ASRS American Society of Retina Specialists

Journal of VitreoRetinal Diseases
2025, Vol. 9(6) 900–903

DOI: 10.1177/24741264251366411
journals.sagepub.com/home/jvrd
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Electroretinographic Response Following Inadvertent Intravitreal Injection of Lidocaine 2%

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Abstract

Purpose: To describe the visual and electroretinographic (ERG) outcomes after inadvertent intravitreal injection of lidocaine. **Methods:** A 75-year-old man with neovascular age-related macular degeneration inadvertently received approximately 0.07 mL of lidocaine 2% intravitreally in his right eye. Serial 30 Hz flicker ERG was performed at 3, 6, and 24 hours postinjection. **Results:** Visual acuity decreased to count fingers immediately after the injection and returned to baseline of 20/30 within 24 hours. ERG showed increased amplitudes at 3 hours in the affected eye (47.7 microvolts) relative to the unaffected eye (35.7 microvolts) that normalized at hours 6 (23.5 microvolts OD, 29.1 microvolts OS) and 24 (13.4 microvolts OD, 11.8 microvolts OS). Mean implicit time (26.97 ms OD, 27.0 ms OS) was symmetric. **Conclusions:** In our case, visual acuity reduction and ERG changes specific to 0.07 mL intravitreal lidocaine 2% seem to be transient and may not require any specific therapy other than immediate intraocular pressure control.

Keywords

intravitreal lidocaine, electroretinography, ERG, retinal toxicity

Introduction

Intravitreal injection is a commonly performed ophthalmic procedure for which local anesthesia can be achieved with a variety of topical or subconjunctival anesthetic agents.[1] The incidence of inadvertent intraocular injection of local anesthetic agents is unknown, and the visual outcomes vary widely. There is no clear guidance on how to best manage such cases.[2–5] Retinal response as recorded by electroretinography (ERG) has only been documented in a handful of cases in humans. Lincoff et al[5] noted a rapidly extinguished ERG waveform after intravitreal injection of lidocaine 2% with slow but partial return of the b-wave amplitude after approximately 4 hours in rabbit eyes. Additionally, Anders et al[6] evaluated 30 Hz flicker ERG in 200 patients undergoing cataract surgery with intracameral lidocaine 1% and found no difference in amplitude or implicit time compared to control. We describe the visual and electrophysiologic outcome in a patient after inadvertent intravitreal injection of lidocaine 2%.

Case Report

A 75-year-old man with neovascular age-related macular degeneration treated with aflibercept intravitreal injections every 5 weeks presented for scheduled injections in both eyes. His visual acuity was 20/50 in each eye with intraocular pressures (IOP) of 13 mm Hg in the right eye (OD) and 11 mm Hg in the left eye (OS). Due to a medication error, the patient received approximately 0.07 mL of non-preserved lidocaine 2% meant for subconjunctival anesthesia intravitreally OD through the pars plana. A variety of factors contributed to a protocol deviation that resulted in a medication error, including: the physician was practicing at a satellite location, the assisting technician was new to working with the treating physician, and the lidocaine syringe was unlabeled. After injection, there was a rapid reduction of visual acuity to count fingers at 2 feet OD and IOP elevation to 85 mm Hg. After prompt anterior chamber paracentesis, the IOP was reduced to 8 mm Hg without change in vision. The patient was started on topical bimatoprost, prednisolone acetate and moxifloxacin.

