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Case Report

A Case of Incidental and Uncomplicated Subretinal Triamcinolone Acetonide

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Keywords

Subretinal injection · Triamcinolone acetonide · Cystoid macular edema · Scleral buckle · Retinal detachment

Abstract

Introduction: Posterior subtenon injection of triamcinolone acetonide (PSTA) is commonly done to treat refractory cases of macular edema. Complications may arise from the procedure as well as from the use of the periocular steroid medications. Side effects include subconjunctival hemorrhage, progression of cataract, scleral perforation (resulting in subretinal, subhyaloid, or intravitreal injection of the drug), retinal detachment, ptosis, orbital fat prolapse, orbital abscess, infectious scleritis, ocular hypertension, and scleral abscess. Here we describe a case of inadvertent subretinal triamcinolone acetonide (TA) deposition from a PSTA procedure without any adverse vision-threatening outcomes. ***Case Presentation:*** We report a patient who presented with a history of superior temporal left eye macula-off rhegmatogenous retinal detachment, which was successfully repaired with a scleral buckle (SB), pars plana vitrectomy, and gas placement. Due to persistent diplopia, the SB was removed after 1 year post-operatively. Due to the development of cystoid macular edema, a PSTA was performed after the patient failed topical steroids and NSAIDs. The procedure was halted early due to unexpected resistance during the injection. A dilated fundus exam showed the presence of subretinal triamcinolone acetonide. The patient was observed and found to have no complications with almost complete resolution of the triamcinolone acetonide after 3 months. ***Conclusion:*** In previous SB patients, it is important to highlight the risk of globe penetration, subretinal deposition of TA, formation of retinal breaks, or reopening of prior retinal breaks with posterior subtenon injection, which could have adverse effects on the local retina as well as the risk of retinal detachment.

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Introduction

Posterior subtenon injection of triamcinolone acetonide (PSTA) is commonly done to treat refractory cases of macular edema including Irvine - Gass Syndrome, diabetic macular edema, macular edema due to retinal vein occlusions as well as non-infectious uveitic macular edema [1]. Despite the procedure being relatively safe, complications may arise from the procedure as well as from the use of the periocular steroid medications. Side effects include subconjunctival hemorrhage, progression of cataract, scleral perforation (resulting in subretinal, subhyaloid, or intravitreal injection of the drug), retinal detachment, ptosis, orbital fat prolapse, orbital abscess, infectious scleritis, ocular hypertension and scleral abscess [1, 2]. Here, we describe a case of inadvertent subretinal triamcinolone acetonide (TA) deposition from a PSTA procedure in a patient post-scleral buckling without any adverse vision-threatening outcomes.

Case Presentation

This is a case report of a 67-year-old male with a significant ocular history of pseudophakia, and a superior temporal left eye macula-off rhegmatogenous retinal detachment, which was successfully repaired with a scleral buckle (SB), pars plana vitrectomy, and gas placement. The SB was removed 1 year after placement due to persistent vertical diplopia. During the post-operative period, cystoid macular edema (CME) occurred which was initially managed with prednisolone acetate and ketorolac drops with minimal improvement after 5 months. Given the refractory CME, PSTA injection (Kenalog®, Bristol Myers Squibb, NY, USA), using the Nozik technique and a 27G needle, was given in the superior temporal quadrant. A conjunctival incision was not made. During the procedure, when around 0.3 cc of triamcinolone was injected, the globe was noted to become firm, so the procedure was stopped prematurely. An anterior chamber paracentesis was done. A dilated fundus exam immediately post procedure revealed subretinal TA in the superior temporal quadrant without any TA in the vitreous cavity (Fig. 1). The retina otherwise was attached, and the intraocular pressure was 6 mm Hg after the anterior chamber tap was done. The patient's axial length was 24.88 mm OD and 25.66 mm OS (SB still in place). The patient was followed up 1 week, 1 month, and 3 months after the procedure and the retina remained attached with complete resolution of the CME and decrease in the subretinal Kenalog (Fig. 2, 3). His uncorrected visual acuity improved from 20/100 to 20/70 and his intraocular pressures remained normal. Near complete resolution of the subretinal TA was noted after 3 months on fundus exam and his retina remained attached.

