



DOI: 10.14744/eur.2021.98698  
Eur Eye Res 2021;1(2):104–106

EUROPEAN  
**EYE**  
RESEARCH

## CASE REPORT

# Femtosecond laser in situ keratomileusis subsequently sterile peripheral necrotizing keratitis: A case report

 **Oznur Iscan,**  **Banu Torun Acar**

Department of Ophthalmology, Biruni University Hospital, İstanbul, Turkey

### Abstract

The purpose of the study was to present the clinical course and treatment of a patient who developed peripheral necrotizing keratitis (PNK) after femtosecond laser in situ keratomileusis (LASIK). We report a 30-year-old female patient who applied for refractive surgery. On the post-operative 1st day, the patient came with severe eye pain. There was an infiltration line extending from 5 to 8 o'clock at the flap border in both eyes. Confocal microscopy showed no signs in favor of fungus, the endothelium adjacent to the flap margin was intact, and there was hyper reflectance at the flap margin. There was no secretion or burring, no cells and flares in the anterior chamber, no wrinkles in the flap, haze at the interface, and epithelial defects. Topical prednisolone acetate and 1 mg/kg oral methylprednisolone were started, clinical improvement started in the post-operative 1st week, and the patient had no complaints. In post-operative 1st month biomicroscopy, the flap margin was observed naturally. Sterile PNK, seen as a rare complication of refractive surgery, has been reported as a form of diffuse lamellar keratitis. It is very important to distinguish the picture from infection and inflammation. We think that the necrotizing keratitis that developed, in this case, is due to the use of high-energy femtosecond laser, which is a rare cause.

**Keywords:** Corneal infiltrates; necrotizing keratitis; refractive surgery.

Corneal infiltrates are a rare complication after refractive surgery, which can sometimes yield significant results. This case report aims to identify causes such as bacterial and fungal keratitis and discuss their treatment. Post-laser in situ keratomileusis (LASIK) sterile corneal infiltrate mostly occurs as diffuse lamellar keratitis (DLK).<sup>[1]</sup>

DLK was first described as a granular white cellular infiltrate by Smith and Maloney<sup>[2]</sup> in 1998 and is one of the complications of LASIK surgery.<sup>[2,3]</sup> It may occur in the form of a sterile inflammatory reaction shortly after surgery and

rarely leads to permanent scarring.<sup>[3]</sup> Factors such as bacterial endotoxins, chemical residue, and surgical gloves or marker pens have been implicated in the etiology of DLK.<sup>[4]</sup> Meibomian gland dysfunction, chronic autoimmune disease and peripheral immune infiltrates, atopy, and iatrogenic epithelial defects are among the major risk factors for DLK.<sup>[5]</sup>

Understanding the distinction between sterile corneal infiltrates such as DLK and peripheral necrotizing keratitis (PNK) is important. In PNK, there is a peripheral ring stromal

**Cite this article as:** Iscan O, Torun Acar B. Femtosecond laser in situ keratomileusis subsequently sterile peripheral necrotizing keratitis: A case report. Eur Eye Res 2021;1:104-106.

**Correspondence:** Öznur İşcan, M.D. Department of Ophthalmology, Biruni University Hospital, İstanbul, Turkey

**Phone:** +90 850 811 12 76 **E-mail:** oiscan@biruni.edu.tr

**Submitted Date:** 11.03.2021 **Accepted Date:** 13.04.2021

Copyright 2021 European Eye Research Journal

**OPEN ACCESS** This is an open access article under the CC BY-NC license (<http://creativecommons.org/licenses/by-nc/4.0/>).



infiltration at the edge of the flap and a clear zone between these infiltrates and the limbus, whereas DLK, known as the sands of the Sahara, is a non-infectious condition with a sand-sprinkled infiltration between the corneal flap and the stromal bed.

Although the pathophysiology of PNK is not yet understood, it can be confused with infectious keratitis. Infectious keratitis should be included in the differential diagnosis as its treatment differs from PNK. Keratitis can be seen after any refractive laser surgery and, if it is contagious, the result is even more dramatic.<sup>[6]</sup>

We present a case of PNK with high-energy laser after LASIK with no blepharitis, meibomian gland dysfunction, or previous corneal disease; history of systemic disease; or chronic medication use.

