Effectiveness of Aflibercept in a Case of TASS-Associated Macular Edema

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ABSTRACT

Toxic anterior segment syndrome (TASS) is an acute, non-infectious postoperative inflammatory reaction of the anterior segment of the eye, that usually develops 12-48 hours after intraocular surgery. Mostly, the inflammation is resolved with appropriate treatment within days or weeks, with a good outcome; however, infrequently complications such as permanent corneal edema, secondary glaucoma, and chronic macular edema can occur jeopardizing the visual outcome. The aim of this study is to present the clinical outcomes of intravitreal aflibercept injection in a case with macular edema associated with TASS.

Keywords: Macular edema, Toxic anterior segment syndrome.

INTRODUCTION

Toxic anterior segment syndrome (TASS) was first described by Monson at al. as an acute, noninfectious postoperative inflammatory reaction of the anterior segment of the eye developing within 12-24 hours after intraocular particularly cataract surgery. The typical clinical signs are limbus-to-limbus corneal edema, fibrin in the anterior chamber and an irregular or fixed dilated pupil that does not respond to light. Anterior vitreous opacities can rarely be seen. Ophthalmic instrument cleaning and sterilization processes and the products used in the surgery were most commonly reported to be associated with TASS. The main treatment of TASS includes intense regimen of topical steroids. Subconjunctival steroid injection can be used for corneal edema that cannot be resolved with topical steroids. In most cases, the inflammation is resolved with appropriate treatment within days or weeks and has a good outcome; but in some cases, complications such as permanent corneal edema, secondary glaucoma, and chronic macular edema can occur resulting in decreased vision. The aim of this study is to report the clinical features and treatment outcome in a patient presenting with resistant macular edema associated with TASS.

CASE PRESENTATION

A 70-year-old Caucasian female was referred to our hospital with the diagnosis of TASS. The patient had undergone an uncomplicated phacoemulsification surgery in another clinic two weeks ago. She complained of decreased vision in the right eye from the first postoperative day and her visual acuity and corneal edema did not improve despite the following intensive medical treatment: dexamethasone (q1hr), moxifloxacin HCl (QID) eye drops for two weeks. She did not have any history of systemic diseases. Her best corrected visual acuity (BCVA) was hand motions in the right eye. The IOP was 15 mmHg under medication (fixed combination of topical dorzolamide HCl/timolol maleate (BID) and oral acetazolamide (QID)). Slit-lamp examination of the right eye revealed severe corneal edema extending from limbus to limbus, which obscured the anterior segment details. Anterior vitreous opacities can rarely be seen. Slit-lamp examination of the right eye revealed severe corneal edema extending from limbus to limbus, which obscured the anterior segment details. Diffuse corneal edema was depicted by anterior segment-optical coherence tomography (AS-OCT) (Optovue RTVue 100-2, Fremont, CA); central corneal thickness was 775 µm (Figure 1A). USG scan revealed no sign of vitreous inflammation. The patient's treatment regimen was rearranged. Moxifloxacin and dexamethasone were discontinued, and hourly prednisolone acetate eye drops (Pred-forte 1%; Allergan,
Irvine, CA, USA), topical NaCl 5% ophthalmic solution (TID), and artificial tear drops without preservatives (Artelac Complete, Bausch& Lomb, Berlin, Germany) (QID) were prescribed. The corneal edema gradually resolved in four weeks and the anterior chamber was silent. While CCT decreased to 558 µm in AS-OCT (Figure 1B), the patient’s BCVA increased to 20/200 in the postoperative 6th week. The IOP was 18 mmHg under medication (fixed combination of topical dorzolamide HCl/timolol maleate). The fundus examination of the right eye revealed a small hypopigmented spot in the temporal of the macula and absent of foveal reflex (Figure 2 A). There were no inflammatory cells in the vitreous. Spectral-domain optical coherence tomography (SD-OCT) (Optovue RTVue 100-2, Fremont, CA) revealed macular edema (ME) and the central macular thickness (CMT) was 346µm (Figure 3 A). The fluorescein angiography (FA) of the right eye revealed mild hyperfluorescence in the early phase starting in the posterior pole (Figure 2 B) and cystoid ME with staining in the nasal aspect of the optic disc in the late phase (Figure 2 C). There was no sign of vasculitis. The left eye revealed normal angiographic features (Figure 2 D). Topical ketorolac tromethamine 0.5% solution (Acular; Pharm Allergan) (QID) was added and topical

**Figure 1:** Anterior-segment SD-OCT scan of the right eye. A) OCT image demonstrated severe corneal edema at initial presentation; B) Complete resolution of corneal edema after treatment.