Discussion

There have been a few case reports of inadvertent subretinal TA from a PSTA; however, this is the first to report it in a patient who had a recent SB removal [3, 4]. Retinal detachment is a known complication of PSTA [5], which was a major concern in this case given the prior history of retinal detachment with the break located around the subretinal TA tracking area. Given that the TA was given superior temporal, it was of utmost importance that the injection was halted soon in this case, which prevented further dissection of the retina and reopening of the break. Fortunately, the injection did not cause any new retinal breaks either, so the patient did not develop a subsequent retinal detachment. As there was no evidence of a retinal detachment, the patient was monitored

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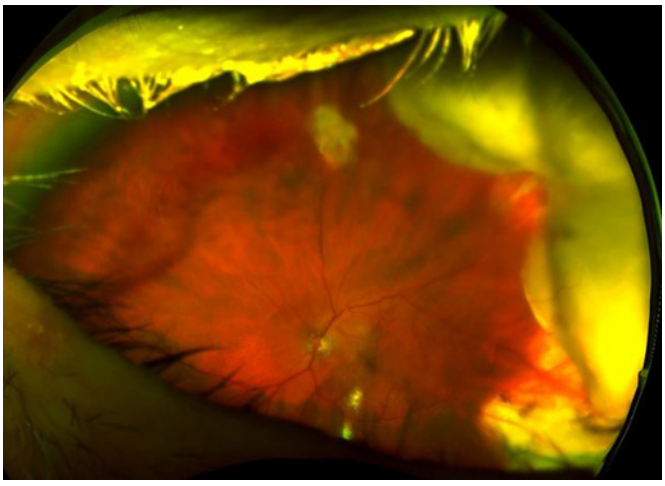

Fig. 1. Colored fundus photo showing superior and temporal subretinal triamcinolone acetonide deposits on day 0.

and the subretinal TA has near resolution after 3 months. The retina has remained stable 3 months after the injection.

One study that evaluated globe perforation from peribulbar and retrobulbar injections showed that patients with a SB are at increased risk of needle penetration of the globe from periocular injections [6]. The study also highlighted other risk factors which include moderate myopia (−2.5 to −7.0 diopters) and high myopia (−7.0 to −11.0 diopters) [6]. Subretinal TA has been shown to be toxic to the retina [7, 8]. Destruction of photoreceptor outer segments and migration of macrophage-like cells in the subretinal space in TA doses above 4 mg have been reported but no morphological changes were noted when 0.5 mg and 1 mg were injected in the subretinal space of a rabbit's eye [7]. Hyperpigmentation changes in the location of the drug deposits were also noted in a rabbit's eye model [8].

Triamcinolone acetonide was administered in this patient for refractory CME. The mechanism for refractory CME in this patient is unclear. However, there is literature that describes the mechanism of CME in post-scleral buckling patients. Post-operatively, retinal capillaries have demonstrated increased permeability due to prostaglandin release and a breakdown of the blood-retina barrier [9]. Combined with age-related changes in retinal vasculature, the manipulations of surgery, resulting in IOP shifts may have resulted in CME in this case [10]. First line therapies for macular edema typically include topical non-steroid anti-inflammatory drugs and steroids, including ketorolac and prednisolone acetate respectively (the patient failed both of these therapies). Carbonic anhydrase inhibitors can also be used; however, they are typically used in cases of taxel-induced CME or retinitis pigmentosa-induced CME [11, 12]. Anti-VEGF use for pseudophakic CME has been documented, but its use is typically reserved for cases unresponsive to conventional treatments [13]. Suprachoroidal injection of TA has recently been introduced and is an effective way of delivering TA with less intraocular complications of increase in IOP and cataract formation [14]. Pharmacological vitreolysis have also been used in cases of CME induced by vitreomacular traction. Surgical options typically are used to relieve a traction component of CME; however, there have been instances where vitrectomy has been shown to improve visual acuity in pseudophakic CME [15]. Obviously, these latter treatments were not an option in this vitrectomized eye. The mechanism for CME resolution in this patient likely stems from a greater penetrance of steroids through subtenon/subretinal means as compared to topical routes.

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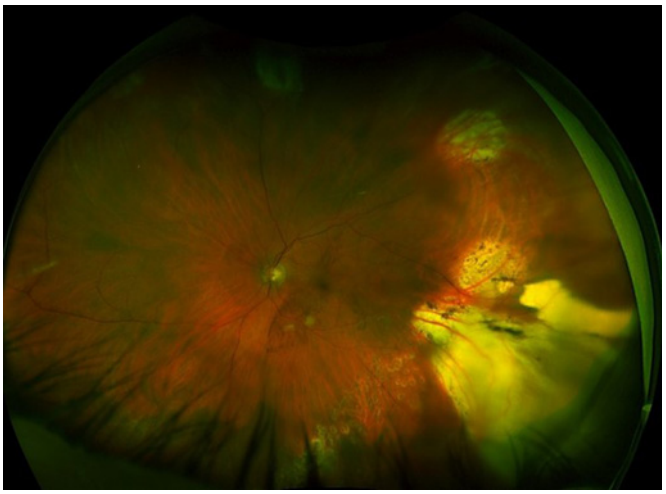

Fig. 2. Colored fundus photo showing almost full resolution the subretinal triamcinolone acetonide deposits after 3 months. Only few deposits remain in the temporal peripheral retina.