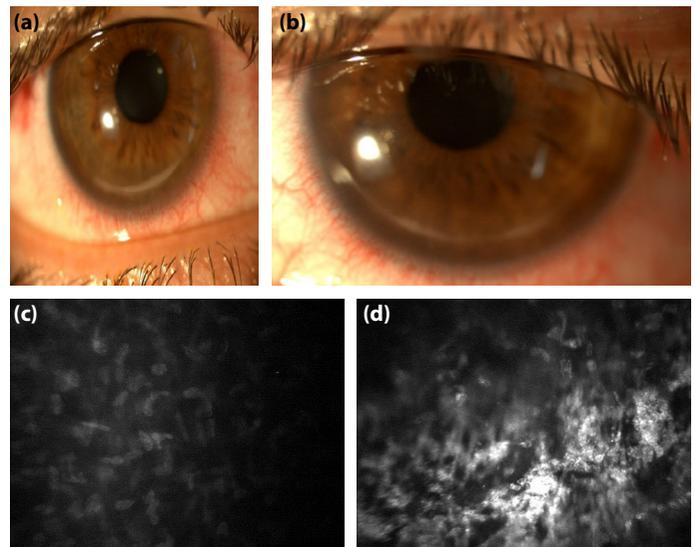
## Case Report

A 30-year-old female who presented for refractive surgery with refraction values  $-4.25 -0.50 \times 175$  and  $-5.00 -0.50 \times 10$  in the right and left eyes, respectively. Cycloplegic refraction was  $-4.00 -0.50 \times 175$  and  $-5.00 -0.50 \times 10$ , and manifest refraction was  $-4.00 -1.0$  and  $-5.00 -1.0$  in the right and left eyes, respectively. Topographic keratometry values were 41.56/42.31 in the right eye and 41.36/42.72 in the left eye.

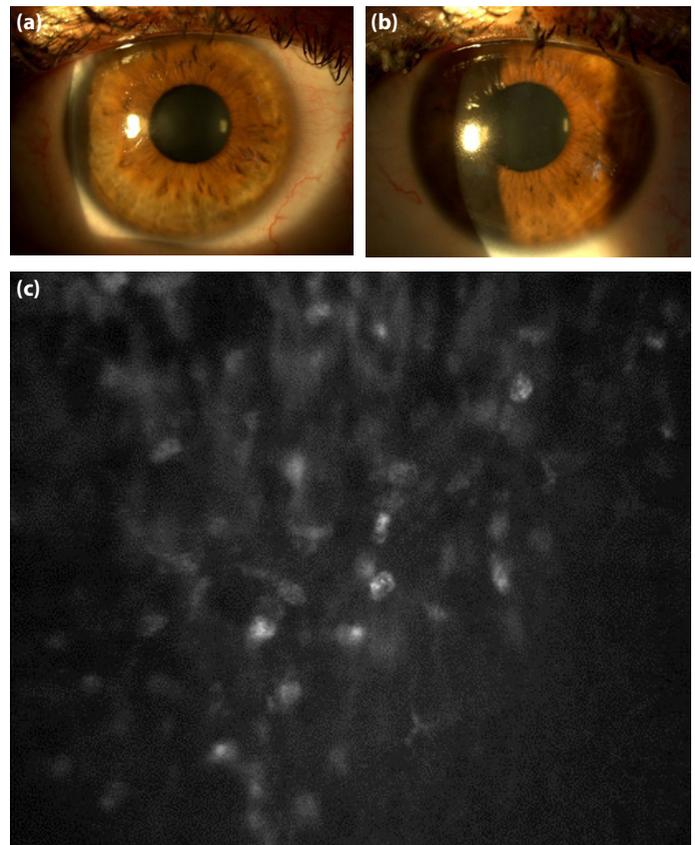
iFS 150 (Abbott Medical Optics) femtosecond laser flap was prepared, VISX Star S4 IR® (Abbott Medical Optics) was ablated with excimer laser (right eye  $-4.00$  D, left eye  $-5.00$  D) Topical fluorometholone and moxifloxacin were started hourly as per treatment protocol.

Postoperatively, the patient came with severe eye pain on the 1<sup>st</sup> day. Uncorrected visual acuity was 0.9 in both eyes and refraction was  $+0.50$  in the right and  $+0.50 -0.50 \times 156$  in the left eye. The patient had mild hyperemia in the conjunctiva in both eyes on biomicroscopy. There was no secretion or burring, cells or flares in the anterior chamber, wrinkles in the flap, corneal interface haze, or epithelial defects. Intraocular pressure was 13 mmHg on the right and 11 mmHg on the left. There was an infiltration line extending from 5 to 8 o'clock at the flap border in both eyes (Fig. 1a and b). Confocal microscopy showed no signs in favor of fungus, the endothelium adjacent to the flap margin was intact, and there was hyper reflectance at the flap margin (Fig. 1c and d).

Topical fluorometholone was discontinued, prednisolone acetate and 1 mg/kg oral methylprednisolone were started, and moxifloxacin was continued every hour. Clinical improvement started in the post-operative 1<sup>st</sup> week, and



**Fig. 1.** On post-operative 1st day, infiltrates were observed at the laser in situ keratomileusis flap margin in both eyes, including the flap interface, and the epithelium was intact. **(a)** Right eye. **(b)** Left eye. **(c)** Stroma and keratocytes are healthy followed up in confocal microscopy. **(d)** Hyper reflectance at the flap margin is seen on confocal microscopy.



