Diffuse Lamellar Keratitis 6 Months After Uneventful Laser in situ Keratomileusis

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ABSTRACT

PURPOSE: Diffuse lamellar keratitis after laser in situ keratomileusis (LASIK) typically occurs between 1 and 7 days after the procedure, and its etiologic factor(s) remain unknown.

METHODS: We describe a case of diffuse lamellar keratitis 6 months after uneventful LASIK in a 25-year-old woman.

RESULTS: Slit-lamp microscopy showed a diffuse infiltrate confined to the interface, extending to the visual axis, with no other relevant findings. Late-onset diffuse lamellar keratitis was our provisional diagnosis, and treatment with topical corticosteroids was instituted, with rapid response and improvement of the clinical signs and symptoms.

CONCLUSIONS: This case supports the theory that a previously inert inciting agent could cause a delayed toxic or inflammatory response of the cornea several months after surgery. [J Refract Surg 2003;19:70-71]

Diffuse lamellar keratitis is an uncommon laser in situ keratomileusis (LASIK) complication described as a noninfectious, diffuse interface inflammation after lamellar corneal surgery. It usually occurs in the first week after the procedure.1,2 We describe a case of diffuse lamellar keratitis 6 months after surgery.

CASE REPORT

A 25-year-old female presented for correction of low myopia at the Instituto Oftalmológico de Alicante, Spain. The patient's ocular, medical, and family history were unremarkable. Preoperative spectacle-corrected visual acuity was 20/20 with -3.00 -1.25 x 0° in the right eye and 20/20 with -3.50 -1.25 x 0° in the left eye. After informed consent, the patient underwent LASIK for myopia on 16 October 2000. The flap was created with the Automated Corneal Shaper microkeratome (Chiron Vision, Claremont, CA) with a 160-μm plate; ablation was performed with the Technolas 217C excimer laser (Technolas Gmb, Bausch & Lomb Surgical, Donarch, Germany). The surgery and immediate postoperative follow-up were uneventful. Three months after surgery, uncorrected visual acuity was 20/20 in both eyes and refraction was plano in the right eye and -0.75 x 0° in the left eye. Slit-lamp microscopy of both corneas showed a well-healed and clear flap with no debris or epithelial cells in the interface. The next follow-up examination was scheduled for 1 year.

Six months after surgery (28 March 2001), the patient came in complaining of loss of visual acuity in the right eye, and mild discomfort and tearing. On examination, uncorrected visual acuity was 20/60, and did not improve with correction. Slit-lamp microscopy showed diffuse and scattered white-grey infiltrate through the inferior half of the flap, confined to the interface, and absence of a dominant focus, epithelial defect, or anterior chamber reaction (Fig 1). There was a mild bulbar hyperemia. Provisional diagnosis of delayed stage I diffuse lamellar keratitis was made, and a topical corticosteroid (prednisolone acetate 1% every 2 hours) was started immediately. Three days later, the infiltrate was reduced to the inferior third of the flap, and uncorrected visual acuity improved to 20/30. One week after therapy, uncorrected visual acuity was 20/20, and the infiltrate had almost disappeared (Fig 2). Topical corticosteroids were tapered slowly over the next week and follow-up has remained uneventful to date.

DISCUSSION

Diffuse lamellar keratitis was first reported by Smith and Maloney in 1998.3 Since then, there have been numerous articles on this new disease.

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Possible etiologies include metallic debris from the microkeratome or blade, bacterial endotoxins, meibomian gland oils, debris from corneal ablation or from absorbent sponges, povidone-iodine solutions, and surgical glove talc. Although diffuse lamellar keratitis is described as an early complication of LASIK, we report a case of diffuse lamellar keratitis presenting 6 months after the procedure.

Harrison and Periman described a case of diffuse lamellar keratitis associated with recurrent corneal erosions 3 months after LASIK. Both manipulation of the flap or an epithelial defect may provoke interfacial keratitis. Because in our case there was no surgical manipulation or epithelial defects, this corneal inflammatory reaction can be eliminated as the causative factor. Although occult trauma such as a healed erosion with secondary lamellar inflammation could also be the cause of the disease, the patient denied any previous trauma, and the oculist examination did not show any signs of it. Thus, we can also exclude from the etiologic factors those involving direct acute physical contact with the LASIK interface. Other reports support the theory that diffuse lamellar keratitis is a nonspecific inflammatory response of the cornea, and any process that causes inflammation in the anterior segment may induce diffuse lamellar keratitis. This would include other possible etiologies such as HSV keratitis, but to date there have been no reports presenting this relationship. Probst and Foley reported late-onset diffuse lamellar keratitis after uneventful LASIK. This patient presented with a single clear inflammatory focus in the interface, and the authors suggested a distinct cause of the diffuse lamellar keratitis, not related to contamination during the procedure. Our case was a typical diffuse and multifocal interface keratitis. We believe that the toxic or allergic reaction to the unknown inciting agent nearly always happens in the early postoperative period, but it could also have a late onset. This might be explained by a delay in the mechanism that causes the disease or because the inciting agent could remain inert for several months and then, due to unknown reasons, activate the inflammatory response. The expected increase of LASIK procedures in the future leads us to speculate that the incidence of this iatrogenic disease will also increase, hence the need for more studies of pathogenic mechanisms and how to avoid them.

REFERENCES