Title

RECALCITRANT CYSTOID MACULAR EDEMA AFTER PARS PLANA VITRECTOMY

Journal

Retina, 36(7)

ISSN

0275-004X

Authors

Alam, Mostafa R

Arcinue, Cheryl A

Mendoza, Nadia B

et al.

Publication Date

2016-07-01

DOI

10.1097/iae.0000000000000892

Peer reviewed

HHS Public Access

Author manuscript

Retina. Author manuscript; available in PMC 2020 July 15.

Published in final edited form as:

Retina. 2016 July ; 36(7): 1244–1251. doi:10.1097/IAE.0000000000000892.

Recalcitrant cystoid macular edema after pars plana vitrectomy

Mostafa R. Alam, M.D.[1], **Cheryl A. Arcinue, M.D.**[1], **Nadia B. Mendoza, M.S.**[1], **William R. Freeman, M.D.**[1]

[1]Jacobs Retina Center, University of California San Diego Shiley Eye Institute, La Jolla, CA

Abstract

Purpose: To evaluate the outcomes of different types of treatment for chronic cystoid macular edema (CME) after pars plana vitrectomy (PPV).

Methods: Retrospective review of eyes that developed chronic CME after PPV treated with intravitreal triamcinolone acetonide (TCA) with or without the addition of anti-VEGF.

Results: Thirty-nine eyes of 37 patients were included, with a median duration between PPV and onset of CME of 5 months (interquartile range (IQR), 3-12). In the majority of eyes (66.7%), the main indication for surgery was for vitreomacular interface (VMI) disorders, such as epiretinal membrane (ERM), vitreomacular traction (VMT), and macular hole. With intravitreal TCA, there was a significant decrease in central foveal thickness (CFT) at 3, 6, and 12 months, compared with baseline (p=0.0171, 0.0401, and 0.0024, respectively). A significant gain in vision was noted at 1 month compared with baseline (p=0.0169), but this was not sustained at 3, 6, and 12 months (p=0.4862, 0.9098, and 0.4312, respectively). The addition of bevacizumab to TCA did not provide any additional benefit for CFT and VA. Thirty-two eyes (82.1%) were started on prophylactic anti-glaucoma drops two weeks after a TCA injection, and no eye needed laser or surgery to control IOP.

Conclusion: Chronic CME after PPV is recurrent and difficult to treat. Intravitreal TCA is effective in reducing CME, but there was only short-term VA improvement even with continued reduction of CFT. IOP did not significantly rise with the use of prophylactic anti-glaucoma drops even with repeated injections.

Keywords

anti-VEGF; bevacizumab; corticosteroids; cystoid macular edema; pars plana vitrectomy; post-surgical CME; steroid-induced glaucoma; triamcinolone acetonide

Introduction

Although modern vitreoretinal surgery has improved visual outcomes, persistent macular edema after retina surgery remains a poorly documented cause of poor visual results. Little

Corresponding author: William R. Freeman, M.D., Shiley Eye Institute, 0946, 9415 Campus Point Dr., La Jolla, CA 92093, Tel. (858) 534-3513, Fax (858) 534-7985, wrfreeman@ucsd.edu.

Financial Disclosures:
No financial disclosures. None of the authors have any proprietary interest.

information exists in the literature regarding its incidence, effect on visual recovery, and predisposing risk factors.[1]

Cystoid macular edema (CME) is observed after various types of intraocular surgical procedures. It is a post-operative complication of cataract, glaucoma and penetrating keratoplasty. However, there are few articles describing the occurrence of this complication after pars plana vitrectomy (PPV).[2] These reports describe the incidence of CME after pars plana vitrectomy but do not describe the efficacy and safety outcomes of different types of treatment for such edema. There are only three articles that discuss the management of post retinal surgery CME by using the intravitreal dexamethasone implant and bevacizumab.[3–5]

