CASE REPORT

The Management of A Patient with Steroid-Induced Intraocular Pressure Elevation by Subtenon Triamcinolone Acetonide Deposit Excision

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ABSTRACT

Subtenon triamcinolone acetonide (TA) injection, one of the periocular injection methods, is effective in the treatment of ocular inflammatory diseases and uveitic macular edema. However, after subtenon TA injection, significant ocular complications such as intraocular pressure (IOP) elevation, cataracts, and upper eyelid ptosis may develop. Patients who develop elevated IOP may require medical and surgical treatment to control IOP. Surgical excision of subtenon deposits has been described as an effective method with a low side effect profile in treating refractory elevated IOP after subtenon TA. Here, we present the case of a 34-year-old female patient who had subtenon TA injection for uveitic macular edema and subsequently developed elevated IOP that could not be controlled with maximum medical therapy. Following surgical removal of the subtenon TA deposits, the patient experienced a decrease in IOP. This report underscores the potential utility of subtenon TA deposit excision in the treatment of steroid-induced ocular hypertension refractory to medical therapy after posterior subtenon TA injection.

Keywords: Ocular hypertension; triamcinolone acetonide; uveitis; macular edema; subtenon injection

INTRODUCTION

Subtenon steroid injections are periocular injection techniques widely used for treating ocular inflammatory diseases.[1] Triamcinolone acetonide (TA) is frequently preferred for subtenon injections due to its water-soluble nature and its ability to deliver long-lasting intraocular steroid effects.[1,2] Posterior subtenon TA (PSTA) injection allows the drug to reach higher concentrations in the posterior segment through transscleral absorption compared to systemic administration.[2] Numerous studies have demonstrated that PSTA injection is effective in managing intraocular inflammation and treating uveitic macular edema in patients with non-infectious uveitis (NIU).[1,3-5] However, serious ocular complications such as elevated intraocular pressure (IOP), cataracts, upper eyelid ptosis, orbital fat prolapse, orbital infections, conjunctival ischemia or ulceration, capsular subtenon TA cysts, and globe perforation may occur after PSTA application.[2,5-7]

Long-term steroid exposure of the trabecular meshwork following posterior subtenon TA injection carries a risk of elevated IOP, particularly among young patients with uveitis, where the likelihood of steroid-induced IOP elevation is notably higher in patients.[6,7] The mechanism of steroid-induced IOP elevation has not yet been fully understood; it is believed that the activation of steroid receptors in trabecular meshwork cells, along with increased myocilin gene expression and greater synthesis of glycoproteins and extracellular matrix, complicates the outflow of aqueous humor.[8,9] Additionally, variations in steroid receptors and genetic predispositions in patients contribute to the development of steroid-induced IOP elevation.[8,9]

Although IOP elevation after periocular steroid injections

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is often controlled with medical therapy, glaucoma surgery may be required to control IOP in refractory cases.[2,3] In the literature, it has been reported in small case series that excision of subtenon TA deposits provides IOP control in cases where persistent IOP elevation developed despite medical treatment after PSTA injection.[7,10,11]

This case report presents a 34-year-old female patient who received a PSTA injection for uveitic macular edema and later developed elevated IOP that could not be managed with medical treatment. The patient underwent excision of subtenon TA deposits for IOP control and showed postoperative drug-free IOP control. This report highlights the potential benefits of surgically excising subtenon TA deposits in treating steroid-induced ocular hypertension that is refractory to medical therapy following PSTA injection.

CASE REPORT

A 34-year-old female patient presented to our uvea clinic with complaints of redness, pain, and photophobia in both eyes that had persisted for 10 days. There was no history of systemic or ocular disease or medication use in the patient's medical history. On examination, the best corrected visual acuity (BCVA) according to the Snellen chart was 0.4 in the right eye and 0.8 in the left eye. IOP values were 12 mmHg in the right eye and 14 mmHg in the left by applanation tonometry. Anterior segment examination showed ciliary injection in both eyes, 4+ anterior chamber cells in the right eye and 2+ in the left eye, along with bilateral posterior synechiae. During the dilated fundus examination, vitreous was quiet in both eyes, and macular edema was noted in the right eye, whereas the left eye's fundus examination was normal. Spectral domain optical coherence tomography (OCT) revealed retinal thickening, intraretinal cysts, and subretinal fluid in the right eye, while the macula in the left eye appeared normal. (Figure 1a-1b)

