

Intralenticular Dexamethasone: A Case Report

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ABSTRACT

Dexamethasone implant is frequently used for macular edema in diabetic retinopathy. It has long-term efficiency; however some problems may be encountered during application. We are reporting a case of a 56 year-old-woman patient with macular edema secondary to diabetes mellitus. A dexamethasone implant was inadvertently injected into the crystalline lens. The patient was followed without the surgical intervention of the implant for six months. In the follow-up time, visual acuity increased, and macular edema was decreased significantly. In the sixth month, the patient had uncomplicated cataract surgery.

Keywords: Dexamethasone, Intralenticular, Diabetic macular edema, Cataract surgery.

INTRODUCTION

The dexamethasone implant (Ozurdex, Allergan, Inc., Irvine, Calif., USA) is indicated in the management of diabetic macular edema, macular edema following branch retinal vein occlusion or central retinal vein occlusion and non-infectious posterior uveitis. The most common adverse ocular reactions of Ozurdex are increased intraocular pressure (IOP) and the onset or progression of cataract. The ZERO study (designed to test the safety and efficacy of Ozurdex intravitreal injections) did not report any intra-operative lens injuries. However, in the literature, there are reports of accidental injection of Ozurdex into the crystalline lens.¹⁻¹¹ This could be the result of the surgeon's lack of experience, the use of an improper surgical technique or an uncontrolled movement of the patient's head during the procedure². Here we present the case of an accidental intralenticular injection of Ozurdex in a patient with diabetic macular edema and the management of this complication.

CASE REPORT

Our patient is a 56-year-old female with macular edema due to proliferative diabetic retinopathy. Her best-corrected

visual acuity (BCVA) (in Snellen) was 0.05 in her right eye and 0.15 in her left eye. Intraocular pressure (IOP) was 14 mmHg Appl. At the slit-lamp examination, the patient was bilaterally phakic and there was no cataract formation. In the right eye, there was vitreous hemorrhage. Central macular thickness (CMT) was 611 µm in the left eye. She had been treated several anti-VEGF injections previously. It was decided to perform a dexamethasone implant for diabetic macular edema (DME). The implant was injected 4.0 mm from the limbus under topical anesthesia with proparacaine. During the procedure, the patient had sudden head movement, and intravitreal dexamethasone implant entered into the lens through its equator. On the first day, there was no reaction, and slit-lamp examination revealed that the dexamethasone implant was located in the crystalline lens. (Figure 1) IOP was normal. One week later, BCVA was 0.15, and the OCT showed that the macular edema decreased. The patient was monitored closely for complications related to Ozurdex. One month later, a slit lamp examination showed the anterior chamber was normal, there was no inflammation, and no cataract had developed. The patient had no apparent visual complaints other than whiteness in the form of a line due to the implant. The BCVA was 0.3, and the IOP was 16 mmHg Appl. Considering the patient's vision had improved, the macular

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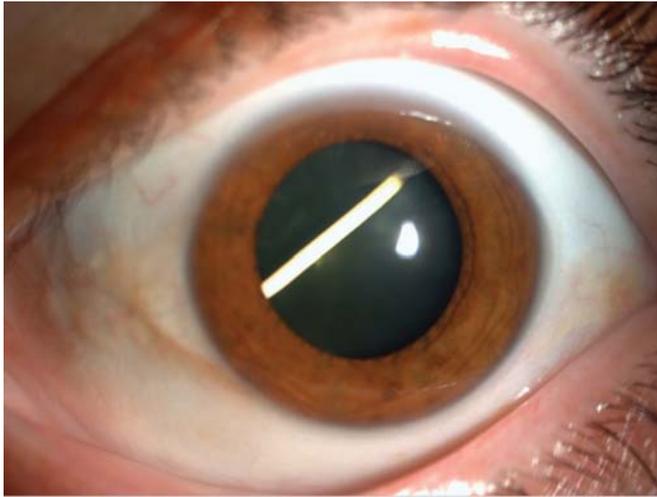


Figure 1: The implant located in the crystalline lens one day later.

edema was regressed, and IOP values were normal, we decided to follow up without surgical intervention.

At 3rd month: the patient was noncompliant. The BCVA was 0.3, and the anterior chamber was quiet, there was no inflammation, there was no cataract development. The IOP was 17 mmHgAppl. OCT showed the improvement of macular edema. Five months following Ozurdex injection; the BCVA was 0.5, there was no inflammation, no signs of implant degeneration, cataract formation. The IOP was 21 mmHgAppl. OCT findings showed a remarkable improvement in edema. (Figure 2)

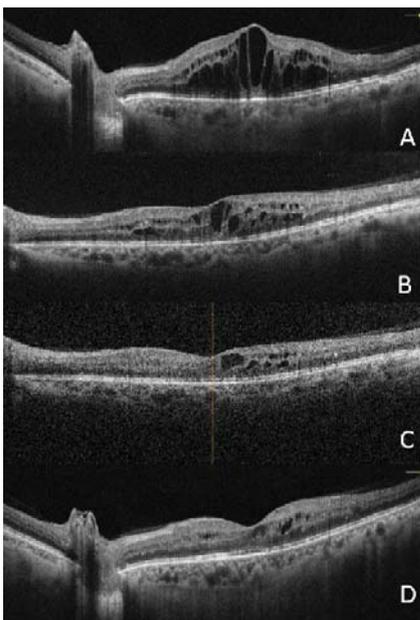


Figure 2: a. SD-OCT of the macular edema prior to Ozurdex injection. (CMT:611 μ m), b. OCT 1 month following ozudex administration, showing resolution of the macular edema, c. OCT 3 months following injection, d. OCT 5 months following injection.

