

CASE REPORT

Management of cataract and uncontrolled high intraocular pressure after inadvertent intralenticular dexamethasone implant injection



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A 67-year-old man developed macular edema in the left eye as a result of branch retinal vein occlusion. An intravitreal dexamethasone implant (Ozurdex) was injected. Despite macular edema regression 4 weeks later, the corrected distance visual acuity decreased from 0.3 decimal to 0.1 decimal. Anterior segment examination showed the dexamethasone implant was inadvertently injected into the crystalline lens. Although only 1 injection was given, 2 dexamethasone implants were observed, 1 located in the posterior pole of the lens and the other in the anterior vitreous. The

intraocular pressure (IOP) was 60 mm Hg. Phacoemulsification surgery was performed. Because of the high IOP, both implants were removed. A 3-piece hydrophobic acrylic intraocular lens was implanted in the ciliary sulcus. At the 3-month follow-up, the visual acuity was 0.6 decimal as a result of the macular complications associated with vein occlusion, the IOP was 18 mm Hg under glaucoma medication, and no complication was observed.

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The Ozurdex dexamethasone implant (Allergan, Inc.) is a water-soluble biodegradable copolymer of lactic acid and glycolic acid and containing micronized dexamethasone, which can have beneficial effects in the treatment of macular edema secondary to retinal vein occlusions, diabetic retinopathy, and uveitis.¹ The implant is a 6.0 mm × 0.46 mm rod-shaped device containing 700 µg of dexamethasone. After 6 months, the concentration of the drug is below the limit of quantitation.² Endophthalmitis, glaucoma, and cataract are well-known complications of the implant. Lenticular injury is rare; however, it has been reported.¹ We describe a patient with elevated intraocular pressure (IOP) resulting from the presence of 2 implants in the eye; 1 implant was inadvertently injected into the lens, and the other was located in the anterior vitreous.

CASE REPORT

A 67-year-old man had a 5-month history of hazy vision in the left eye. The corrected distance visual acuity (CDVA) was 0.3 decimal. Fundus examination showed branch retinal vein occlusion (BRVO) with dot and blot hemorrhages, cotton-wool spots, dilated tortuous veins, and macular edema. Diffuse macular edema was determined by optical coherence tomography. Fundus fluorescein angiography showed capillary nonperfusion areas and

dye extravasation caused by retinal neovascularization. The IOP was 19 mm Hg. Accordingly, an intravitreal dexamethasone implant injection followed by retinal laser photocoagulation were scheduled.

Despite macular edema regression 4 weeks after dexamethasone implant injection, the CDVA decreased from 0.3 decimal to 0.1 decimal. The IOP was 60 mm Hg. Two dexamethasone implants were observed; 1 was located in the posterior pole of the crystalline lens and the other in the anterior vitreous (Figure 1, a and b). The dexamethasone implant was inadvertently injected into the crystalline lens. Posterior subcapsular and cortical cataract development was noted (Figure 1, a). Mannitol 20.0% (300 cc) was administered intravenously and brimonidine, latanoprost, and a fixed combination of dorzolamide–timolol were prescribed for the treatment of the elevated IOP. It was hypothesized that the implant might have been divided into 2 pieces during injection given the sustained high IOP despite maximum antiglaucoma treatment. In the same session, phacoemulsification cataract surgery with intraocular lens (IOL) implantation and extraction of both pieces of the implant was scheduled.

Phacoemulsification cataract surgery was performed 1 week later. After nucleus removal, a posterior capsule defect was noted around the dexamethasone implant. Anterior vitrectomy was performed (Figure 1, c). Because of the high IOP, both implants were removed. A 3-piece hydrophobic acrylic IOL was implanted in the ciliary sulcus (Figure 1, d). Extraction of the fragmented implant pieces was performed as follows: After removal of the lens nucleus by phacoemulsification, the cortical materials (located far from the intralenticular implant and defective posterior capsule) were

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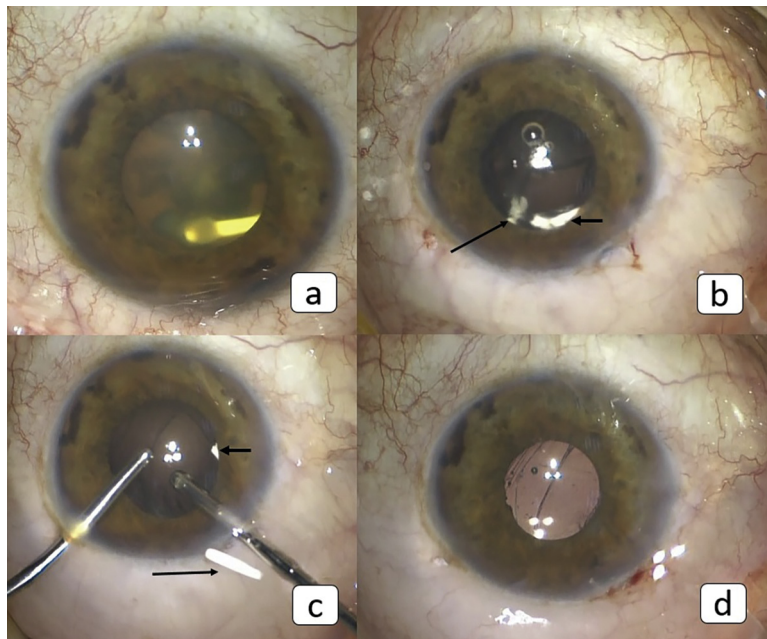


