mm OS with slit lamp revealing "dry patches." Initial evaluation at BFS revealed uncorrected visual acuity of 20/15 in each eye. His OSDI score was 39.6 (average subgroup scores: A = 1.8, B = 2.5, C = 0). Corneal fluorescein staining was Oxford 1 OD and 0 OS. Soft lenses and PROSE devices were trialed on each eye; neither lens type was comfortable or reduced light sensitivity symptoms; hence, this path toward reduction of symptoms was not pursued. Of note, he also reported a generalized somatic hypersensitivity disorder with "trigger points." He was referred to MEE for confocal study of corneal nerves and consideration of AST, and it was also suggested that he seek evaluation by a pain specialist. A pain specialist prescribed gabapentin 300 mg daily, but the patient elected to discontinue after 1 month because of a lack of symptom relief.

At initial MEE evaluation 3 months later, he reported persistent symptoms of eyestrain, achiness, and light sensitivity for which lubricant drops provided no relief. Schirmer test with anesthesia showed 6 mm of wetting OD and 7 mm OS. Examination showed trace SPK OD and 2+ SPK OS. *In vivo* confocal microscopy showed significant diminishment of his corneal nerves, beading, and increased presence of immune dendritic cells in both eyes (Fig. 1). The patient was treated with pulse dosing of loteprednol, followed by low maintenance dose of loteprednol, and flaxseed oil 2000 mg/d. Two weeks later, the patient reported limited change in symptoms. Trace SPK was observed OU. The patient was started on AST eight times per day.

Four months later, the patient reported improvement in light sensitivity symptoms, which he rated as 5 of 10, but still reported difficulty with harsh lighting and watching TV. He reported eye fatigue and occasional pain upon awakening. Tear breakup time was 6.5 seconds OU. Examination revealed trace SPK OU. *In vivo* confocal microscopy showed increased nerve density and reduced inflammatory cells OU. The patient was instructed to continue his current regimen but to taper loteprednol to twice per week. Three months later, at the end of the study period (17 months after surgery), the patient reported improved symptoms, with 0 of 10 pain, mild grittiness and dryness, and intermittent light sensitivity. Tear breakup time was 5.5 seconds OU. Examination showed trace SPK OD and 1 to 2+ SPK OS. *In vivo* confocal microscopy showed increased nerve density with increased inflammatory cells OD. The patient was instructed to continue loteprednol twice per week, AST eight times per day, and flaxseed oil 2 tbsp/d.

DISCUSSION

Although dry eye syndrome is common after LASIK, the patients in this study were referred for dry eye syndrome that was persistent and unresponsive to standard therapy. In each case, the clinical signs of dry eye and tear metrics were inconsistent with the patient's level of discomfort. Each patient reported symptoms of pain, burning, soreness, achiness, and light sensitivity. Foreign body sensation or grittiness was not a prominent feature for any of them. Unlike typical LASIK-associated dry eye disease, the symptoms experienced by these patients did not gradually wane with time after surgery but rather persisted or increased. We believe that these patients had dry eye syndrome that was neuropathic in etiology, warranting treatment of underlying neuralgia rather than treatment of aqueous deficient or evaporative dry eye.

Ocular Surface Disease Index responses were distinctive in that each patient noted more severe discomfort in the *Function* subgroup compared with the *Environmental Triggers* or *Symptoms* subgroups. Of note, two patients reported initial response of symptoms to the improved environment offered by a PROSE device while it was in place, consistent with dampening of what might be neuropathic peripheral signaling of evaporation. Complete relief of symptoms with proparacaine that these patients initially experienced suggest a peripheral source of neuropathic pain. Improving the ocular surface environment may down-regulate ectopic, spontaneous, or hypersensitive signaling in nociceptor pathways. Only partial relief with PROSE devices or proparacaine suggests a central basis for pain.

In vivo confocal microscopy revealed abnormality of the corneal SNP in these patients, with reduced nerve density and altered morphology as the most prominent features. Increased tortuosity of subbasal nerves is seen after chronic damage in diabetes[27,28] and Sjögren syndrome.[29] Decreased nerve density is present in the three patients reported here, as compared with normal eyes, although it remains unclear if the pattern and time course of SNP abnormalities are different in these patients than in those who recover more typically from post-LASIK dry eye. Nerve regeneration is visible on IVCM in almost all LASIK patients 1 month after surgery, and in one quarter of patients, regenerated nerves have reached the corneal apex by 3 months.[30] The use of AST was associated with improvement in symptoms in each of these patients. Although placebo effect might account for this improvement, the lack of relief with numerous other interventions suggests a more specific mechanism.