In the patient with bilateral non-granulomatous anterior uveitis and cystoid macular edema (CME) in the right eye, laboratory tests for infectious and non-infectious inflammatory diseases revealed no pathology. The rheumatology consultation was assessed as normal. Topical steroid and mydriatic treatment was started in both eyes with the diagnosis of idiopathic noninfectious anterior uveitis. Due to severe CME causing vision loss in the right eye, following local anesthesia with 0.5% proparacaine hydrochloride ophthalmic solution (Alcaine, Novartis, Basel, Switzerland), 40 mg/0.1 ml TA (Kenacort-A, Deva Holding, Istanbul, Turkey) was injected into the subtenon space in the superior temporal region using a 25G needle, advancing posteriorly towards the macula region.

At the one-month follow-up, BCVA was 1.0 in both eyes, IOP was 17/15 mm Hg, and the anterior segment inflammation findings, along with CME in the right eye, improved with treatment (Figure 1c-1d). The patient was

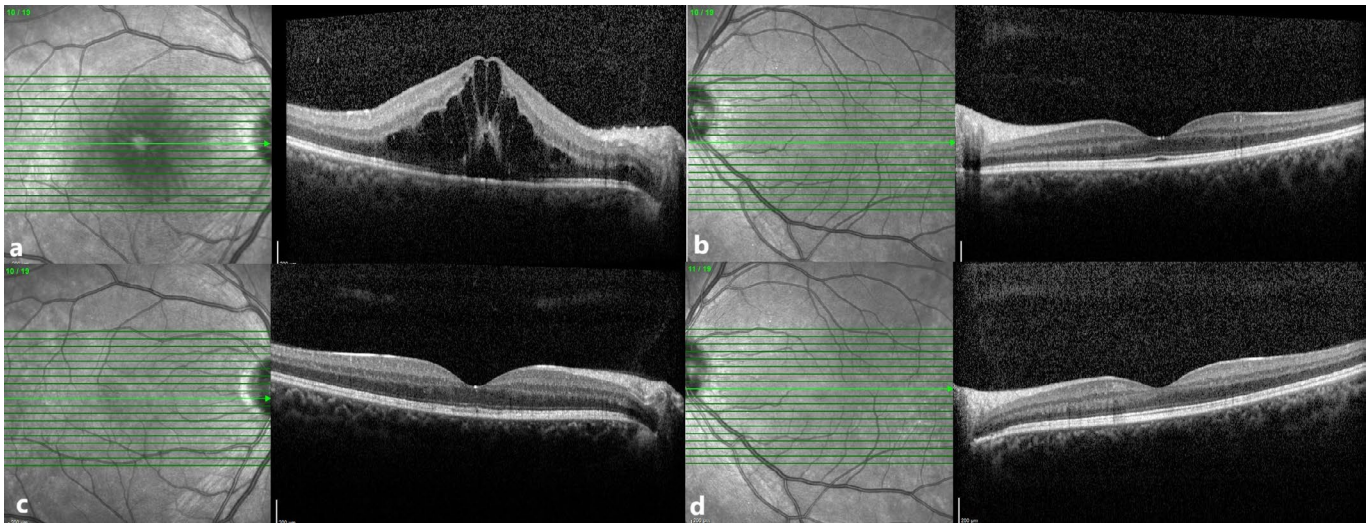

Figure 1. *Optical coherence tomography (OCT) images show cystoid macular edema in the right eye at the initial presentation (a), while the left eye exhibits a normal macular OCT (b). One month after the subtenon triamcinolone acetonide injection, regression of macular edema is observed in the right eye (c), with the left eye maintaining a normal macular OCT (d).*

followed up with a plan to gradually reduce the use of topical steroids. At the second month follow-up, the patient showed no signs of intraocular inflammation and the IOP measured 30 mmHg in the right eye and 15 mmHg in the left eye. The patient who developed steroid-induced IOP elevation in the right eye after PSTA was administered a topical fixed combination of dorzolamide and timolol (Tomec, Abdiibrahim İlaç, Istanbul, Turkey). At the third month follow-up, the anterior and posterior segments were quiet, IOP was evaluated as 18/15 mm Hg bilaterally. The patient's topical steroid treatment was discontinued, while topical anti-glaucomatous treatment was planned to be continued.

