Dexamethasone Implant Migration Into Anterior

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ABSTRACT

Here, we reported clinical findings, treatment process and complications in a case with the diagnosis of Irvine-Gass syndrome in which intravitreal dexamethasone implant migrated into anterior chamber.

Dexamethasone implant (OZURDEX, Allergan) is a safe, effective and easily applicable treatment method which is widely used in the treatment of macular edema. The implant is injected via intravitreal route under topical anesthesia. The implant may migrate to anterior chamber. A 66-years old patient underwent pars plana vitrectomy plus IOL implantation with scleral fixation for nucleus drop and was diagnosed with Irvine-Gass syndrome during follow-up. The patient was treated with intravitreal dexamethasone implant injection. However, the patient presented with blurred vision 3 weeks after implantation. It was found that the dexamethasone implant migrated to anterior chamber and that there was corneal edema. The implant was removed via surgery. Early diagnosis and timely intervention prevented development of corneal bullous changes in the patient.

Keywords: Aphakic, Dexamethasone implant, Migration into anterior chamber.

INTRODUCTION

Dexamethasone implant (OZURDEX, Allergan Inc. Irvine, CA, ABD) is a rod-shaped, biodegradable, sustained-release dexamethasone implant (6 mm in length and 0.46 mm in diameter) which is injected into intravitreal space using 22 G needle.[1] It contains 0.7 mg dexamethasone polymer without any preservative and the polymer is degraded into lactic acid and glycolic acid. It is used in the treatment of diabetic maculopathy, non-infectious uveitis and macular edema secondary to Irvine-Gass syndrome. Subconjunctival hemorrhage during implantation is the most common complication of the dexamethasone implant. Other complications include glaucoma, cataract, lens implantation, endothelial damage, corneal edema, intravitreal hemorrhage, macular hole, retinal tear, retinal detachment, endophthalmitis and migration into anterior chamber in eyes without posterior capsule support.[2-4]

Here, we reported clinical findings, treatment process and complications in a patient presented with implant migration into anterior chamber following dexamethasone implant injection.

CASE REPORT

A 66-years old woman was referred to our clinic with nucleus drop and intravitreal nucleus fragments were removed by pars plana vitrectomy; in addition IOL implantation (Poly Methyl Acrylate [PMMA] lens) with scleral fixation was performed in the same session.

At postoperative month 1, visual acuity and intraocular pressure (IOP) were measured as 0.7 and 12 mmHg in the left eye, respectively. Biomicroscopy and fundoscopy were found to be normal and there was no abnormal finding on optical coherence tomography (OCT). At postoperative month 3, the patient suffered from impaired vision in the left eye; the visual acuity was 0.1 and IOP was 16 mmHg in the left eye. In biomicroscopy, it was found that cornea was transparent and intraocular lens was centralized. In fundoscopy, it was observed that there was macular thickening in the left eye. On OCT, subretinal and intraretinal fluid was detected in the left eye (Figure 1A). It was decided to perform intravitreal dexamethasone injection with diagnosis of Irvine-Gass syndrome.

The patient presented with blurred vision in the left eye 3 weeks after dexamethasone implant injection. In the

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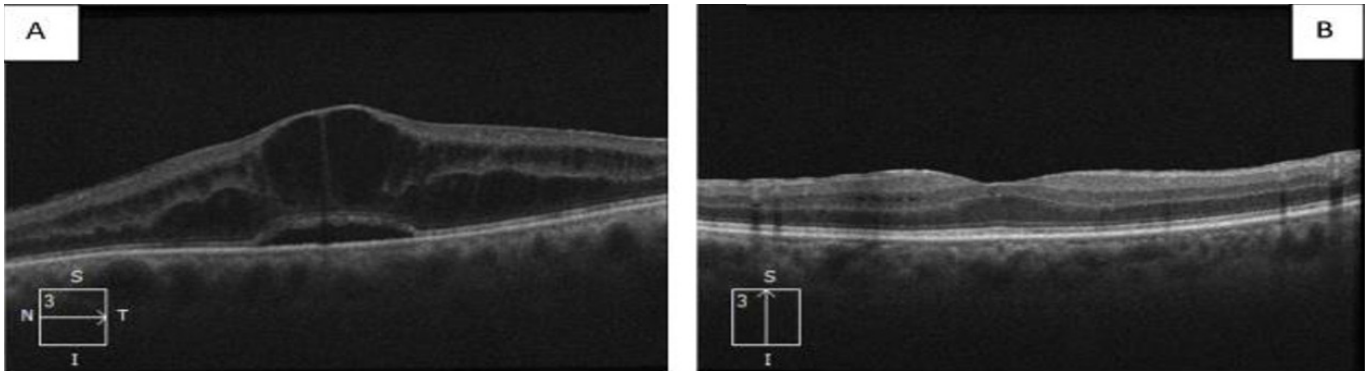

Figure 1. *Optical coherence tomography (OCT) A; subretinal and intraretinal edema, B; edema was regressed after dexamethasone.*

ophthalmological examination, visual acuity was 0.05 and IOP was 18 mmHg in the left eye. In biomicroscopy, corneal edema and descemet folds were detected in the left eye while intraocular lens was centralized. However, it was observed that dexamethasone implant migrated into anterior chamber and localized to iridocorneal angle at inferior segment (4-6 O'clock direction) (Figure 2). No abnormal finding was detected in fundoscopy. There was no macular edema on OCT (Figure 1B).

