Transient complete visual loss after intracameral anesthetic injection in cataract surgery

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Abstract

Purpose: We describe a case of transient visual loss following cataract surgery with unpreserved intracameral lidocaine.

Method: A 50-year-old man with posterior polar cataract underwent phacoemulsification. Following capsulorhexis and hydrodelineation with 0.5 cc of unpreserved lidocaine 1%, a portion of fluid reached behind the crystalline lens and caused the posterior capsule rupture. Cataract extraction and anterior vitrectomy were performed. Anesthetic administration was repeated to relieve the discomfort felt by the patient. A three-piece hydrophobic acrylic intraocular lens was implanted in the ciliary sulcus.

Results: On the first postoperative morning, the patient's vision was recorded as having no light perception. The relative afferent pupillary defect (RAPD) was found to be 4+. The retina and optic nerve head appeared normal. In the afternoon, the visual acuity (VA) was improved to 3-m count-finger. On the second postoperative morning, the patient's VA was improved to 4/10. On the third postoperative day, his VA returned to normal at 20/20 without RAPD.

Conclusion: In the event of posterior capsular rupture, to reduce retinal toxicity risks, intracameral lidocaine should not be repeated.

Keywords: Intracameral lidocaine; Retinal toxicity; Amaurosis; Vision loss

Introduction

Using unpreserved intracameral lidocaine as an adjunct anesthetic to topical anesthesia has become a widespread technique in cataract surgery. While safety and efficacy of intracameral lidocaine is well-documented, retinal toxic effects and transient visual loss caused by lidocaine have been suggested.

We report a case of transient, complete visual loss, following posterior polar cataract surgery with posterior capsular rupture.

Case report

A 50-year-old man presented to our clinic complaining of blurred vision for one year in his right eye. Preoperative best-corrected visual acuity was 10/20. Slit-lamp examination revealed a posterior polar cataract. His intraocular pressure and other ocular examinations including relative afferent pupillary defect (RAPD) were unremarkable. He was accordingly scheduled for phacoemulsification cataract surgery.

A small-incision cataract surgery was performed under topical anesthesia and intracameral lidocaine. Following capsulorhexis and hydrodelineation with 0.5 cc-unpreserved lidocaine 1%, a portion of fluid reached behind the crystalline lens and caused the posterior capsule rupture.

Cataract extraction and anterior vitrectomy were performed. During the anterior vitrectomy procedure, the patient experienced an intense pain. This prompted us to repeat the anesthetic.

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An additional 0.5 cc of lidocaine 1% was administered to relieve his discomfort. A three-piece hydrophobic acrylic (HOYA) intraocular lens (IOL) was placed in the ciliary sulcus. At the conclusion of surgery, the eye was patched, and the patient was discharged.

On the first post-operative morning, the patient complained of not being able to see anything. His vision was recorded as having no light perception. The RAPD was found to be +4. The fundus was examined under full pupillary dilation. The retina appeared normal, and the optic disc was not edematous. In the afternoon, the patient’s visual acuity (VA) was improved to 3-m count-finger. On the second postoperative morning, the patient’s VA was improved to 4/10. Vision assessment on the early morning of the third postoperative day was surprising. The patient’s VA had improved to 20/20 without RAPD, and all other ocular examinations were normal.

**Discussion**

The preferred technique for cataract surgeons in the United States is topical anesthesia (37%; range 22–63%). In a survey conducted by David Learning, 76% of respondents preferred using topical anesthesia with intracameral lidocaine injection. Intracameral unpreserved lidocaine augments analgesia and significantly decreases intraoperative pain perception. Lidocaine is a useful adjunct, particularly in cases involving pupillary manipulation and peripheral iridectomy.

While lidocaine has been reported to be safe and effective, temporary visual loss and retinal toxicity following the use of intracameral lidocaine have been highlighted in recent reports. An increased possibility of adverse effects of lidocaine on the retina and optic nerve is introduced in the event of compromise to the posterior lens capsule.5

Our patient had experienced transient visual loss while receiving intracameral lidocaine and had an associated posterior capsule rupture, which has an incidence of between 26% and 36% in posterior polar cataract surgeries. Normally, during cataract surgery, the lens capsule, zonules, and vitreous humor have a barrier effect against instilled intracameral lidocaine, and hence, the retina will not be affected. Pars plana lensectomy and vitrectomy facilitate the diffusion of the lidocaine into the posterior chamber.4

Gills et al.9 reported amaurosis in four patients following the use of intracameral lidocaine, and in each case, the posterior capsule was not intact. All four patients recovered completely within hours. Hoffman and Fine4 reported on a patient with complete visual loss after intracameral lidocaine to repair a traumatic corneal graft dehiscence with a capsular tear, which was fully recovered several hours postoperatively. Falzon et al.10 reported a case of transient, complete loss of vision following phacoemulsification with an intracameral ophthalmic viscosurgical device (OVD) and lidocaine solution complicated by posterior capsule rupture, which improved to 20/80 after one day and 20/25 in one week.

Full recovery of visual acuity in our patient required three days, in contrast to the majority of reports (Table 1) with capsular tear that had complete VA improvements within
hours. The longer time span in recovery of VA in our patient might be explained by a higher volume of lidocaine that was diffused in the vitreous.

Lincoff et al. reported three cases of inadvertent intravitreal lidocaine injections, all of which resulted in immediate decreased vision, with recovery occurring within four hours. Schechter reported a case of inadvertent intraocular lidocaine injection with an immediate visual acuity of NLP (no light perception) that improved to 20/40 after one day and to 20/20 in one week.

Toxicity of lidocaine to rodent retinal ganglion cells has been documented. Lidocaine affects the pigment transport in retinal cells of crayfish and frogs. This inhibition is probably due to an anesthetic-induced disruption of intercellular ionic balance and increased plasma membrane permeability. Furthermore, intravitreal injection of lidocaine has also been investigated in cats, rabbits, and rats. In these animal studies, the electroretinogram analyses revealed a reversible reduction in the amplitude and extinguished b-waves which fully recovered within 10–24 h. In cats, intravitreal injection of lidocaine has been followed with vacuolization of nerve layers and presence of microscopic lesions in synapses between horizontal, bipolar cells, and photoreceptors. In addition, histopathological retinal structure changes near the injection sites have been detected.

One limitation of our study is that the patient did not consent to perform the ERG and mERG tests. However, in the presence of positive RAPD, normal retinal examination, and full recovery of vision, the most plausible cause of vision loss would be attributed to lidocaine retinal toxicity. In view of the above, when posterior capsular rupture occurs, it is wise not to repeat intracameral lidocaine to minimize potential retinal toxicity risks.

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