At the five-month follow-up, the patient's anterior and posterior segment examination appeared stable; however, under topical dorzolamide/timolol fixed combination (Tomec, Abdiibrahim İlaç, Istanbul, Turkey), IOP elevation was again detected in the right eye (45/14 mmHg). For IOP control, the patient's medical treatment was arranged as triple topical agent including brimonidine (Brimogut, Bilim Pharmaceuticals, Istanbul, Turkey) and dorzolamide/timolol combination (Tomec, Abdiibrahim Pharmaceuticals, Istanbul, Turkey), and oral acetazolamide (Diazomid, Sanofi Pharma, Paris, France). Despite receiving medical treatment, the patient was monitored closely for about six weeks, and the IOP in the right eye could not be reduced below 30 mmHg. During the anterior segment examination at the follow-up visit, white TA deposits were observed under the conjunctiva in the upper temporal quadrant where the PSTA injection had been administered. (Figure 2) Surgical excision of the TA deposits was planned for the patient whose IOP remained elevated despite maximum treatment.

After local anesthesia, 10% povidone iodine (Betadix, Naturel İlaç Company, Istanbul, Turkey) was applied to the periocular area, followed by the placement of a sterile drape. The ocular surface was cleansed with 5% povidone iodine (Betadix, Naturel İlaç Firması, İstanbul, Turkey), and the conjunctiva and Tenon were opened near the TA plaque, allowing access to the TA deposits through conjunctival dissection. The TA deposits, which were plaque-shaped, were surgically excised along with Tenon (Figure 3). The wound was closed using 8.0 vicryl sutures, and topical antibiotics were administered for one week post-surgery.

Preoperative IOP was 30 mmHg in the right eye despite maximum topical treatment. After excising the TA deposit, the IOP decreased to 28 mmHg in the right eye on the first postoperative day. Treatment with Brimonidine (Brimogut, Bilim Pharmaceuticals, Istanbul, Turkey) and Acetazolamide (Diazomid, Sanofi Pharma, Paris, France) was discontinued. During the first postoperative week, IOP was 10 mmHg in the right eye, and treatment with Dorzolamide/Timolol (Tomec, Abdiibrahim Pharmaceuticals, Istanbul, Turkey) was also discontinued. In the second postoperative week, IOP was 12 mmHg in the right eye, and IOP remained stable without medication. The patient continues to be monitored. In the third postoperative