The corneal edema observed was attributed to mechanical effect of implant in anterior chamber and it was decided to remove implant. Thus, the patient was admitted to hospital. To prevent displacement into vitreous, miosis was achieved using pilocarpine and the patient was placed into head position. Following topical anesthesia with proparacaine HCl, transparent corneal incisions were made using MVR blade at 3 7 O'clock directions. Implant was removed using micro-forceps and spatula. The incision site was closed with stromal hydration. In the same session, intravitreal triamcinolone acetonide (IVTA; 4 mg/0.1 cc) was injected to the eye. Topical dexamethasone eye drop (6x1) and moxifloxacin eye drop (4x1) were prescribed for one week. In addition, Sodium Chloride (hypertonic ophthalmic solution, 6x1) was given for corneal edema.

Due to persistent corneal edema, hypertonic ophthalmic solution (4x1) was maintained for 3 months. Two IVTA injections (4 mg/0.1 cc) were performed at month 6 and 12 after ex-plantation (Figure 3). In final control visit, visual acuity was 0.4 in the left eye with transparent cornea and centralized intraocular lens. The IOP was measured as 12 mmHg and mild epiretinal membrane formation was observed on fundoscopy. There was mild epiretinal membrane on OCT but no edema was detected.

DISCUSSION

Here, we presented treatment of corneal edema caused by intravitreal dexamethasone implant migration into anterior chamber.

In the literature, there are case reports and series reporting migration of dexamethasone implant into anterior chamber.[5] Goncalves et al. retrospectively reviewed 486 patients underwent dexamethasone implant injection more than one center and reported prevalence of implant migration into anterior chamber as 1.6%.[6] Dexamethasone implant migration into anterior chamber was first described in a patient having IOL with iris fixation.

It was reported that highest risk for implant migration into anterior chamber was within 1-3 week after injection of the

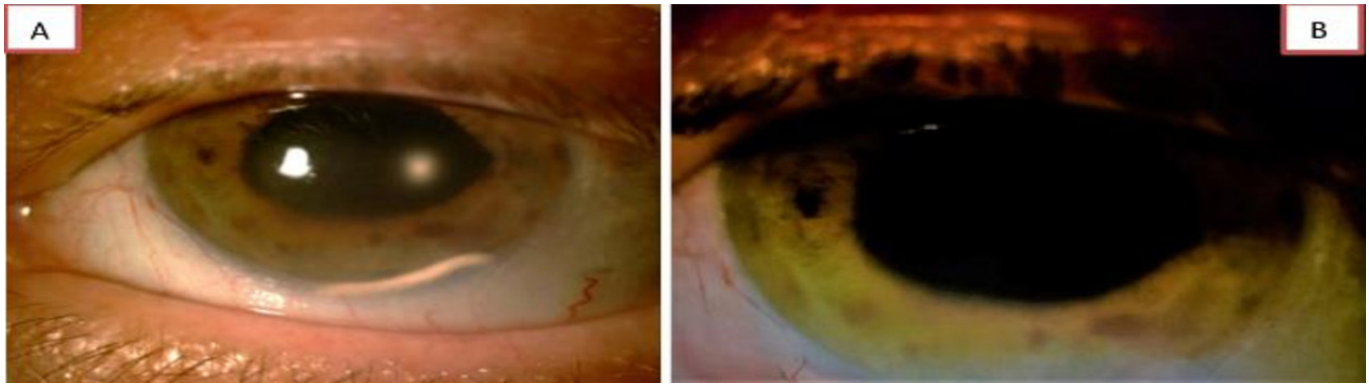

Figure 2. Dexamethasone implant in anterior chamber.