Three hours after the injection, visual acuity OD remained decreased at 20/200 with IOP 28 mm Hg. A 30 Hz flicker ERG

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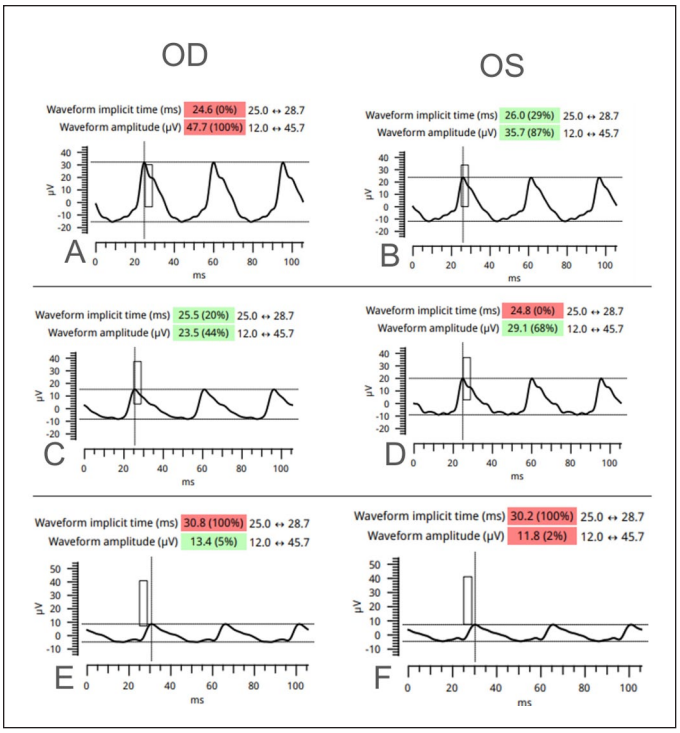

Figure 1. 30 Hz flicker ERG in the right and left eye taken 3 hours (A, B), 6 hours (C, D) and 24 hours (E, F) after inadvertent intravitreal injection of approximately 0.07 mL lidocaine 2% in the right eye.

was obtained in each eye utilizing the handheld RETeval (LKC Technologies, Gaithersburg, MD) device. ERG data at 3 hours were obtained after the patient's eyes were dilated and after at least 10 minutes of steady background illumination.[7] Flicker ERG showed an increased b-wave amplitude of 47.7 microvolts OD and a normal amplitude of 35.7 microvolts OS (Figure 1). Implicit times were within normal limits at 24.6 ms OD and 26.0 ms OS. Six hours postinjection, visual acuity improved to 20/70 with IOP 28 in the right eye. Corresponding dilated flicker ERG at 6 hours showed normalization of b-wave amplitudes to 23.5 microvolts OD and 29.1 microvolts OS. Implicit times remained relatively unchanged at 25.5 ms OD and 24.8 ms OS. Twenty-four hours after injection, visual acuity returned to baseline at 20/30 with IOP 12 mm Hg OD. Undilated flicker ERG showed symmetric and normal amplitudes (13.4 microvolts OD and 11.8 microvolts OS) and implicit times (30.8 ms OD and 30.2 ms OS). Visual acuity 10 weeks postinjection remained at baseline, 20/50. The patient was followed closely thereafter and did not show any further IOP elevations, intraocular inflammation or endophthalmitis.

Optical coherence tomography (OCT) taken prior to the injection of lidocaine 2% showed a macular central subfield thickness of 203 μm OD and 230 μm OS. Retinal thickness as well as microanatomy, including outer retinal layer integrity, remained stable OD at 1, 4, 10, and 17 weeks postinjection (Figure 2).

Discussion

The described case demonstrated a profound but transient reduction of vision after inadvertent intravitreal injection of approximately 0.07 mL lidocaine 2% with full and sustained recovery occurring within 24 hours. There was no evidence of associated retinal drug toxicity on OCT up to 17 weeks after injection. The 30 Hz flicker ERG showed initial increased b-wave amplitude in the affected eye 3 hours postinjection that normalized and became symmetric with the unaffected eye within 6 hours. The implicit time appeared unaffected at all time points.

