
Sterile interface keratitis associated with micropannus hemorrhage after laser in situ keratomileusis

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ABSTRACT

Numerous etiologies have been suspected to lead to sterile interface keratitis after laser in situ keratomileusis. This tan interface haze with a rippled appearance has been called Sands of the Sahara. We present 2 cases in which red blood cells entered the interface after a small hemorrhage from peripheral corneal vascularization during the microkeratome pass. Although this bleeding was controlled and all visible blood cells were removed at surgery, both patients developed the appearance of a focal interface keratitis on the first postoperative day. *J Cataract Refract Surg* 1999; 25:1679–1681 © 1999 ASCRS and ESCRS

Focal lamellar keratitis may result from hemorrhage into the interface after transection of the micropannus vessels during laser in situ keratomileusis (LASIK).

Case Reports

Case 1

A 46-year-old woman consulted the Casey Vision Correction Center for correction of myopic astigmatism. She had been diagnosed with adult-onset diabetes 6 months previously that was under good control; she was otherwise healthy. Her preoperative manifest refraction was $-9.00 +1.75 \times 35$ in the right eye and $-9.00 +1.75 \times$

The patient was examined 1 month later and was correctable to 20/20 with $-2.00 +0.25 \times 10$. The interface cells had disappeared. There was no evidence of interface scarring. Three months after the initial treatment, an enhancement was performed in the right eye by manually lifting the flap. No interface hemorrhage was noted at the time of treatment. Three months later, UCVA was 20/20⁺².

The other eye was treated with a Hansatome microkeratome (Chiron Vision Corp.), which was used to create a 9.5 mm flap. Less bleeding occurred in this case as the Hansatome created a superior flap hinge and transected fewer superior pannus vessels than the nasal hinge created with the ACS. This bleeding was easily controlled during surgery. On the first postoperative day, no interface haze was noted and the UCVA was 20/30. Five months later, the patient required an enhancement in this eye; 3 days later, UCVA in her left eye was 20/20⁺².

Case 2

A healthy 45-year-old woman with a 6 year history of soft contact lens wear consulted the Casey Vision Correction Cen-

ter for correction of myopic astigmatism. Her preoperative manifest refraction was $-4.75 +0.50 \times 70$ degrees in the right eye and -4.75 sphere in the left eye. After an uneventful LASIK procedure in her left eye, she elected to have LASIK in her right eye. On the day of surgery, a Hansatome microkeratome was used to create a 9.5 mm flap. Bleeding was noted inferiorly at the 6 o'clock position at the flap edge. The bleeding was controlled by placing a Murocel-soaked sponge with Neo-Synephrine 2.5% for approximately 20 seconds before the flap was retracted. The patient was treated with a 6.0 mm optical zone and 7.5 mm transition zone.

As in Case 1, the bed was wiped to remove excess red blood cells and other intravascular contents from the interface after the laser treatment. Gentle irrigation was performed with BSS and the flap allowed to float back into position. The patient was evaluated 5 and 40 minutes after the flap was placed back in position; flap position was excellent, with no interface bleeding noted.

On the first postoperative day, the patient reported a burning sensation. There were fine cells inferiorly in small

vertical wave-like columns in the flap interface (Figure 2). At that time, the UCVA was 20/30. The flap was in excellent position and the central cornea clear. The patient was treated with tobramycin and dexamethasone (TobraDex) 4 times a day. By the fifth postoperative day, the interface haze was barely visible. At this visit the visual acuity was 20/25⁺² with a correction of $-1.50 +0.50 \times 180$. The TobraDex was tapered over the following 2 weeks. The patient will have subsequent retreatment for undercorrection.

Discussion

These cases demonstrate a potential etiology of interface opacification in the early period after LASIK. Unlike previously described cases of diffuse lamellar keratitis, these cases illustrate a focal lamellar keratitis. In both patients, the interface cellular debris was directly adjacent to the vascularization and the site of intraoperative hemorrhage. The larger (9.5 mm) flap created by the Hansatome microkeratome made it more likely to sever a peripheral corneal vessel. Because the interface was clear at the end of the surgery, it seems likely there was a small amount of leakage from the blood vessel into the interface in the first 24 hours postoperatively. In both cases, the cells were laid in a linear fashion with an axis perpendicular to the direction of the microkeratome pass.

These cases demonstrate another potential cause of post-LASIK sterile interface keratitis. Current theories as to the etiology include, but are not limited to, the following: lubricant or rust from the microkeratome,¹

debris on the microkeratome blade, meibomian gland secretions, lid debris, excimer laser energy, BSS, benzalkonium chloride, or particulate matter. We believe these 2 cases may have been caused by blood vessel leakage of red blood cells and other intravascular contents into the interface in the early postoperative period. The inflammation in both cases was focal or sectorial and limited to the area in which the hemorrhage occurred in the interface. This is unlike other reports that describe the haze as a diffuse lamellar keratitis.² In the current cases, because the haze did not involve the visual axis, it did not have a significant impact on visual function.

Micropannus formation is commonly seen with soft contact lens wear, and if the microkeratome pass transects these vessels, hemorrhage may occur. This complication may be frequently encountered with the increasing use of larger flaps (9.0 to 9.5 mm) and larger treatment zones needed for hyperopia. We caution surgeons to be aware that blood vessel leakage may be a cause of post-LASIK sterile interface keratitis or the so-called Sands of the Sahara syndrome.

References

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2. Smith RJ, Maloney RK. Diffuse lamellar keratitis: a new syndrome in lamellar refractive surgery. Ophthalmology 1998; 105:1721–1726