We report the case of a 58-year-old man who developed macular edema due to superior temporal vein occlusion in his right eye. A dexamethasone implant was inadvertently injected into the crystalline lens. Slitlamp examination revealed the position of the implant. The intralenticular dexamethasone implant was clearly imaged in front of the posterior lens capsule by high-frequency ultrasound biomicroscopy. The patient was followed without repositioning of the dexamethasone implant. During the 6-month follow-up, no additional side effect was observed, the macular edema decreased, and the vision improved.

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Retinal vein occlusion is considered the second most common retinal vascular disorder after diabetic retinopathy.1 Cystoid macular edema (CME) is a common sight-threatening complication of retinal vein occlusion.2 Multiple treatment strategies have been attempted for the treatment of macular edema, including laser photocoagulation, antivascular endothelial growth factor, and corticosteroid injections.3

The dexamethasone intravitreal implant (Ozurdex), a biodegradable implant of dexamethasone (0.7 mg) used as injection into the vitreous cavity, has recently become a valuable treatment option for eyes with visual impairment due to macular edema related to central retinal vein occlusion and branch retinal vein occlusion. This rod-shaped implant is 6.0 mm in length and 0.46 mm in diameter. The implant dissolves in the vitreous while slowly releasing the dexamethasone for up to 6 months. It has been approved by U.S. Food and Drug Administration for the treatment of macular edema secondary to retinal vein occlusion and noninfectious uveitis affecting the posterior segment.4,5 As a consequence of increased popularity of intravitreal injections, many complications such as intraocular pressure (IOP) elevation, cataract formation, endophthalmitis, intraocular hemorrhage, hypotony, and retinal detachment have been reported.4,5 We report an inadvertent injection of a dexamethasone implant into the crystalline lens. To our knowledge, this is the first report of a patient with an inadvertent dexamethasone implant without repositioning in the absence of side effects.

CASE REPORT

A 58-year-old man presented with a 3-day history of decreased vision in his right eye. The corrected distance visual acuity (CDVA) was 1.0 logMAR (decimal equivalent of 0.1) in the right eye and 0 logMAR (decimal equivalent of 1.0) in the left eye. Intraocular pressure was 17 mm Hg and 16 mm Hg, respectively. Anterior segment examination revealed early nuclear cataract in both eyes. Fundus examination showed macular edema and retinal hemorrhages in the superior retina of the right eye (Figure 1, A). Optical coherence tomography (OCT) revealed CME with a central macular thickness of 579 μm (Figure 1, B). Clinical findings and investigations confirmed superior branch retinal vein occlusion with macular edema in the right eye. Injection of a dexamethasone implant was planned, and the injection was done in the superotemporal quadrant of the right eye. The needle was introduced 4.0 mm posterior to the limbus. However, the applicator needle was not perpendicular to the scleral surface during injection, so the needle was advanced into the lens and the dexamethasone implant was injected into the crystalline lens.

On the first postoperative day, slitlamp examination of the right eye showed the implant had penetrated the crystalline lens (Figure 2, A). The IOP was 22.0 mm Hg at that time. The intralenticular dexamethasone implant was clearly imaged...
by high-frequency ultrasound biomicroscopy using the HiScan system (Optikon 2000 SpA) in front of the posterior lens capsule (Figure 2, B).

The patient was followed without repositioning the implant. During the 6-month follow-up, the anterior segment of the right eye remained quiet, with no progression of cataract; the implant was still seen in the crystalline lens (Figure 3). The IOP remained within normal limits. By 6 months, the CDVA in the right eye had improved to 0.2 logMAR (decimal equivalent of 0.63) and the macular thickness had decreased to 352 μm (Figure 4).

**DISCUSSION**

Endophthalmitis, uveitis, hemorrhage, elevated IOP, vitreous incarceration, retinal tears, cataract formation, lens dislocation, zonular tears, and lens touch are reported complications of intravitreal injections.7,8 Although there are some anecdotal reports of iatrogenic lens damage, the exact incidence rate is unknown.9 In several large safety surveys, the rate of lens injury during intravitreal injection varied.10,11 Meyer et al.9 reported the rate as 0.0009% in phakic eyes.

Three cases in the literature report inadvertent injection of triamcinolone in eyes with age-related macular degeneration12 and diabetic macular edema.13,14 Jalil et al.12 operated on the eye 4 weeks after the injection because the lens remained opaque and fundus assessment was not possible. Rajak et al.13 reported that the lens did not opacify until 7 months after the procedure and then, although the cataract had progressed, the patient did not have cataract extraction as the IOP was elevated to 42 mm Hg. Kumar et al.14 found that the patient had developed a cataract and elevated IOP 3 days after the procedure. In these cases, 0.1 mL triamcinolone acetonide (40 mg/mL) was injected into the vitreous cavity with a 1 mL syringe with a 27-gauge needle. In our case, the dexamethasone implant (0.7 mg) was injected intravitreally with the same method through the pars plana but with a single-use 22-gauge applicator system.

Complications of intravitreal dexamethasone implant have been reported as case reports or small case series.15-17 The main complication in these reports was anterior chamber migration of the implant.
In our case, because the applicator needle was not perpendicular to the vitreous cavity, the needle was advanced into the lens and the dexamethasone implant was injected into the crystalline lens. This is a rare complication of intravitreal injections. Koller et al. followed a patient with inadvertent injection into the crystalline lens and retinal vein occlusion up to 11 months without cataract surgery or repositioning the dexamethasone implant. Posterior subcapsular cataract was observed in the fifth month after the injection and progressed promptly. Eleven months after the injection, the patient's vision decreased to hand motion and they performed cataract surgery with intraocular lens (IOL) implantation. During this time, they had to inject another dexamethasone implant because of resistant macular edema. Munteanu and Rosca reported an inadvertent injection of the dexamethasone implant into the crystalline lens in a patient with noninfectious posterior uveitis. The authors performed cataract surgery with IOL implantation and repositioned the implant into the vitreous cavity in the same session. In our patient, the CDVA improved (from 1.0 logMAR to 0.2 logMAR) and macular thickness decreased (from 579 to 352 µm) in the sixth month after the injection even though the position of the implant was incorrect. Also, we did not observe any progression of cataract formation during the follow-up.

In cases of inadvertent injection into the crystalline lens, patients should be observed closely and followed for development of cataract or elevation in IOP. In the absence of such complications, patient might be followed with a further surgical procedure such as repositioning.

Ophthalmologists should be aware of this rare complication. During the intravitreal injection, the direction of the needle should always be perpendicular to the scleral surface to prevent this complication.

To our knowledge, this is the first report of a patient who had a decrease in macular thickness despite intralenticular localization of dexamethasone implant without any side effect.

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Figure 4. The OCT examination showed that macular thickness had decreased to 352 µm by 6 months after the injection.
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First author: Aylin Karalezli, MD
Department of Ophthalmology, Baskent University Faculty of Medicine, Konya, Turkey