

Pain and Vision Loss After PRK

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**BY GEORGES BAIKOFF, MD; CHRISTOPHER D. WIRBELAUER, MD;
AND A. JOHN KANELLOPOULOS, MD**

CASE PRESENTATION

A 30-year-old male underwent bilateral PRK in January 2007. After a few days of severe pain immediately postoperatively, he was given several eye drops, including a topical anesthetic.

During this time, the patient's vision deteriorated, and he came to our practice for a second opinion. At this time, he was using exocin (ofloxacin) and dexamethasone (Maxidex; Alcon Laboratories, Inc., Fort Worth, Texas) every hour, Tobradex (Alcon Laboratories, Inc.) and ketorolac tromethamine (Acular; Allergan, Irvine, California), NaCl 5%, four times daily, and an unknown special solution prescribed by the treating doctor.

Five months later, the patient's vision was at counting fingers, with both eyes presenting photophobia and pain. Figures 1 and 2 reveal a 90% epithelial defect, corneal haze, an opaque crystalline lens, and significant iris ectropion with correctopia toward 6–o'clock.

What course of action would you take to address this patient's serious complications?



Figure 1. Patient at examination.



Figure 2. Eye with fluorescein staining.

GEORGES BAIKOFF, MD

This patient's condition is quite impressive. The clinical case resembles either a toxic anterior segment syndrome or a caustic burn.

This could be explained by several postoperative months of the excessively prescribed eye drops (eg, anesthetics, Acular, and the special solution). Excessive use of topical anesthetic eye drops may induce, what in France is called *painful anesthesia*: The first drop calms the pain, but it returns very quickly, inciting the patient to continue its use. Moreover, anesthetic products penetrate the cornea, creating a toxic effect on the endothelium and, possibly, even on the iris structures. NSAID eye drops are also aggressive on a cornea that has had the epithelium and Bowman's membrane removed. It is important for surgeons to remember that NSAID drops should only be prescribed for 1 or 2 days. In my practice, I have had several patients with fragile corneas experience complications from topical NSAID eye drops after cataract surgery.

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Faced with such a situation, my first reaction would be to stop all aggressive therapy, especially because exocin can cause damage to the cornea in the long run. While waiting for the epithelium to heal, we will have to fight this vicious cycle of pain—which will take up to several weeks to disappear.

I would, therefore, suggest an intensive therapeutic mydriasis (atropine six times daily). This is not so much

to dilate the atrophic pupil, but more to rest the ciliary body and calm the corneal pain. I would add rifamycin three times daily to avoid additional bacterial infection of the ulcer and prescribe a bandage lens and an oral analgesic treatment if necessary.

I have encountered several cases of iatrogenic toxic corneal syndromes in my practice, and after a few days, it is possible to calm the pain these patients suffer. Once the epithelium is healed, we will be able to see how the corneal stroma reacts—as its transparency is linked to the condition of the endothelium and to the stromal scars induced by the long-standing corneal ulcer.

If the endothelium is still functional, there is a chance that after approximately 6 months, we will be able to recuperate a more or less transparent cornea. We would then decide whether a lamellar or penetrating corneal graft is necessary and whether the crystalline lens would have to be removed. Atrophic mydriasis, however, is typically permanent.

CHRISTOPHER D. WIRBELAUER, MD

The described case reveals a dramatic course following PRK with vision loss, corneal opacification, cataract formation, and iris ectropion. The clinical information is unfortunately sparse, but the intraocular pressure (IOP) was within normal range.

There are three main problems in this case. The first is the superficial wound healing after PRK with a large epithelial defect and corneal opacification. Whether this condition was caused by a toxic keratopathy due to prolonged application to topical anesthetics (or other unknown eye drops or by some underlying pathologic conditions) remains unclear. Here, I would discontinue all medications, except for artificial lubricants, to promote epithelial healing. After completed epithelial closure, I would treat corneal haze with mild topical steroids, such as fluorometholone, for several weeks.

The second problem is ectropion uveae, which is caused by proliferation of endothelial cells and/or a collagenous membrane with myofibroblasts on the anterior surface of the iris. It most frequently occurs in secondary neovascular glaucoma. However, the patient's IOP was normal. Other reasons for this ectropion could be a chronic intraocular inflammation, a viral (herpetic) cause, endothelitis, or an endothelial dystrophy (eg, ICE syndrome). The last is usually more focally localized and one-sided, but would also explain the dramatic course after PRK. In the case of a congenital endothelial dystrophy, this patient was not a good candidate for refractive corneal surgery. Further gonio-

scopic examination would be helpful to elucidate this pathology and the occurrence of peripheral anterior synechiae. Unfortunately, the uveal ectropium will not reverse completely.

Third, vision loss seems to be mainly related to corneal opacification and cataract. The optic nerve and retina, however, should be examined to exclude further posterior segment pathologies, in particular ischemic eye processes, which would need a specific therapeutic approach (eg, photocoagulation). Depending on the course and clearing of the corneal clouding, this patient will probably need further surgery in the long-term—such as cataract extraction with IOL implantation and some form of lamellar or penetrating corneal transplant surgery—to finally restore vision.

A. JOHN KANELLOPOULOS, MD

To address this patient, we first had him discontinue all medication. Next, we started him on preservative-free prednisolone acetate 1% and homologous serum (prepared at his local hematology lab) every hour. The patient used this treatment for almost 2 months until the injection subsided and the epithelium healed.



Figure 3. The patient had a scarred cornea and cataract postoperatively.

The patient was left with a scarred cornea and dense cataract in both eyes (Figure 3), but I believe that penetrating keratoplasty combined with cataract extraction will sufficiently rehabilitate this patient's vision.

When I was training in the early 1990s in inner-city hospitals in New York, I encountered numerous patients with similar epithelial toxicity. The cause in these cases, however, was not PRK, but crack cocaine abuse. We learned that the toxic fumes from smoking crack irritated the cornea, but also offered analgesia.

Some patients started using cocaine topically on their eyes and developed severe epithelial toxicity and eventually, perforation.

Topical anesthetic abuse has the same damaging potential, and clinicians should be extremely scrupulous in their use of topical anesthetics in PRK patients. Patients with chronic epithelial toxicity should be questioned about anesthetic abuse or self-medicating with another toxic agent. ■

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