LASIK Interface Complications: What Is the Appropriate Term for PISK?

To the Editor:

We read with great interest the article “LASIK Interface Complications: Etiology, Management, and Outcomes” by Randleman and Shah.1 We agree completely with the authors when they stated: “Clarifying the nomenclature used for each entity is critical to enhancing future communication.” It is especially important in the most rare conditions, including the one they called “pressure-induced stromal keratopathy (PISK)” as Tourtas and Cursiefen2 suggested.

It is true that in the early postoperative period of LASIK, corneal edema that might lead to accumulation of fluid in the interface or to interface and stromal haziness is almost invariably secondary to high intraocular pressure (IOP) as result of steroid response. But it does not mean that the ocular hypertension is the ultimate cause of the condition. It has been shown both experimentally and in the clinical setting that corneal edema not related to topical steroids may cause the accumulation of fluid. In an elegant ex vivo experimental study, Dawson et al. demonstrated that both endothelial cell damage and high IOP may cause swelling of the LASIK interface.3

In a recent report, a 37-year-old man underwent a bioptics procedure in which a corneal flap was created with a mechanical microkeratome followed by the implantation of an angle-supported foldable anterior chamber. He presented 43 months after surgery with interface fluid accumulation in the right eye related not to topical steroids or ocular hypertension but to endothelial dysfunction and subsequent corneal edema.4

We recently suggested a new term based on pathophysiology of the condition.5 Because the primary causative factor is edema, not IOP, we think that an appropriate term for the condition has to exclude the words “pressure-induced.” We proposed the term “post-LASIK edema-induced keratopathy (PLEK).” It may be applied to the whole spectrum of the condition, with or without apparent interface fluid, and related or not to ocular hypertension. Because the number of post-LASIK patients undergoing phacoemulsification will increase progressively, undoubtly we will see more frequently PLEK cases related to endothelial dysfunction and not related to either acute steroid response or high IOP of other causes.6

Virgilio Galvis, MD
Alejandro Tello, MD
Mario L. Revelo, MD
Paul Valarezo, MD
Centro Oftalmológico Virgilio Galvis
Floridablanca, Colombia

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Reply

We thank Galvis and colleagues for their interest in our recent review article1 and welcome the opportunity to further discuss the appropriate terminology for the entity most commonly known as PISK. This entity has been given a variety of names over the years, including pressure-induced stromal keratitis (PISK)2 and interface fluid syndrome,3 among others. We recently supported the term pressure-induced stromal keratopathy (PISK) as proposed by Tourtas and Cursiefen.4,5 Galvis and colleagues now propose a new term based on their understanding of the underlying pathophysiology; however, we disagree with their proposal and the basis for their argument.

The authors make this proposal based on their concept of the underlying mechanism for the disorder, namely, keratopathy induced by edema in the LASIK interface. However, keratopathy is not the primary problem in PISK and edema is not the primary causal factor. Endothelial dysfunction is the causal factor for
all of the various clinical presentations. Although it is true that interface fluid can arise from mechanisms other than increased intraocular pressure (IOP), such as intraocular surgery after LASIK, these other mechanisms are both readily identifiable and clinically insignificant with regard to early identification and management. Treatment of corneal edema resulting from endothelial dysfunction without increased IOP is the same in eyes with and without LASIK. Further, endothelial dysfunction without increased IOP is never the cause of interface fluid in the early postoperative period after primary LASIK, and in some cases even a delayed presentation of PISK after intraocular surgery is related to steroid-induced increased IOP.

Finally, in PISK, the “keratopathy” is, in effect, an incidental finding that may lead physicians away from the appropriate diagnosis and toward the diagnosis of diffuse lamellar keratitis if they do not recognize the underlying relationship to IOP and steroid use. The amount of fluid present or absent is insignificant because treatment is the same: specifically, cessation of steroids and IOP-lowering medications. This management is critical to employ early to prevent ultimate vision loss from optic nerve damage.

Interestingly, the case presentation the authors recently published, in which they propose their new terminology, is a perfect example of why the term PISK should be used and fully understood to represent the entire range of clinical scenarios. The authors suggested that their patient had two different syndromes, interface fluid syndrome in one eye and PISK in the other, due to different amounts of fluid visible in the interface. In fact, the underlying process and ultimate treatment was exactly the same for both eyes. To be clear, relying on direct visualization of interface fluid to diagnose “interface fluid syndrome” will only delay appropriate diagnosis and management in cases with no clinically obvious fluid.

We therefore strongly encourage the use of PISK as the single most appropriate term to describe the clinical scenario that can arise in the first few weeks after LASIK, requiring steroid cessation and IOP lowering for resolution. If physicians do not recognize that PISK is a singular entity in the early postoperative period and treat appropriately, regardless of the amount of fluid visible, devastating outcomes can occur.

J. Bradley Randleman, MD
Rupa D. Shah, MD

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