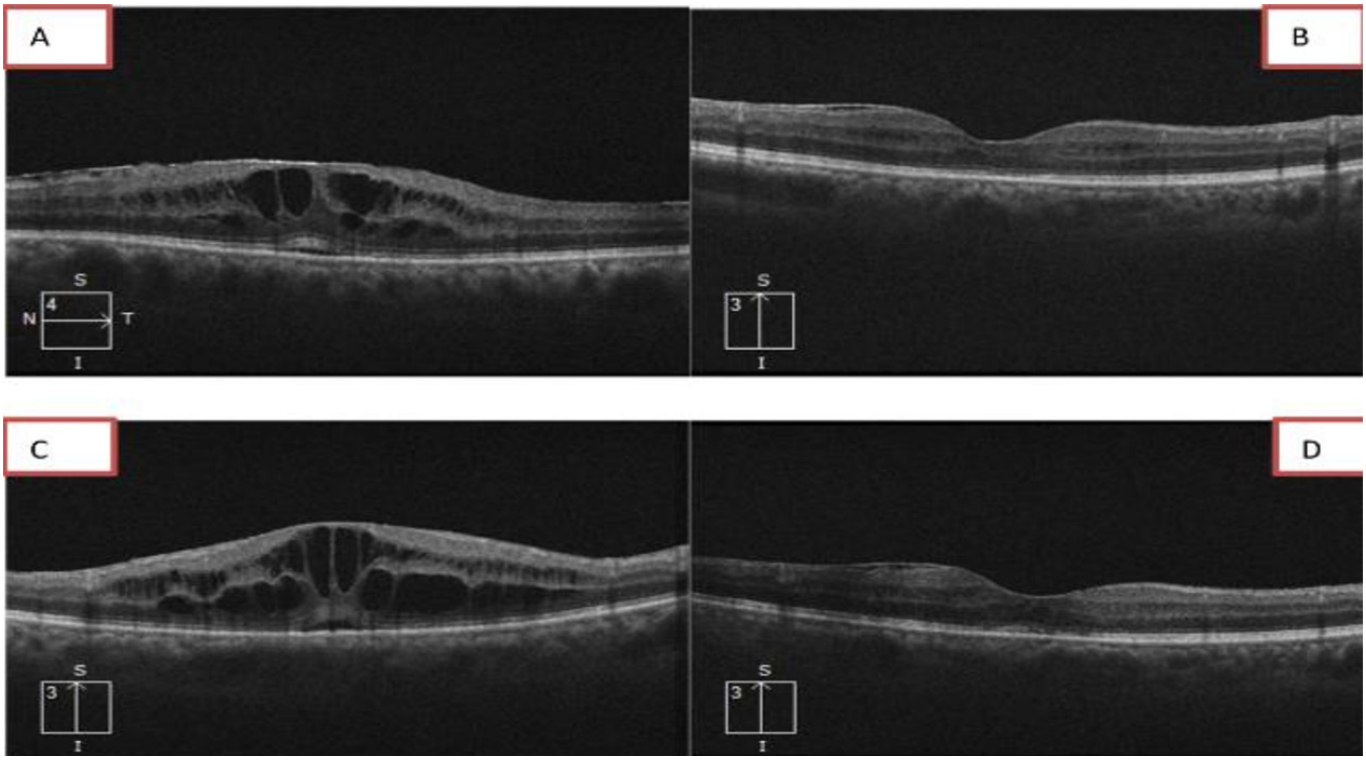

Figure 3. Before and after intravitreal triamcinolone acetonide

implant. In the same study, non-intact posterior capsule, vitrectomized eye and presence of zonule defect were reported as risk factors. It was recommended not to use the dexamethasone implant in patients with risk factors or to avoid prone position at early period [5].

In our patient, dexamethasone implant migrated into anterior chamber 3 weeks after implant injection. Corneal edema and descemet folds were major complication of implant migration. It has been proposed that corneal edema may be due to direct contact with corneal endothelium or chemical toxicity related to high-dose steroid released from implant. It is well-known that high-dose dexamethasone leads apoptosis and necrosis in the corneal endothelium [7]. Prolonged corneal edema may result in bullous keratopathy requiring keratoplasty. In our case, refractory corneal edema was developed and corneal transparency was recovered following treatment with hypertonic ophthalmic solution over 3 months. In the literature, it was reported that migration into anterior chamber 5 weeks after implant injection did not lead corneal edema.

In the management, the first step is to attempt to move implant towards pupillary space by kindly tapping glob inferiorly at supine position after pupil dilatation; then, between iris and GIL, and finally into vitreous by placing patient into erected position. If the maneuver fails, surgical ex-plantation is recommended in operating room [8]. It is also recommended to fragment implant into small pieces with YAG laser fragmentation if it is localized between iris and GIL [9]. In our study, implant migrated into anterior chamber despite recommendations regarding positioning, centralized GIL and lack of pupillary dilatation or deformation. Thus, implant ex-plantation was preferred.

Dexamethasone implant migration into anterior chamber is rare but possible. The risk factors for implant migration into anterior chamber include posterior capsule defect, previous history of anterior vitrectomy or pars plana vitrectomy, mydriasis, GIL implantation with scleral fixation of iris fixation and presence of zonule defects. In addition, implant migration into anterior chamber through iridectomy was also reported [10]. In such cases, it should kept in mind that implant may migrate into anterior chamber and benefit: risk balance should be taken into account, considering alternative therapies. It is important to prevent corneal bullous changes by early and timely intervention and to ensure ocular health without need for keratoplasty.

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