



Intralenticular Ozurdex Implant: A Case Report

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Abstract

We present the case of a 54-year-old male with deterioration of vision in the right eye. CME due to non-infectious, intermediate uveitis was diagnosed. The patient was treated with an intravitreal ozurdex implant that was inadvertently injected into the lens. The lens remained transparent for 8 months, but high, uncontrolled intra-ocular pressure and posterior sub-capsular cataract developed. The patient underwent phacoemulsification cataract surgery, posterior capsulotomy and anterior vitrectomy. An IOL was implanted in the bag.

Case Presentation

A 54-year-old male presented to our clinic for evaluation for deterioration of vision in his right eye over the last year. He had known neurofibromatosis (NF) without systemic manifestations and low grade myopia. Ocular history included cystoid macular edema (CME) in the right eye that was treated unsuccessfully with intraocular anti-VEGF. On examination, visual acuity was 20/60 in the right eye and 20/20 in the left eye. Ophthalmologic examination of the right revealed clear anterior chamber, open angle with large amount of pigment without peripheral anterior synechia, cataract grade NS+1, vitreous cells grade 2 without haze or snow balls. The retina was attached with inferior pigmented atrophic retinal hole with condensed vitreous above it. Macular examination revealed CME. Left eye exam was within normal limits.

Investigation

Spectral-domain optical coherence tomography revealed CME of 685 μm and sub-retinal fluid (Figure 1). Fluorescein angiography demonstrated late leakage from the inferior part of the optic disc and CME. He had no complaints of difficulty swallowing or long standing cough; no weight loss or diarrhea; no fever, arthritic pain or neurological signs. Systemic workup for uveitis included complete blood count, chemistry, erythrocyte sedimentation rate, C-reactive protein, serum angiotensin-converting enzyme (ACE), Mantoux test for tuberculosis and serology for detecting treponema. All were within normal limits. MRI of the brain and orbit was performed to rule out brain involvement of NF and demyelinating disease. A 6 mm meningioma near the falx cerebri on the right side was the only finding. Chest X-ray was normal. These findings suggested a diagnosis of noninfectious, intermediate uveitis with CME.

Management

Due to previously unsuccessful anti-VEGF treatment, we decided to treat the CME with sustained-release 0.7 mg dexamethasone intravitreal implant (Ozurdex; Allergan, Irvine, CA). Barrier laser photocoagulation around the atrophic hole was performed. On examination it was seen that intraocular Ozurdex implant was inadvertently injected into the lens (Figure 2). In addition, local retinal detachment developed around the atrophic hole, which was treated with barrier laser photocoagulation around the area of the detachment. During the next 8 months, the patient's vision improved significantly to 20/40 pinhole, 20/25. CME resolved and remained at 244 μm to 300 μm . The crystalline lens remained transparent for 8 months and a posterior, sub-capsular cataract gradually developed. The Ozurdex implant was horizontally oriented involving the visual axis, which could explain the visual acuity improvement with pinhole. The IOP gradually increased to 32 mmHg. The patient was placed on maximum medical topical and oral doses. However, he could not tolerate the oral drugs due to side-effects. It was decided to perform cataract surgery and to remove the ozurdex implant simultaneously. The ozurdex implant penetrated the posterior capsule into the clear lens where the PSC developed. Routine phacoemulsification cataract surgery was performed under topical anesthesia. After a clear corneal incision and insertion of anterior chamber maintainer, standard capsulorhexis, hydrodelamination, hydrodissection, and phacoemulsification were performed. At the end of nucleus removal, a tear in the posterior capsule was identified. The

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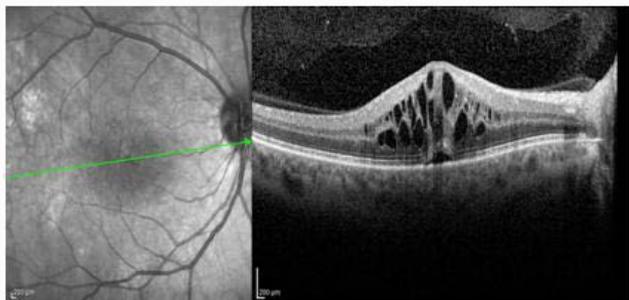


Figure 1: Spectral-domain optical coherence tomography revealed CME of 685 μm and sub-retinal fluid.