**Fig. 2.** One month postoperatively, the necrotizing inflammation was entirely resolved, and the flap margin was observed. **(a)** Right eye. **(b)** Left eye: Widespread hyper reflection observed at the previous examination has disappeared. **(c)** A small number of keratocyte hyper reflectance is seen on confocal microscopy.

the patient had no complaints. Prednisolone acetate and moxifloxacin were reduced to 5×1, and a tapering dose of oral prednisolone was planned before it was stopped. In post-operative 1<sup>st</sup> month biomicroscopy, the flap margin was observed naturally (Fig. 2a and b), and small number of keratocyte hyper reflectance is seen on confocal microscopy (Fig. 2c). The uncorrected visual acuity was 1.0 in both eyes.

## Discussion

Sterile PNK, seen as a rare complication of refractive surgery, has been reported as a form of DLK after LASIK.<sup>[6]</sup> Sterile PNK caused by chronic blepharitis, meibomian gland dysfunction, systemic inflammatory and autoimmune disease, chronic topical NSAID or anesthetic use, previous corneal inflammation.<sup>[7,8]</sup> It is essential to distinguish between infection and inflammation. Lack of symptoms such as conjunctival ciliary injection and anterior chamber reaction and the presence of intact epithelium exclude the presence of infection or inflammation.

In the case presented in this report, a detailed patient history showed the absence of systemic disease, chronic drug use, previous corneal disease, and any signs of this excluded us from the diagnosis of infection or inflammation. Auto-immune and staphylococcal hypersensitivity mechanisms were held responsible for post-LASIK sterile infiltrates. No known autoimmune disorder, ocular rosacea, or blepharitis was seen in this patient.

Lifshitz et al. state that “the immune reaction alone appears to be an inadequate explanation” for this complication and suggest that the effects of the laser, in combination with yet-to-be demonstrated ocular and systemic factors, trigger PNK.<sup>[6]</sup>

de Medeiros et al. showed that LASIK flaps could be created with IntraLase with a different energy, they emphasized that larger corneal inflammations occur in patients with rheumatoid arthritis, which is related to the immunological process.<sup>[9]</sup>

Although cases with sterile keratitis due to high-energy femtosecond laser have been reported in literature, sterile keratitis has also been reported in patients treated with low-energy femtosecond laser.<sup>[1,9]</sup>

The primary goal should be to rule out infectious keratitis as its treatment and management are very different from

DLK, and its potential outcome is worse. It must be distinguished from DLK, which constitutes a majority of the post-LASIK sterile corneal infiltrates. In conclusion, we think that the PNK developed in our case was due to high-energy femtosecond laser, which is a rare cause.

**Informed Consent:** Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

**Peer-review:** Externally peer-reviewed.

**Authorship Contributions:** Concept: B.A.; Design: B.A.; Supervision: Ö.İ., B.A.; Resource: Ö.İ.; Materials: B.A.; Data Collection and/or Processing: B.A.; Analysis and/or Interpretation: Ö.İ.; Literature Search: Ö.İ., B.A.; Writing: Ö.İ.; Critical Reviews: Ö.İ., B.A.

**Conflict of Interest:** None declared.

**Financial Disclosure:** The authors declared that this study received no financial support.

## References

1. Bucci MG, McCormick GJ. Idiopathic peripheral necrotizing keratitis after femtosecond laser in situ keratomileusis. *J Cataract Refract Surg* 2012;38:544–7. [\[CrossRef\]](#)
2. Smith RJ, Maloney RK. Diffuse lamellar keratitis. A new syndrome in lamellar refractive surgery. *Ophthalmology* 1998;105:1721–6. [\[CrossRef\]](#)
3. Parolini B, Marcon G, Panozzo GA. Central necrotic lamellar inflammation after laser in situ keratomileusis. *J Refract Surg* 2001;17:110–2. [\[CrossRef\]](#)
4. Yuhan KR, Nguyen L, Wachler BS. Role of instrument cleaning and maintenance in the development of diffuse lamellar keratitis. *Ophthalmology* 2002;109:400–3; discussion 403–4.
5. Fogla R, Rao SK, Padmanabhan P. Diffuse lamellar keratitis: Are meibomian secretions responsible? *J Cataract Refract Surg* 2001;27:493–5. [\[CrossRef\]](#)
6. Lifshitz T, Levy J, Mahler O, Levinger S. Peripheral sterile corneal infiltrates after refractive surgery. *J Cataract Refract Surg* 2005;31:1392–5. [\[CrossRef\]](#)
7. Ambrósio R Jr., Periman LM, Netto MV, Wilson SE. Bilateral marginal sterile infiltrates and diffuse lamellar keratitis after laser in situ keratomileusis. *J Refract Surg* 2003;19:154–8.
8. Moshirfar M, Welling JD, Feiz V, Holz H, Clinch TE. Infectious and noninfectious keratitis after laser in situ keratomileusis occurrence, management, and visual outcomes. *J Cataract Refract Surg* 2007;33:474–83. [\[CrossRef\]](#)
9. de Medeiros FW, Kaur H, Agrawal V, et al. Effect of femtosecond laser energy level on corneal stromal cell death and inflammation. *J Refract Surg* 2009;25:869–74. [\[CrossRef\]](#)