Fluorescein angiography (FA) has been the gold standard for the diagnosis of CME after surgery and it has been shown to be more sensitive to anatomical macular changes than is the clinical examination. Optical coherence tomography (OCT) is a newer method for high-resolution, cross-sectional imaging of the retina. Optical coherence tomography has become an invaluable diagnostic tool to assess ME and offers several advantages over the gold standard FA, including patient safety and comfort. Moreover, OCT provides quantitative and continuous measurements of retinal thickness as opposed to the subjective and categorical interpretation of leakage on FA.[6–8] The purpose of our study was to evaluate the efficacy and safety outcomes of different types of treatment for chronic cystoid macular edema (CME) after pars plana vitrectomy (PPV). In all cases, both fluorescein angiography and spectral-domain OCT were used simultaneously to characterize the anatomical condition and ETDRS visual acuity measurements were used to evaluate vision outcomes.

Materials and methods

This was a retrospective review of eyes that developed chronic CME after vitreoretinal surgery. We evaluated the results of different types of treatment of this specific type of post-surgical CME. The patients were seen at the Jacobs Retina Center, University of California San Diego (UCSD) Shiley Eye Institute. Written informed consent was obtained for each patient prior to evaluation, surgery and all intravitreal injections. UCSD Institutional Review Board (IRB) approval was acquired for the review and analysis of patient data.

All patients that developed CME after PPV (between January 2009 and July 2014) were identified through a thorough search of the database of hospital surgical and imaging records and were included in the analysis of patient data. Chronic CME was defined as observation of increased retinal thickness and intraretinal cysts by OCT and/or leakage on fluorescein angiogram (FA) for > 6 months, associated with a decrease in visual acuity. Exclusion criteria included: (a) presence of preoperative CME, (b) CME secondary to other surgical procedures such as cataract, glaucoma, or corneal surgery, an (c) other ocular conditions that may be responsible for the CME, such as uveitis, ERM, diabetic macular edema, retinal vein occlusion (RVO), and ocular trauma.

The data that we abstracted from these charts included baseline demographic characteristics including patient age, sex, eye laterality, indication for surgery, and duration between the surgery and onset of CME; baseline preoperative clinical data including best-corrected

visual acuity (BCVA) using a standard Early Treatment Diabetic Retinopathy Study (ETDRS) chart, central foveal thickness (CFT) assessed by SD-OCT (Spectralis HRA+OCT, Heidelberg Engineering, Carlsbad, CA) from horizontal or vertical scans passing through the central fovea and measured using the calipers feature or thickness profile of the Spectralis device, and intraocular pressure (IOP); presence of intraoperative complications; postoperative data including IOP, BCVA, CFT, at months 1, 3, 6 and 12, and occurrence of postoperative complications; different methods of treatment of CME, including type of treatment, regimen and number of injections, dose given; need for other procedures such as repeat vitrectomy with ILM peel, laser, or both; lens status during follow-up and need for cataract surgery; presence of IOP elevations and need for glaucoma medications, laser, or glaucoma surgery for IOP control. ETDRS visual acuities were in logarithm of the minimum angle of resolution (logMAR) notation, with 0 being the highest score corresponding to 20/20 visual acuity and a value of 1 corresponding to 20/200 visual acuity. Snellen charts were not used.

Because the eyes were vitrectomized and intravitreal anti-VEGF monotherapy was felt to be of limited duration and efficacy[9], our initial therapy was decanted triamcinolone injected intravitreally. In eyes failing this therapy, anti-VEGF was added.

Intravitreal injection of therapeutics was carried out under aseptic conditions in the clinic. Preservative-free lidocaine gel[10] was instilled in the eye for at least 3 doses five minutes or longer prior to injection. A lid speculum was placed to keep the lids open and 5% povidone iodine solution was instilled on the conjunctival sac prior to injection. The intravitreal injection was performed using a 27 or 30-gauge needle (triamcinolone or anti-VEGF respectively) in the superotemporal or superonasal quadrant, 3.5-4.0 mm posterior to the limbus depending on phakic status

For intravitreal injection of triamcinolone acetonide (TCA) 40 mg/ml (Kenalog, Bristol-Myers Squibb, Princeton, NJ), the TCA was decanted by removing the preservative solution as much as possible, and retaining the TCA particles in the bottom. To achieve a dose of 20 mg TCA, 0.1 ml was injected, and 0.05 ml for a dose of 10 mg TCA.[11] No topical antibiotic drops were prescribed after the procedure.