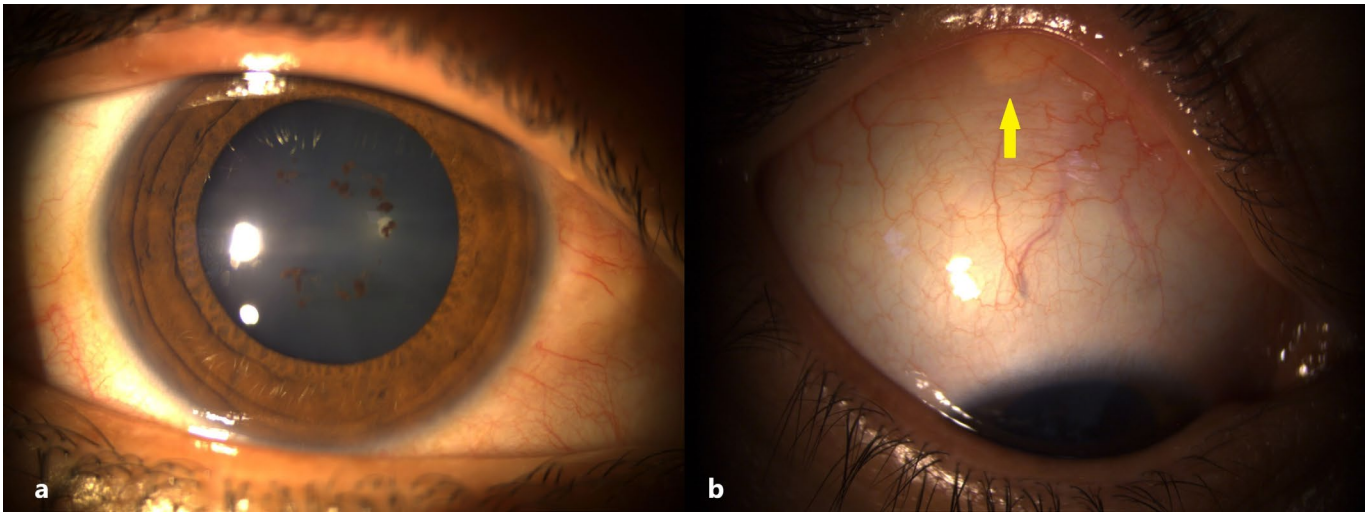

Figure 2. *In the anterior segment image of the right eye, iris pigments are observed on the lens due to previous anterior uveitis (a). In the patient's downward-inward gaze, white subtenon triamcinolone acetonide deposits (yellow arrow) are seen in the superior temporal region (b).*

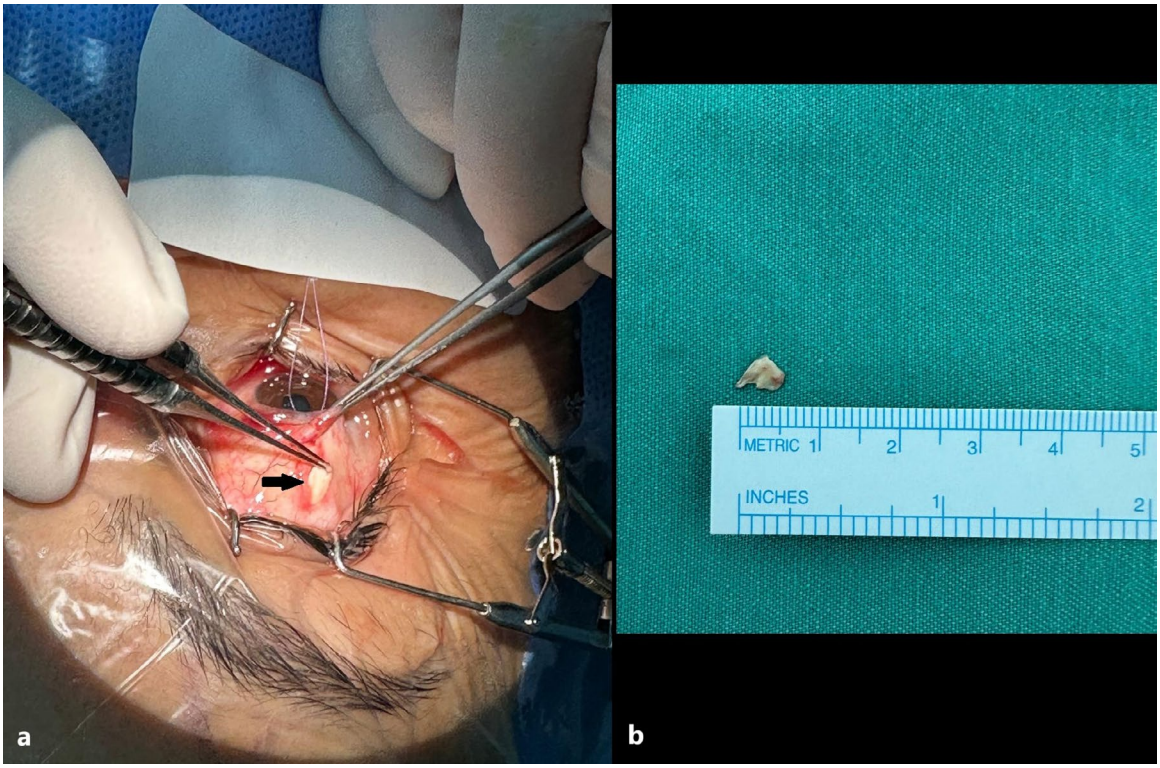

Figure 3: *Images of the triamcinolone acetonide (TA) deposit during and after surgical removal. Following conjunctival dissection, the TA deposit adherent to the Tenon capsule is observed (a, black arrow), and the excised TA deposit appears in a plaque-like form.*

month, IOP was 14 mmHg in the right eye and 15 mmHg in the left. During follow-up, IOP did not increase again, and no new flare-ups of uveitis occurred.