The patient underwent phacoemulsification surgery at 6th months after the injection. The implant was into the nucleus, so standard phacoemulsification surgery was performed. (Figure 3) A gentle hydrodissection-hydrodelineation was performed, and low phaco parameters were used during the surgery of the cataract. Cortex aspiration was performed carefully, preventing zonular fibers. No capsular defect was noted intraoperatively, and no vitreous was encountered. The posterior chamber IOL was placed in the bag. Topical antibiotics and steroids were prescribed. One week later, BCVA was 0.5, IOP was 13 mmHgAppl. There was no macular edema. In the first month, the BCVA was 0.2, and there was intraretinal and subretinal edema on OCT. A new dexamethasone implant was injected through pars plana. One month later, the BCVA was 8/10, and the IOP was 14 mmHgAppl. The macular edema was resolved.

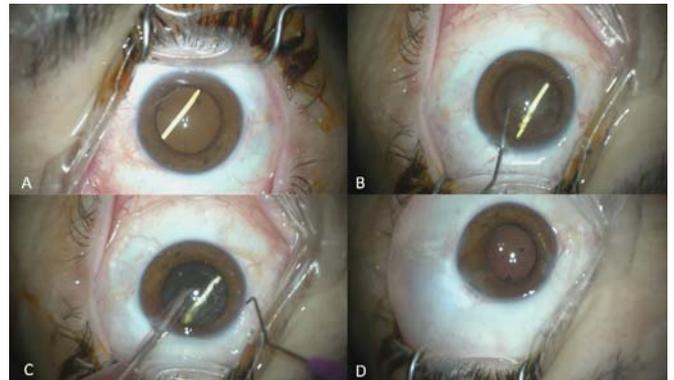


Figure 3: a. Intralenticular ozurdex implant, b. Hydrodissection and hydrodelineation, c. Creating a Groove with phaco probe, d. Intact posterior capsule and intraocular lens implantation

CONCLUSION

There are studies reporting that dexamethasone implant is effective with anti-inflammatory effects in the treatment of resistant DME in which anti-VEGF drugs are ineffective.^{12,13} Inadvertent injection of sustained-release intravitreal dexamethasone implant into the crystalline lens is an uncommon but possible complication. This complication should be borne in mind during the intravitreal injection.

The therapeutic effect of intralenticular Ozurdex can be questionable since the implant was delivered into the capsular bag, and the lens substance is not directly exposed to aqueous or blood circulation that might serve as a clearance route.¹

The literature reveals previous cases of inadvertent intralenticular administration of Ozurdex. Some authors,^{2,3,6,7,9} feel expectant waiting is better, while others suggest immediate phacoemulsification for the cataract.^{1,5,11}

Valvende-Megias et al. reported a case with an intralenticular dexamethasone implant. They follow up with the patient for twenty months without additional treatment.¹⁴ Rogan et al. reported a case of intralenticular sustained-release dexamethasone implant present for one year with effective treatment of refractory DME without rapid cataract formation.² Clemento-Thomas reported a case with branch retinal vein occlusion. They demonstrated that the therapeutic effect of the implant is continued without complication during an 18-month follow-up.⁹

Although the exact mechanism is unknown, the kinetics of the dexamethasone implant inside the lens differs from those in the vitreous humor. In our case, the implant showed no signs of degradation at five months. Valvende-Megias et al. stated that after 20 months, there was no degradation in the implant.¹⁴ Clemento-Thomas reported the implant in the lens presented no signs of degradation at five months, and they observed slowly progressing decomposition on the outer edge of the device that started to accelerate at 14 months post-implant injection.⁹

In cases of accidental injection of the dexamethasone implant in the crystalline lens, cataract formation followed by removal of the lens has been reported.⁴ Inefficacy of treatment in these cases has been attributed to the insufficient release of the drug due to the different biochemical properties of the lens with respect to the vitreous.⁴

Baskan et al. reported that Ozurdex totally located inside the crystalline lens might not have therapeutic effects.¹⁰ In our case, the implant was completely into the lens, and the capsule was intact. Despite its intralenticular location, the implant may still improve or resolve macular edema. Since the lack of capsule contact postpones the development of cataract, it may be possible to benefit from the effect of the ozurdex implant for a longer time in these patients. In our case, at the 6th month, cataract did not develop and macular edema did not increase, but our patient underwent phacoemulsification surgery after she stated that she was uncomfortable with the white reflux of the implant. Macular edema was observed in the 1st month after cataract surgery. The inflammatory response caused by the surgery and uncontrolled diabetes may have contributed to the increase in macular edema.

Once this complication occurs, early phacoemulsification and repositioning of the implant into the vitreous is the frequently preferred management strategy. However, a remarkable decrease in macular edema and visual acuity improvement can also be achieved without immediate surgical intervention. If there is no increase in IOP that

cannot be controlled by topical treatment, and if the capsule has not contacted and did not develop cataract, it may be an option to closely monitor the development. Prompt surgery is not required.

Conflict of Interest: The Author(s) declare(s) that there is no conflict of interest

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