Endothelial keratoplasty for corneal decompensation leaded by a dexamethasone implant dislocation in anterior chamber

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Abstract

Background: Dexamethasone intravitreal implant (DEX) largely showed his safety and efficacy for the treatment of cases of macular edema. Even if uncommon, delivery dislocation in anterior chamber has been described in Literature as complication of the injection procedure, leading to irreversible endothelial cell loss in the majority of cases.

We report a case of a 66-year-old man with pain and vision loss in his left eye. The anamnesis revealed a recent intravitreal injection of DEX implant for a persistent cystoid macular edema related to central retinal vein occlusion. Anterior segment examination showed corneal edema and the rod implant adherent to corneal endothelium. A large peripheral iridectomy was evident with retroillumination and IOL appeared good centered in the bag. The implant was removed but corneal decompensation was irreversible. One month later, an endothelial keratoplasty was successfully performed restoring corneal transparency.

DEX intravitreal implant can migrate from vitreous cavity to anterior chamber and lead to irreversible corneal decompensation by mechanical and chemical toxicity on corneal endothelium. Removal of the implant is necessary to avoid total endothelial decompensation. Despite this, in some cases endothelial keratoplasty had to be performed.

Keywords: Macular edema; intravitreal dexamethasone implant; side-effects; corneal; DSEK.

Case report

Ozurdex® (Allergan Inc., Irvine, CA, USA) is a biodegradable intravitreal slow release delivery largely used for the management of macular edema due various pathological conditions such diabetes [1] retinal vein trombosis [2] Irvine-Gass syndrome [3] and noninfecctious uveitis [4]. Anterior chamber dislocation of DEX implant was described as rare complication of the intravitreal injection [5].

Several cases had to be managed by surgical removal of the implant in presence of corneal endothelial decompensation or anterior complications [6]. However, repositioning of the implant into the vitreal cavity was described as efficient and safe procedure when anterior chamber migration does not generate corneal decompensation [7].

In this paper, we describe the case of a Descemet stripping endothelial keratoplasty in a patient who
suffered of corneal endothelial decompensation leaded by antherior chamber migration of an intravitreal Ozurdex implant.

A 66-year-old man with a history of recent dexamethasone intravitreal implant injection presented with pain and progressive loss of vision in his left eye. The patient reported previous diagnosis of cystoid macular oedema due to central retinal vein occlusion (CRVO) refractory to anti-VEGF therapy in his left eye. For this reason he was scheduled for DEX intravitreal injection in the left eye. He also reported phacoemulsification with intraocular lens implantation in both eyes two years before the diagnosis of CRVO. Slit lamp examination of his left eye showed marked pericheratic injection, total corneal decompensation and the steroid implant adherent to corneal endothelium (Figure 1).

Figure 1. Ozurdex dislocated in anterior chamber

Although the rear structures were poorly evaluated, a large peripheral iridectomy was evident with retroillumination and IOL appeared in position. Best spectacle corrected visual acuity (BSCVA) was hand motion. Intraocular pressure (IOP) was 28 mmHg.

Because of the incipient corneal decompensation and the high intraocular pressure, he was scheduled for removal of the implant from the anterior chamber, after administration of 500 cc of mannitol to 18% in solution, in order to lower the IOP. The removing was performed using the same procedure described by Stelton et al. [8].

Topical 5% hypertonic salted solution and 0.2% Betamethasone associated with 0.5% Chloramphenicol eyedrops was administered 4 times a day for one month.

One month after surgery, BSCVA was hand motion and slit lamp biomicroscopy showed the persistence of corneal oedema, leaded by the irreversible endothelial cell loss caused by the dislocated implant. Endothelial cell count was 450 cell/mm².

In view of this scenario, we decided to perform Descemet stripping endothelial keratoplasty (DSEK) to restore corneal transparency. DSEK was performed through a 4 mm temporal corneal incision. An anterior chamber cannula was inserted for paracentesis. Descemet membrane of the recipient was stripped under air infusion from the posterior corneal stroma over a region corresponding to the dimensions of the graft. The existing peripheral iridectomy helped to avoid postoperative pupillary block. The graft was inserted into the eye through the 4 mm corneal incision using a Busin glide (Asico, Westmont, IL). Then, the corneal incision was closed with 2 to 3 interrupted 10-0 nylon sutures and a complete air filling was performed.

Fluid that accumulated between the recipient’s stroma and the graft was drained through small incisions made in the midperipheral cornea of the recipient. Following the procedure, the patient received a systemic dose of 4 mg betamethasone and topical application of 0.3% gatifloxacin and 0.1% betamethasone eyedrops 4 times daily, tapering the drops during 3 months.

At 1 month follow up, BSCVA was 0.53 logMar. Corneal edema was almost completely resolved and corneal endothelial cell count was 2100 cell/mm² (Figure 2).

Figure 2. One month after DSEK

Discussion

DEX intravitreal implant can migrate from vitreous cavity to anterior chamber.

Prior vitrectomy, disrupted posterior capsule, previous iridectomy, aphakic lens status, face down position are known risk factors for the anterior chamber migration [9], [10].

Corneal decompensation is the most important
complication which can occur in this case.

Dexamethasone rod resulted toxic for corneal endothelium with two possible mechanisms. First, the mechanical trauma due to the rod, especially during the first month, when the implant is more rigid [11]. Secondly, a chemical toxicity as from DEX, which induces both apoptosis and necrosis to corneal endothelial cells, as other components of the implant (lactic acid or glycolic acid) [12]. Surgery is necessary in the majority of cases for successful removing the implant, but often corneal edema resulted irreversible.

In this cases, endothelial keratoplasty have to be performed to restore corneal transparency and to obtain a visual recovery.

References