DISCUSSION

Subtenon TA injections are commonly used in patients with NIU to manage ocular inflammation, aiming to minimize the side effects of systemic steroids or assist with systemic treatment.[2,3,12,13] However, PSTA injection is not as innocent as it appears; various ocular complications can arise. The most commonly reported ocular complications include the development of cataracts following IOP elevation.[2-4] In this report, PSTA injection was effective in the treatment of uveitic macular edema in a young female patient with unilateral uveitic macular edema, but steroid-resistant ocular hypertension developed after the injection.

Following PSTA injection, IOP elevation generally begins within weeks and is typically managed with medical anti-glaucomatous treatment.[6-8] Studies have reported that in younger patients, IOP elevation may occur earlier, and achieving IOP control may take longer in these individual patients.[2,7,11] Furthermore, glaucoma surgery is advised for cases that are resistant to medical treatment and demonstrate progression.[2,10] In the study by Okada et al.[13], it was noted that IOP elevation in patients with uveitis occurred within 2 to 3 months after PSTA injection. In 93% of the cases, IOP was managed with a single topical anti-glaucomatous medication, and these medications were discontinued within 6 months. In the study by Leder et al.[4], in a large patient group of 156 eyes treated with periocular TA injection for the treatment of uveitic macular edema, medical treatment was sufficient for most eyes that developed IOP elevation, while 2.5% of these eyes required glaucoma surgery to control IOP.

In the literature, surgical excision of subtenon TA deposits has been described as a less invasive, low-risk, and effective alternative treatment to glaucoma surgery for the treatment of IOP elevation following subtenon TA injection.[10,11] In the study by Okka et al.[10], which included 14 patients with IOP elevation unresponsive to medical treatment, a significant reduction in IOP was achieved after subtenon TA excision. It was reported that 64% of the patients achieved IOP control without medication within an average of one month.[10] Subsequently, Chan et al.[11] reported similarly successful

outcomes in a study involving seven patients with steroid-induced ocular hypertension that was resistant to treatment, demonstrating significant IOP reduction following the excision of subtenon TA deposits. Additionally, the authors emphasized the rapid decrease in IOP subsequent to the excision of the subtenon deposit (average 2.5 days, ranging from 1 to 5 days). They noted that factors such as older age, limited use of antiglaucomatous medications before surgery, and a longer interval between PSTA injection and IOP elevation were linked to faster IOP normalization.[11] In these studies, no patients underwent glaucoma surgery; however, it was emphasized that glaucoma surgery may be necessary in cases where IOP elevation persists despite subtenon TA excision.[10,11] In our case, following subtenon TA excision, IOP returned to normal levels within two weeks, allowing us to discontinue anti-glaucomatous medications.

The rapid normalization of IOP after the excision of subtenon TA deposits has been suggested to be associated with the cessation of gene expression due to the elimination of steroid exposure, as well as the restoration of the normal physiological function of trabecular meshwork cells and the extracellular matrix.[11] In our case, although an initial reduction in IOP was achieved with medical treatment, IOP elevation persisted during follow-up, and no response to medical therapy was observed. This may be attributed to the ongoing steroid effect on the trabecular meshwork due to the presence of subtenon TA deposits in the superior temporal quadrant. The rapid normalization of IOP after the excision of these subtenon TA deposits can be explained, as mentioned above, by the removal of the steroid effect on the trabecular meshwork cells.

In conclusion, patients who receive PSTA injections should be closely monitored for IOP elevation. If IOP remains elevated despite medical treatment, the presence of subtenon TA deposits should be considered, and the injection site should be carefully examined. In cases where subtenon TA deposits are detected during examination and ocular hypertension persists despite medical therapy, excision of subtenon TA deposits may be considered as an initial treatment option before proceeding with glaucoma surgery.

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