The mechanism of lidocaine-induced vision loss is likely related to inhibition of voltage-gated sodium channels in the neural membranes of retinal ganglion cells.[8] The effect of lidocaine on photoreceptors, bipolar cells and other retinal glial cells has not been fully elucidated. Lincoff et al[5] injected lidocaine 2% in rabbit eyes and noted a rapidly extinguished ERG response with return of a-wave amplitudes coincident with reperfusion of the central retinal artery. Liang et al[9] injected lidocaine and bupivacaine intravitreally in rabbit eyes at increasing dosages while maintaining physiologic IOP and found that only the b-wave of a Ganzfeld ERG was diminished without diminution of the a-wave and with full recovery of the ERG waveform back to baseline values within 24 hours. Taken together, this suggests that electrophysiologic dysfunction may be related to IOP elevation at least in some cases. In our patient, a modest volume of approximately 0.07 mL was injected, yet there was a profound rise in IOP necessitating anterior chamber paracentesis.

Increased 30 Hz flicker ERG amplitudes have been documented in the handheld RETeval system in both ischemic and inflammatory conditions, such as retinal vein occlusion, diabetic retinopathy, intraocular foreign body, early active birdshot retinochoroidopathy, and during the postoperative period of cataract surgery.[10] Ischemic and inflammatory conditions increase the production of nitric oxide, which is known to stimulate retinal cells, and this mechanism has been offered to explain the ERG findings in the conditions listed above. Lidocaine and other local anesthetic agents act via inhibition of voltage-gated sodium channels on neural membranes, thereby halting propagation of action potentials. Grosskreutz et al[8] demonstrated that glutamate antagonists were protective to retinal ganglion cells exposed to lidocaine, suggesting that there may be a component of direct lidocaine-induced excitotoxicity. Unopposed retinal excitation could lead to increased ERG amplitudes.

Unpreserved, diluted intracameral lidocaine hydrochloride 1% to 4% is used in cataract surgery without evidence of sustained vision loss or retinal toxicity.[6] Nevertheless, animal models appear to demonstrate an increased risk of retinal toxicity and retinal ganglion cell atrophy when local anesthetic agents are delivered intravitreally at higher concentrations, such as with lidocaine 2%.[8,11,12] Analysis of rabbit retinas exposed to lidocaine 2% have found no distinctive histologic changes aside from nonspecific swelling of the retinal ganglion