The primary treatment outcome was change in visual acuity and central foveal thickness compared with baseline. Secondary safety outcomes included changes in IOP compared with baseline and the need for any glaucoma medications, laser, or surgery; and lens status changes and need for cataract surgery during the follow-up period.

Descriptive statistics using Kruskal Wallis and Wilcoxon Rank Sum tests were performed for continuous variables when comparing treatment groups, and Fisher's exact test was performed for categorical variables. Univariate mixed models for longitudinal analysis were created using 5 measures (baseline, 1 month, 3 months, 6 months and 12 months) for VA, CFT and IOP. Paired T-tests were performed for comparison of baseline with measurements for months 1, 3, 6, and 12 for each of the 3 variables for VA, CFT and IOP. For all hypothesis tests, statistical significance was set at a level of $p<0.05$. Statistical analyses were

performed using SAS software version 9.3 (SAS Institute, Cary, North Carolina, USA) and R version 3.0.0 (http://www.r-project.org/).

Results

A total of 39 eyes of 37 patients with chronic, recalcitrant post-PPV CME were included in the review and analysis of data. Baseline demographic characteristics are shown in Table 1. The median age of the patients was 71 years old (interquartile range (IQR), 65-78), with a 1:1 male:female ratio (48.7% males, 51.3% females). The median duration between PPV and onset of CME was 5 months (IQR, 3-12). In a majority of eyes (66.7%), the main indication for surgery was for vitreomacular interface (VMI) disorders, such as epiretinal membrane (ERM), vitreomacular traction (VMT), and macular hole; followed by retinal detachment (RD) and RD-associated procedures such as silicone oil removal (28.2%); and dislocated lens (5.1%). Mean CFT at baseline for all eyes was 433.95 um (range, 216-739 um). All patients/eyes in this study underwent both FA and SD-OCT and have shown evidence of macular edema on both imaging studies, specifically leakage of fluorescein in the macula on FA and increased retinal thickness with intraretinal cysts on SD-OCT.

Treatment outcomes for all eyes that received TCA for post-vitrectomy CME are shown in Table 2 and Figures 3 and 4. Anatomically, there was a significant decrease in central foveal thickness (CFT) at 3, 6, and 12 months, compared with baseline (p=0.0171, 0.0401, and 0.0024, respectively). At 1 month, there was a decrease in CFT but this was not found to be statistically significant compared with baseline (p=0.2950). In terms of visual acuity improvement, there was a significant gain in vision at 1 month compared with baseline (p=0.0169), but the visual improvement was not sustained thereafter at 3, 6, and 12 months (p=0.4862, 0.9098, and 0.4312, respectively).

The average time between TCA and bevacizumab was 2.9 ± 0.7 months (range 2-4 months). The average number of bevacizumab injections given was 3.3 ± 2.4 injections (range 1-10 injections). The average CFT change after the addition of bevacizumab was 16.3 ± 142.8 um (range −94 – 382 um). There was only one case that we detected a decrease in CFT by 382 um, with the other cases showing either no change in thickness or an increase in thickness. Comparison of the eyes that received TCA alone and the eyes that received additional bevacizumab (TCA + bevacizumab) is shown in Table 3 and Figures 3 and 4. As shown on this table, the addition of bevacizumab to TCA for recalcitrant post-vitrectomy CME did not provide any additional benefit (i.e., no significant differences were noted between baseline and months 1, 3, 6, and 12 months for CFT, VA, and IOP). Three eyes received bevacizumab alone due to the presence of underlying advanced glaucoma; no significant anatomical or functional benefit was noted in these 3 eyes (results not shown on the table).

The indication for surgery and the duration between surgery and onset of CME were not significantly associated with predicting visual acuity values. A greater number of TCA injections was significantly associated with poorer visual acuity (p=0.012). The indication for surgery also did not predict CFT values, and the different types of treatment were not significantly associated with predicting CFT as well. Later time of onset of CME from vitrectomy was associated with lower CFT values (p=0.048). Significantly higher values of

CFT were observed for more TCA injections (p=0.001), because understandably, worse eyes required more injections.