Figure 1. a: Cataract and intralenticular dexamethasone implant. b: Short arrow indicates intralenticular part of implant. The long arrow indicates intravitreal part of implant. c: Anterior vitrectomy. Intralenticular part of implant (short arrow) and removal of the intravitreal part of implant (long arrow). d: At the end of the surgery.

aspirated using bimanual irrigation/aspiration (I/A) probes. Then, anterior vitrectomy was performed around the posterior capsule rupture area through the main corneal incision. The piece of implant located in the vitreous became free when the vitreous around the implant was removed; the piece was moved toward the main corneal incision. This piece was easily extracted through the application of fluid dynamics. Bimanual I/A was performed to remove remaining cortical materials. The intralenticular implant was held using an aspiration probe and transported to the anterior chamber. A dispersive ophthalmic viscosurgical device was injected in front of the posterior capsule rupture area and anterior chamber. Then, this piece of the implant was held with a 23-gauge serrated retinal forceps through the main corneal incision. Meanwhile, the biodegradability of the implant caused the implant to divide into 2 pieces. These pieces were gently held and removed. Small particles (formed after the fragmentation of the implant) were aspirated using a bimanual I/A probe.

On the first day after surgery, the patient's CDVA was 0.4 decimal and the IOP was 19 mm Hg. At the final visit at 3 months, the CDVA was 0.6 decimal as a result of macular complications associated with retinal vein occlusion, the IOP was 18 mm Hg on glaucoma medication (brimonidine 2 times a day), and no complication was observed.

DISCUSSION

Lenticular damage during intravitreal injections was reported to be 0.009% in a multicenter case series study.³ The technique of injecting dexamethasone implants is different from other intravitreal injections because of the larger bore of the 22-gauge needle. To avoid complications, the location of injection should be 3.5 to 4.0 mm posterior to the limbus. The needle should be sustained on the intrascleral path approximately 1.0 mm parallel to the limbus. Then, the direction must be changed toward the center of the vitreous cavity.

There are several reports on iatrogenic crystalline lens injury during the intravitreal injection of dexamethasone implants. In most reports, intralenticular dexamethasone implant injection led to cataract progression in weeks and

cataract surgery was performed in 3 to 6 months.^{4–6} Two patients had cataract surgery within 1 week after injection to prevent the development of cataract progression or IOP elevation.^{7,8} Caglar⁹ monitored a patient for 12 months and performed the phacoemulsification procedure. However, Poomachandra et al.¹⁰ observed no cataract progression after 10 months of follow-up. In most of these cases, the IOP was increased and treatment with topical antiglaucoma agents was required. In 2 cases, the IOP was normal.^{4,11} In a case by Lee et al.,¹² the IOP fluctuated between 19 mm Hg and 40 mm Hg despite treatment with glaucoma medications; cataract surgery was performed after 10 months of follow-up.

For some patients, proactive approaches are more suitable than conservative approaches to address increased IOP, an abnormal anterior segment, and cataractous changes in the crystalline lens. If any of these is detected early, another option is to perform immediate phacoemulsification with IOL implantation and repositioning of the implant into the vitreous cavity.¹³ A risk with this approach is the migration of the implant into the anterior chamber through the posterior capsule defect, which can cause corneal decompensation.¹⁴

In the present case, cataract surgery, including extraction of the implants, should have been performed because of increased IOP and cataract progression. Although only 1 injection was performed, 2 implants were observed in the eye. This might be attributed to the fragmentation of the implant during or after injection. There are several reports of the fragmentation of the implant after injection. Roy and Hegde¹⁵ pointed out that after segmentation, an increased surface area might cause faster dissolution of the implant and release of a higher concentration of the drug. In addition, the authors indicated that the risk for glaucoma is higher because of the increased surface area. However, Im et al.¹⁶ compared the visual and anatomic outcomes and

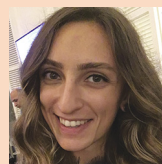
complications of 2 groups of patients with macular edema resulting from BRVO. In one group, the dexamethasone implant fragmented after injection and in the other group it did not fragment. There was no difference in the rate of macular edema recurrence, frequency of IOP elevation, or cataract progression between the 2 groups. In our case, an elevation in the IOP might have been associated with a higher concentration of drug being delivered when the implant fragmented. We believe that this is the first reported case in which 2 independent dexamethasone implants, which might have split during or after injection, were observed in the crystalline lens and anterior vitreous at the same time.

In conclusion, iatrogenic lens damage from inadvertent dexamethasone implant injection can cause the implant to fragment. Patients should be monitored for cataract development or IOP elevation. Immediate surgical removal is not always required; however, if there is an uncontrolled increase in the IOP or cataract development is detected, surgery should be scheduled as soon as possible.

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