**SD-OCT:** Spectral domain optical coherence tomography

**Figure 2:** A) Color fundus photo of the right eye, B) FA of the right eye, early-phase, C) late-phase D) FA of the left eye.
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NaCl drops were discontinued. In addition, a sub-Tenon’s injection of triamcinolone acetonide was performed. At the visit six weeks later, which was the 3rd month following cataract surgery, macular edema was increased, SD-OCT revealed a CMT of 419 μm (Figure 3 B). Since the ME was resistant to treatment, intravitreal aflibercept (Eylea; Bayer HealthCare, Berlin, Germany) injection was recommended. Intravitreal anti-VEGF therapy was preferred instead of intravitreal corticosteroid due to risk of intraocular pressure elevation. After obtaining an informed consent, she was treated with three consecutive monthly intravitreal injections of aflibercept (2 mg/0.5 mL). Following after the third injection BCVA increased to 20/30; the SD-OCT scan showed resolution of ME and CMT decreased to 283μm at the postoperative 6th-month visit. However, there was a small intraretinal cyst with a slight deterioration in the ellipsoid zone (Figure 3 C). No side effects were seen or no recurrence was observed during the one-year follow-up period. Her BCVA was 20/30 and CMT was 277 μm (Figure 3 D).

DISCUSSION

The incidence of TASS following phacoemulsification surgery was reported to be 0.1-2%. Vision-threatening complications such as permanent corneal edema, secondary glaucoma and chronic macular edema can be seen as the consequences of TASS.

Based on the literature review, macular edema secondary to TASS is a rarely seen condition and is considerably caused by a mechanism triggered by the severe inflammation, as in Irvine-Gass syndrome (pseudophakic cystoid ME). Several potential mechanisms were concerned in the pathogenesis Irvine-Gass syndrome. However, release of inflammatory mediators (such as prostaglandins, complement components, cytokines, and VEGF), increased retinal capillary permeability and disruption of the blood-retina barrier appear to be the major factors. The ME was reported to resolve spontaneously in most of the cases. Topical non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids are commonly used in the first-line treatment of Irvine-Gass; however, refractory ME cases that did not respond to therapy were reported.

Although there is no standard treatment regimen for the management of refractory ME cases, periocular and/or intravitreal corticosteroids and anti-VEGF treatment have been shown to be effective. On the other hand, intravitreal steroid injections bear the risk of intraocular pressure rise. To the best of our knowledge, there is a small number of TASS-associated macular edema cases reported in the current literature. Data regarding treatment was available for only one of these cases. Ugurbas et al reported that the patient responded well to intravitreal triamcinolone injection.

In this current case, ME developed due to severe inflammation was unresponsive to previous treatment with
NSAIDs and topical and periocular steroids. Therefore, it was decided to administer intravitreal injection; corticosteroids were avoided due to a risk of intraocular pressure rise. Studies showed that anti-VEGF agents are useful in the treatment of macular edema secondary to various retinal diseases. The significant improvement in BCVA was observed after three consecutive monthly injections. The CMT decreased from 419 μm at baseline to 277 μm. In the follow-up, permanent regression of CME and increased visual acuity were achieved without additional treatment. OCT provides important information about retinal microstructure change as well as macular thickness. The chronicity and severity of CME might cause permanent changes in the photoreceptor layer, and therefore low visual acuity. Previous studies showed that the structural changes in the photoreceptor layer (particularly photoreceptor inner and outer segments -ellipsoid zone-) and the external limiting membrane is helpful in predicting the final visual outcome in macular diseases. Hunter et al. reported that persistent anatomic alteration of photoreceptors defined by OCT correlated with reduced BCVA in patients with pseudophakic CME. In this case report, the patient's BCVA did not return to 20/20 despite resolution of the CME. It was thought that disruption to ellipsoid zone might have a negative effect on visual acuity.

CONCLUSION

Based on the review of the literature, this is the first case that TASS-related macular edema which was treated with intravitreal aflibercept injection. In conclusion, intravitreal injection of aflibercept was found to be an effective and safe therapy for the treatment of ME associated with TASS. It may be a valuable, alternative treatment of refractory ME especially in patients who have the risk of IOP rise. However further studies are needed to confirm these findings.

REFERENCES