Transient no light perception visual acuity after intracameral lidocaine injection

Richard S. Hoffman, MD, I. Howard Fine, MD

ABSTRACT

The use of intracameral lidocaine for augmenting analgesia during intraocular surgery is becoming increasingly popular. We report a case of complete visual loss after the use of intracameral lidocaine to repair a traumatic corneal graft dehiscence. Full visual recovery returned several hours after surgery, suggesting this medication's relative lack of retinal toxicity. *J Cataract Refract Surg 1997; 23:957–958*

The use of nonpreserved intracameral lidocaine to assist in analgesia during intraocular surgery has become increasingly popular as more surgeons are performing clear corneal cataract surgery using topical anesthesia. Although intracameral lidocaine is believed to be safe for the cornea and anterior chamber,[1–3] concerns persist regarding the possible toxic effects of lidocaine on the retina. We report a case of transient complete visual loss in a patient receiving intracameral lidocaine for the repair of a traumatic corneal graft dehiscence under topical anesthesia.

Case Report

A 40-year-old man suffered a limbus-to-limbus corneal laceration in his left eye from a fishing gaff hook 2 years earlier. One week after returning to shore, he presented for repair of his laceration, which was followed shortly after by a pars plana lensectomy, pars plana vitrectomy, and scleral buckle. His visual acuity could be corrected to 20/40, and interferometry revealed a potential acuity of 20/15. He was unable to tolerate a rigid, gas-permeable contact lens and eventually had penetrating keratoplasty and secondary iris-fixated intraocular lens (IOL) implantation and pupilloplasty, which yielded an uncorrected visual acuity of 20/70.

Four months after the transplant, the patient placed his thumb into his unshielded left eye during sleep, dehiscing the graft from 12 to 6 o'clock, which resulted in complete hypotony and flattening of the anterior chamber. His visual acuity before repair was hand motions. The dehiscence was repaired under topical anesthesia using lidocaine 4% topical drops and a 0.5 cc of intracameral nonpreserved lidocaine 1%, which was injected into the anterior chamber through the graft dehiscence. Viscoelastic was placed in the anterior chamber to re-form the eye, and an air bubble was placed in the anterior chamber to help avoid phototoxicity from the operating microscope and posterior chamber lens. The repair was performed without difficulty. The eye was not patched after surgery so that the patient could place frequent antibiotic and steroid drops in the eye.

Immediately after surgery, visual acuity was checked and found to be no light perception to indirect ophthalmoscopy, which revealed a flat retina without retinal ischemia and a healthy optic nerve head. A 4+ relative afferent pupillary defect was present in the left eye before fundus evaluation. One hour later, his visual acuity was light perception. He was sent home and contacted several hours later by telephone, at which time he stated that his vision had returned and he was now able to easily see his fingers in front of him. One day postoperatively, uncorrected visual acuity was 20/200 through a mildly edematous cornea. One week later, the graft was clear and visual acuity was 20/50 with pinhole. At 1 month, best corrected visual acuity was 20/25.

From Oregon Eye Associates, Eugene, Oregon, USA.

Reprint requests to Richard S. Hoffman, MD, Oregon Eye Associates, 1550 Oak Street, #5, Eugene, Oregon 97401, USA.

Discussion

Gills' technique of intracameral nonpreserved lidocaine to augment analgesia during surgery under topical anesthesia has been a great advancement in ophthalmic anesthesia for cataract surgery. Recent reports of transient visual loss following the use of intracameral lidocaine have brought attention to the possible effects of this medication on the retina and optic nerve, especially in the presence of compromise to the posterior lens capsule or zonules.

We believe our patient's complete visual loss was caused by total anesthesia of the retinal nerve fiber layer at or near the optic nerve head. Because all ganglion cell axons converge at the optic nerve head, even a small amount of anesthetic at this location could result in complete visual loss. Ordinarily, intracameral lidocaine instilled during cataract surgery does not affect the retina because of the barrier effect of the lens capsule, zonules, and vitreous humor. This patient, having had a pars plana lensectomy and vitrectomy, had essentially no barrier for diffusion of the lidocaine into the posterior chamber. Visual recovery coincided with the anesthetic duration of action of lidocaine, suggesting that this medication was the cause of the profound temporary visual loss.

Although no electroretinograms were performed to document complete return of retinal function, the patient felt that full function had returned within several hours other than the generalized decrease in visual acuity resulting from corneal edema he had experienced after his initial transplant.

This case demonstrates anecdotal evidence for the relative lack of retinal toxicity of nonpreserved lidocaine in small intracameral concentrations. Patients who may suffer mild or profound loss of visual acuity after cataract surgery with topical and intracameral lidocaine secondary to breaks in the posterior capsule can be reassured that visual function will return in several hours. This, however, should not preclude a thorough retinal evaluation to exclude the possibility of antibiotic toxicity, occlusive retinovascular disease, or retinal detachment. We have observed several cases in which additional anesthetic was placed in the eye after a capsular tear had developed and additional analgesia was necessary. These patients had unexplained mild visual loss immediately after surgery that returned to normal by the first postoperative day. We now believe that intracameral anesthesia to the retinal nerve fiber layer may have been the cause for their temporary visual loss.

In the case presented here, intracameral lidocaine allowed the procedure to be performed without general anesthesia or local injections. Before the procedure, the patient was having significant ciliary body pain and photosensitivity. Immediately after the intracameral injection, his periorbital pain and photosensitivity were totally relieved, which allowed the repair to be completed with both the patient and surgeon much more comfortable and relaxed.

Further investigations should ultimately reveal the safety and efficacy of intracameral lidocaine use in the removal of cataracts and the repair of traumatic corneal lacerations in cooperative patients.

Reference

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