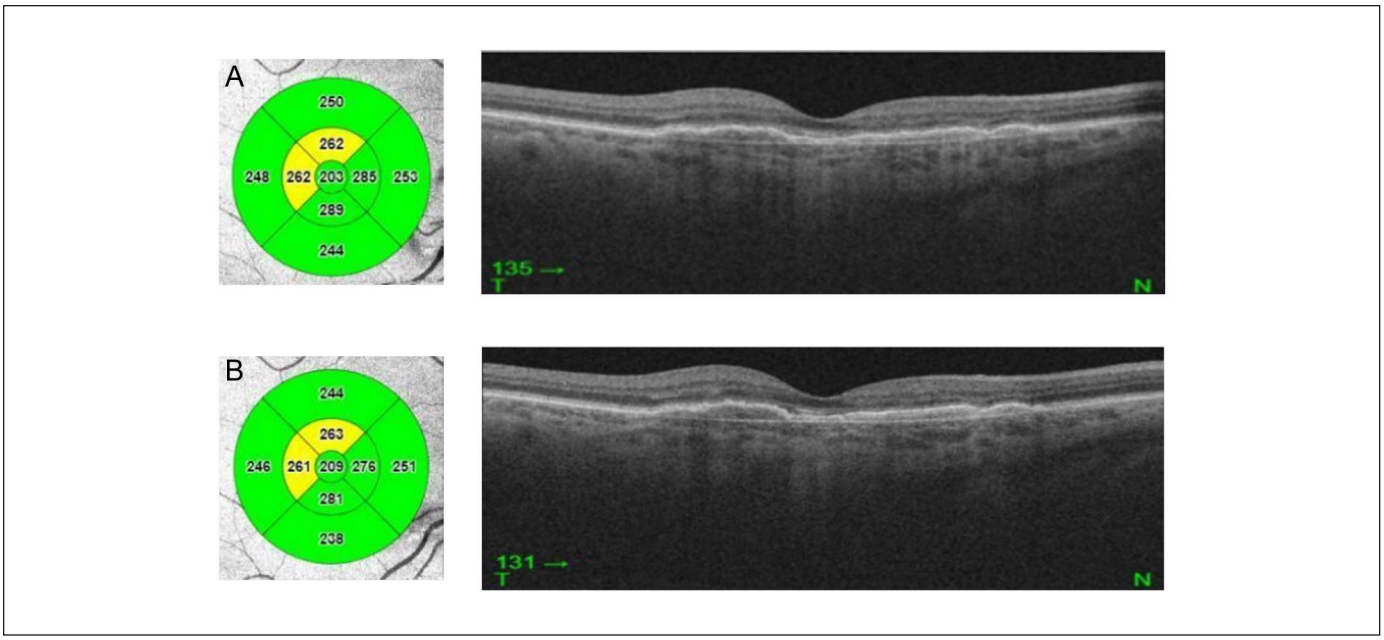

Figure 2. OCT of the right eye, just prior to (A) inadvertent intravitreal injection of approximately 0.07 mL lidocaine 2% and at 17 weeks postinjection (B).

cells.[5,10,11] Our patient did not demonstrate signs of toxic retinopathy on OCT or dilated funduscopic exam up to 17 weeks postinjection. The serial flicker ERGs in this case provide insight into the natural history and clinical outcome of intravitreal lidocaine-induced vision loss, even if ERG data is limited to a 30 Hz flicker protocol in this case.

The rate of wrong-site or wrong-medication intravitreal injection is not known and likely under-reported in the literature. With the proliferation of FDA-approved intravitreal medications and biosimilars, there are more opportunities for incorrect medication or dosage administration in a busy retina practice. Several strategies to mitigate safety events associated with intravitreal injection have been reported. A safety analysis performed at the Kellogg Eye Center identified factors that lead to intravitreal injection-related safety events at their institution, including: staffing inexperience/shortage, incomplete notes or documentation, incorrect order placed in the electronic record, inconsistent use of multiple patient identifiers during procedural time out, and omission of injected medication during the procedural time out. After addressing these pitfalls through modification of the time out and procedural protocol, the rate of intravitreal injection-related safety events at the Kellogg Eye Center dropped dramatically.[13] Patel et al[14] conducted a root cause analysis of intravitreal injection-related safety events at their institution and found that by implementing an institutional protocol that facilitated clear templated documentation of the treatment plan and required independent verification of both medication and injection site by the injecting physician and an allied health professional, the annual rate of safety events reduced from 0.1% to 0.01%. Inadvertent injection of intravitreal lidocaine in our case occurred because of a deviation in the practice protocol with several contributing factors, including the use of an unlabeled medication, staff inexperience and practicing at a satellite location with a team unaccustomed to executing the institutional injection protocol. Strict adherence to an injection protocol with specification of the anesthesia plan and treatment plan may have averted wrong medication administration in our case.

In summary, inadvertent intravitreal lidocaine injection may be an under-reported adverse event in clinical practice. Management recommendations are not clearly formulated in the literature, and anecdotally range from observation to immediate pars plana vitrectomy. In our patient, intravitreal lidocaine 2% caused profound yet transient loss of vision and associated electrophysiologic changes that completely resolved within 24 hours without any signs of toxicity on long-term follow-up. In cases of inadvertent intravitreal lidocaine 2% injections, the authors recommend close attention to issues related to intraocular pressure and possible intraocular inflammation or endophthalmitis. However, visual acuity reduction as well as ERG changes specific to approximately 0.07 mL intravitreal lidocaine 2% seem to be transient and may not require any specific therapy.

Ethical Approval

Ethical approval for this study was waived by the Institutional Review Board because of the study design as a retrospective case report.

Statement of Informed Consent

Written informed consent was obtained from the patient, and permission was given to publish the case without identifying patient information.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

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