This presentation of symptoms out of proportion to signs of desiccation along with persistent abnormality of the SNP is consistent with and characteristic of neuropathic pain. Neuropathic pain has been operationally defined as an abnormal pain state that arises from a damaged peripheral nervous system or central nervous system. There may be residual involvement of nociceptors at the site of the original injury, creating a mixed nociceptive-neuropathic pattern. In neuropathic pain, there is perpetuation of pain initiated by neural tissue damage that is beyond simple nociception.[31] In the cases presented here, injury to sensory nerves at the time of LASIK might have triggered a pain pathway resulting in refractory symptoms historically attributed to dry eye disease. We suggest that these and similar cases be categorized as corneal neuralgia after LASIK or post-LASIK neuralgia.

We emphasize that correlations can be drawn to CRPS, which has as a hallmark pain that is out of proportion to the inciting lesion.[23] Traditionally a skin and soft tissue disorder, the pathophysiology suspected for CRPS involves neurogenic inflammation and elevated neuropeptides,[32] as well as autonomic dysfunction.[33] Notably, increased neuropeptide levels in patients after LASIK have been reported.[34] Our proposal of a neuropathic basis for symptoms in these patients is consistent with previous descriptions of dysfunctional lacrimal functional unit in dry eye patients and, specifically, the occurrence of neurogenic inflammation of interconnecting innervation after insult to the ocular surface.[35] Symptoms of CRPS typically include burning and aching pain, allodynia, edema, and sweating or temperature changes.[23] Complex regional pain syndrome is further classified into type I, in which no nerve damage is obvious, and type II, in which nerve damage can be confirmed.[23] We suggest that the patients in this study might represent a variant of CRPS type II because they present with confirmed nerve damage, pain out of proportion to the injury, symptoms of burning pain, and autonomic dysfunction evidenced by marginally lower Schirmer test results. Of note, the light sensitivity these patients report could be characterized as photoallodynia[36] analogous to allodynia in CRPS.

The approach to CRPS is typically multimodal with local and systemic treatments invoked. The goal is to reduce pain signaling in the short run and centralization of pain in the long run, both of which interfere with patient function. The patients reported here were treated locally and systemically with multiple agents and all reported improvement over time; common variables were introduction of AST, which can contribute to nerve regeneration,[37,38] PROSE devices, which can reduce peripheral signaling, topical anti-inflammatory agents, which may reduce local inflammation that might up-regulate nerve signaling, and systemic agents, which can gate or modify pain signaling.

There are reports on the use of neurotrophic factors, particularly nerve growth factor, demonstrating reduction of neuropathic pain.[39,40] Therapeutic strategies resulting in regeneration of damaged corneal nerves may improve patient symptoms. Administration of neurotrophic factors has been shown to result in postinjury repair of peripheral nerves and their functional recovery.[41] Autologous serum tears may have played such a role in these patients.

Depression and emotional life events have also been associated with CRPS,[42] although this association remains unproven.[23] One patient with preexisting anxiety reported a resurgence of anxious and depressive symptoms around the time his ocular symptoms began. Another patient experienced severe depression in association with this syndrome, requiring hospitalization on two occasions. Systemic medications with neurologic and psychiatric activity were part of the recovery of these two patients. Pain without stain is real[20] and, as illustrated by these cases, may best be considered a neurologic problem with psychiatric implications. Clinicians should consider involvement of pain specialists, neurologists, and psychiatrists in the care of these patients. Describing the syndrome as a variant of CRPS type II may be helpful in guiding therapy.

Multimodal treatment including AST, PROSE treatment, topical ophthalmologic agents, and systemic psychiatric and neurologic medication was used in these three patients; thus, the relative contribution of each therapy to the patients' improvement cannot be rigorously assessed. These patients ultimately achieved resolution or near resolution of symptoms. These patients shared a common time course of a highly symptomatic first year of treatment of dry eye after LASIK followed by improvement once multimodal therapy directed toward neuropathy was introduced. Further studies of refractive surgery populations are warranted to identify risk factors for developing this syndrome. Additional data from IVCM studies are also likely to be useful in identifying patients with corneal neuralgia after LASIK. *In vivo* confocal microscopy may serve as a metric for disease in corneal neuralgia and for monitoring response to treatment.

In summary, patients with persistent dry eye symptoms out of proportion to clinical signs after LASIK have a syndrome that may best be classified as corneal neuralgia. *In vivo* confocal microscopy can be informative as to the neuropathic basis of this condition. In keeping with current understanding of CRPS, early multimodal treatment directed toward reducing peripheral neurologic signaling is warranted to avoid subsequent centralization and persistence of pain on that basis. Distinguishing this syndrome from typical post-LASIK dry eye remains a challenge.

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Deborah S. Jacobs
Boston Foundation for Sight
464 Hillside Ave Ste 205
Needham, MA 02494
e-mail: djacobs@bostonsight.org