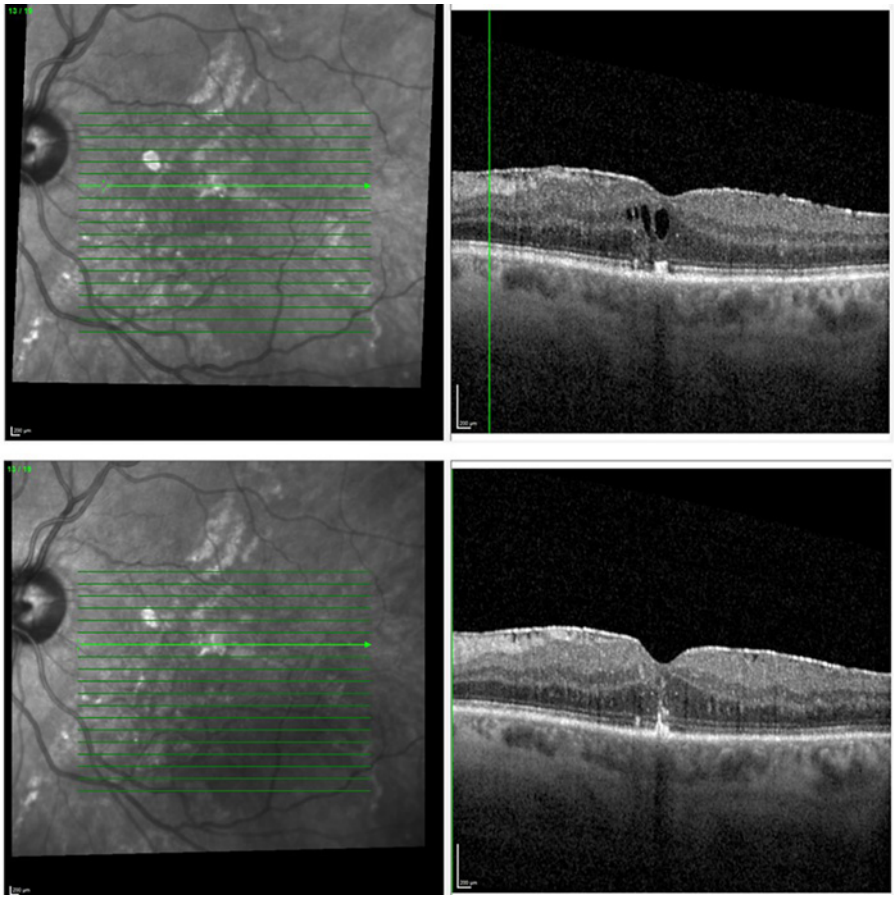

Fig. 3. OCT of the macula showing cystoid macular edema (CME) before the injection of triamcinolone acetonide (TA) above and after injection of TA below with resolution of CME and some outer retinal changes. The epiretinal membrane remained stable.

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In summary, we presented a case of subretinal triamcinolone acetonide after a PSTA injection in a post-SB patient. In such cases with history of scleral buckling and removal, the sclera is thin and the risk of perforation is high. Based on our experience, it is advisable to use other routes of steroid delivery. Peripheral subretinal TA needs close surveillance to rule out small retinal break formation from needle penetration and future development of retinal detachment. In the absence of other complications, the presence of triamcinolone subretinally is benign as it usually reabsorbs without consequences unless it is under the fovea, or threatening it, in which case surgery should be considered. In patients who develop a retinal detachment, a timely surgical intervention is recommended. In this case, there were no sight-threatening adverse consequences likely due to the peripheral location of the subretinal TA and absence of new retinal breaks or reopening of the prior one.

Conclusion

Scleral thinning in patients who have SB removal should be taken into consideration when planning to treat any form of recalcitrant macular edema. It is important to highlight the risk of globe penetration, subretinal deposition of TA, formation of retinal breaks, or reopening of prior retinal breaks with posterior subtenon injection, which could have adverse effects on the local retina as well as the risk of retinal detachment. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000539190).

Statement of Ethics

Ethical approval is not required for this case report in accordance with local and national guidelines. Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

You Zhou, Rodney Guiseppi, and Touka Banaee contributed to data collection, data analysis, manuscript writing, manuscript editing, and manuscript review.

Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material. Further inquiries can be directed to the corresponding author.

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