

Inadvertent Intravitreal Injection of 0.1mls of Unpreserved Lidocaine 2%

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Citation

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Abstract

Background: There is limited literature on inadvertent intraocular Lidocaine injections in humans. There is usually an element of ocular trauma, haemorrhage and increase of intraocular pressure IOP contributing to reduced function. In addition there may be other compounds in the injected solutions, which may confound the assessment of ocular toxicity. Finally because of the accidental nature of these injections it is difficult to establish the exact volume and concentration of Lidocaine in the vitreous. We report the effect of inadvertent intravitreal injection of a known volume of unpreserved Lidocaine 2%.

Methods: 1 patient accidentally receiving 0.1ml of intravitreal Lidocaine 2% instead of 0.1ml of Ranimizumab for cystoid diabetic macular oedema. Best corrected visual acuity (BCVA), contrast sensitivity, IOP, and central macular thickness (CMT) were recorded before and after the intravitreal injection. The patient was followed up for 4 months

Results: BCVA, contrast sensitivity and CMT were all marginally improved 1 month after the intravitreal injection of Lidocaine 2%.

The BCVA and contrast sensitivity remained stable and the CMT improved further after subsequent intravitreal Ranimizumab injections over a 4 months period.

Conclusion: There were no previous reports on the effect of intravitreal unpreserved Lidocaine of a known volume and without coexisting ocular injury and IOP rise. In our case, 0.1 mls of intravitreal unpreserved Lidocaine 2% in an eye with diabetic maculopathy showed no adverse events and was associated with marginal improvement of visual acuity, contrast sensitivity and OCT measured central macular thickness.

INTRODUCTION

Small volumes of unpreserved Lidocaine 1% are used “off label” intracamerally in cataract surgery. This is useful for augmenting topical anaesthesia and facilitating further pupillary dilatation. There are no reports on the biologic and clinical effects of a known volume of unpreserved Lidocaine in vivo in the human eye. This is to our knowledge the first case of an inadvertent pars plana intravitreal injection of unpreserved Lidocaine 2% of a known volume (0.1ml).

MATERIALS/METHODS

A 75 year old diabetic female with persistent diffuse diabetic macular oedema was entered into a clinical trial and randomised to receive monthly intravitreal anti-VEGF injections.

Her medical history was significant for type 2 diabetes

mellitus and hypercholesterolaemia. Her ophthalmic history included diabetic maculopathy with one previous macular laser session in the right eye and moderate non-proliferative diabetic retinopathy in both eyes. There was no history of trauma or previous eye surgery.

During her baseline examination before commencing the trial, her best corrected visual acuities (BCVA) on the Early Treatment of Diabetic Retinopathy Study (ETDRS) scale were 61 letters OD (equivalent to 20/60) and 63 letters OS (equivalent to 20/55). Her baseline contrast sensitivities with Peli Robson chart were 24 letters OD and OS (contrast sensitivity score 1.05). Her intraocular pressures (IOPs) were measured at 15mmHg and her central macular thickness (CMT) in the study eye (OD) was 468µm on Optical Coherence Tomography (OCT).

Before her 4th intravitreal Ranimizumab injection her

BCVAs were the same as baseline and her central macular thickness 387µm on OCT.

Her Right eye was prepped and topical anaesthesia and povidone Iodine 5% were instilled in the conjunctival sac. Unpreserved Lidocaine 2% and 0.1mls of Ranimizumab were drawn in 2 separate 1ml tuberculin syringes. Subconjunctival injection of lidocaine 2% was administered at the injection site and the eyeball was massaged gently. The patient then accidentally had a pars plana injection of 0.1mls of Lidocaine 2%. There was no vitreous leak at the injection site and no change in the visual status immediately after the injection. The injecting ophthalmologist looking at the needles on the tuberculin syringes spotted the mistake. The safety and critical incident processes were put in motion immediately and the patient was informed and counselled.

Her post injection ophthalmic examination showed a mild subconjunctival haemorrhage at the site of the injection and mild punctuate corneal epithelial staining. Her IOP was 22mmHg (OD) and there was neither vitreous haemorrhage nor retinal tears or detachment. It was decided that she should skip her intravitreal Ranimizumab injection on that occasion. A safety check by telephone, the following day and a clinic review 3 days post injection were unremarkable. The patient decided that she would like to continue her intravitreal anti VEGF injections.

During her following visit one month later her BCVAs were 64 letters OD (equivalent of 20/50) and 71 letters OS (equivalent of 20/40). Her Peli Robson contrast sensitivities were 27 letters OD and 29 letters OS (contrast sensitivity score 1.20 and 1.30 respectively). Her OCT of the Right eye showed a reduction of CMT to 378µm. In subsequent visits and further intravitreal Ranimizumab injections the CMT was reduced to 287 µm and the BCVA remained stable at 66 letters OD on ETDRS scale.

DISCUSSION

The following adverse events are known to be associated with intraocular injection of Lidocaine: 1) increase of IOP, 2) corneal haze, 3) transient electroretinography changes. The IOP elevation is a consequence of the increased intraocular volume and not due to a pharmacologic effect of Lidocaine. There are few published data on the biologic effect of various concentrations of Lidocaine in in vitro and animal eye studies. These are focused mainly on the anterior segment structures (i.e. corneal endothelium), whereas posterior segment data are not very well explored.

Intracameral use of preservative free lignocaine 1% has been used in selected patients undergoing cataract surgery to augment topical anaesthesia. In a review article by Karp CL et al (1), it appears that in short term studies the above concentration is safe to the corneal endothelium but there is controversy on the effectiveness and the ideal timing for anaesthesia.

Animal studies show a dose and exposure time dependent corneal endothelial toxicity of unpreserved Lidocaine (2,3).

Intravitreal injections in animals caused a dose dependant transient decrease of b-wave on electroretinography(4,5,6). This short-lived inner retinal dysfunction was not associated with any permanent histological changes.

It is difficult to assess ocular morbidity from the limited literature on inadvertent Lidocaine injections in humans. There is usually an element of ocular trauma, haemorrhage and increase of IOP contributing to reduced function. In addition there may be other compounds in the injected solutions, which may confound the assessment of ocular toxicity (7). Finally because of the accidental nature of these injections it is difficult to establish the exact volume and concentration of Lidocaine in the vitreous.

In our case a volume of 0.1mls of unpreserved Lidocaine 2% was administered through the pars plana without any subsequent retinal injury or IOP elevation. This particular brand contains a Lidocaine solution with almost neutral Ph.

The patient experienced no visual disturbance after the injection and both her ETDRS visual acuities and contrast sensitivities were marginally improved and this was associated with a reduction in central macular thickness.

CONCLUSION

0.1 mls of intravitreal unpreserved Lidocaine 2% in an eye with diabetic maculopathy showed no adverse events and was associated with marginal improvement of visual acuity, contrast sensitivity and OCT measured macular thickness.

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