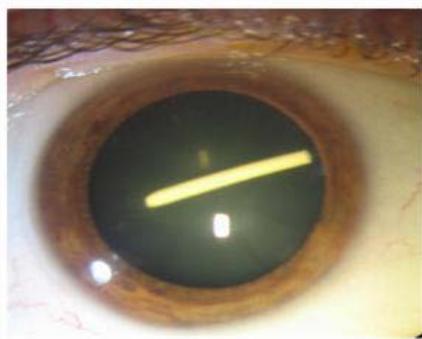


Figure 2: Intraocular Ozurdex implant was inadvertently injected into the lens.

anterior chamber was filled with ophthalmic viscoelastic devices (OVD), a continuous, circular posterior capsulotomy was performed in addition to anterior vitrectomy and PCIOL was implanted in the bag. Post-operatively, maximal anti-glaucoma therapy was initiated, along with topical antibiotics and steroids (See attached video of the surgery). Complications involving the anterior segment after injection of intravitreal dexamethasone implants are rare and mainly involve migration of the implant into the anterior chamber in pseudophakic eyes with posterior capsular tear [1-3]. A few cases regarding inadvertent injections of dexamethasone implants into the crystalline lens have been reported [4-9]. In all cases, cataract surgery was performed and a 3-piece IOL was implanted in the sulcus. Except for one case [10], fixation of the intraocular lens (IOL) into the capsular bag was enabled due to fibrosis of the capsular defect.

Follow-up

During the first month after surgery, the anti-glaucoma therapy was gradually tapered as the IOP decreased. UCVA was 20/40. On the fifth week after surgery, CME reappeared to 487 μm . It was decided with the patient to treat with periocular transeptal methylprednisolone injection (Depo-Medrol, Pfizer, New York). One month after the injection, CME worsened to 598 μm and subretinal fluid developed. It was decided to start prednisone 60 mg per os and anti-glaucoma drops. However, the CME recurred when steroids were tapered to 20 mg. We then decided to inject Ozurdex implant intravitreally, which successfully resolved the CME. The IOP was controlled at around 18 mmHg by a combination of carbonic anhydrase inhibitors and beta-blockers.

Discussion

Uveitis refers to a group of intraocular inflammatory diseases that cause 10% to 15% of blindness in the developed world. Despite advances

in immunosuppressive therapy, corticosteroids remain the mainstay of treatments. They can be administered systemically or locally by topical, periocular, or intravitreal routes. Persistent inflammation and cystoid macular edema) secondary to ocular inflammation are often vision-threatening and pose a significant therapeutic challenge. The Ozurdex[®] (Allergan, Inc., Irvine, CA, USA) dexamethasone drug delivery system is a biodegradable intravitreal implant that delivers sustained release of 700 μg of preservative-free dexamethasone to the retina and vitreous. It is approved by the United States Food and Drug Administration as a first-line therapy for the treatment of macular edema following branch or central retinal vein occlusion, as well as for noninfectious posterior uveitis [11-13]. Injecting steroids into the vitreous cavity has the advantage of minimal systemic side-effects. The use of Ozurdex is encouraging due to its potency, dose consistency, extended duration of action, and minimal adverse effects [14-18]. However, we also need to manage local side-effects such as cataract formation, IOP elevation, subconjunctival hemorrhage, hyperemia, and conjunctival edema. These are often temporary and can be managed medically [19,20]. Dexamethasone has potent anti-inflammatory properties with a favorable side-effect profile [11,12,21]. Previous studies demonstrated that dexamethasone in a biodegradable drug delivery system (Ozurdex[®]; Allergan Inc, Irvine, California, USA) can improve visual acuity and macular thickness in a variety of settings [11,12,21]. In our case, the Ozurdex implant was inadvertently injected into the visual axis of the lens, but it remained clear for 8 months. PSC developed only in the area where the Ozurdex implant penetrated the lens. Our decision to perform cataract surgery due to the implant in the lens, the cataract and the high IOP that arose and needed full medical treatment. The entrance tear in the posterior capsule that was caused by the implant, was converted to posterior capsulotomy and PCIOL was successfully implanted in the bag. To the best of our knowledge, this is the only case reported where the IOL was implanted in the bag. Because the Ozurdex was implanted in the crystalline lens it degraded much more slowly than it normally would have. The very slow, sustained release of the dexamethasone in the implant prevented CME from developing for 8 months until the lens and the implant were extracted. During the long follow-up period, the patient underwent several intravitreal ozurdex injections with successful resolution of CME, with only two anti-glaucoma drugs required.

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