A greater number of TCA injections were predictive of higher IOP values (p=0.007). Thirty-two eyes (82.1%) were started on prophylactic anti-glaucoma drops two weeks after a TCA injection, and no eye in this cohort needed laser or surgery to control IOP. At baseline, 37 eyes (94.9%) were pseudophakic and 2 eyes were aphakic (5.1%). Hence, there were no lens status changes and need for cataract surgery during the follow-up period.

Discussion

The objective of this study was to evaluate the efficacy and safety outcomes of different types of treatment for chronic cystoid macular edema (CME) after pars plana vitrectomy (PPV) and in this paper, we report the treatment outcomes of decanted TCA intravitreal injections with or without additional bevacizumab for chronic post-vitrectomy CME. Although it would have been valuable, determination of the incidence of post-PPV CME could not be performed because many of our cases were referred to us by other retinal surgeons.

Chronic CME after pars plana vitrectomy has been reported in only a few articles. No one has evaluated the therapy of this disease except for 3 studies that discussed a single treatment for each of their cohort. The first study discussed treatment using a single injection of the 0.7 mg intravitreal dexamethasone implant in a small number of eyes (8 eyes) with refractory macular edema secondary to combined cataract extraction and vitrectomy for macular pucker.[3] The group found that best-corrected visual acuity and central retinal thickness (CRT) significantly improved, and IOP significantly increased, after a mean follow-up of 6 months. The second study showed no significant improvement in visual acuity or CMT after intravitreal bevacizumab, compared to control, in 26 eyes (of 26 patients) with persistent macular edema after idiopathic macular ERM surgery.[4] Taney, Baumal and Duker also evaluated the effect of sustained-release dexamethasone intravitreal implant for persistent macular edema after vitrectomy for ERM in 5 eyes, demonstrating that there was an improvement in visual acuity and macular thickness in 4 eyes because the authors attributed the refractory macular edema to inflammatory factors.[5] To the best of our knowledge, our study is the first study reporting the efficacy and safety outcomes of different types of treatment for post-vitrectomy CME, and we have the largest number of eyes (39 eyes) evaluated, with follow-up of up to 1 year.

In our study, the cases that we included showed leakage on FA in the late phase, which indicates that the most likely mechanism of CME is inflammation causing a breakdown of the blood-retinal barrier. In the literature, there are two proposed mechanisms of development of postoperative CME: The first mechanism concerns the cases with postoperative CME without vascular leakage. The hypothesized mechanisms are based on the iatrogenic damage of the Muller cells secondary to the removal of ERM and peeling of ILM. In these cases, the weakening of the Muller cells due to the evolution of ERM could lead to the formation of intraretinal cysts. The absence of leakage during fluorescein angiography could confirm that the intraretinal cysts are not related to the breakdown of the

blood–retinal barrier but are related to a structural damage of the retina. ERM with intraretinal cysts could exert great force on the underlying retina including the photoreceptor layer, and promote inner segment/outer segment (IS/OS) junction disruption. The second mechanism concerns the occurrence of postoperative CME, with leakage highlighted by fluorescein angiography. The postoperative CME could be due to an inflammatory process due to a breakdown of the blood–retinal barrier, as seen in our study.[2]

Corticosteroids are potent anti-inflammatory agents that can counteract many of the pathological process thought to play role in the development of macular edema by several ways: they prevent leukocyte migration, reduce fibrin deposition, stabilize endothelial cell tight junctions, and inhibit synthesis of vascular endothelial growth factor, prostaglandins, and pro-inflammatory cytokines.[12] On the other hand, anti VEGF agents inhibit the VEGF involved in the VEGF-induced pathogenic pathway, blood-ocular barrier breakdown, and hyperpermeability,[13] which is known to be the cause of macular edema in various diseases such as diabetes, AMD, and vein occlusion. However, in our study, the cause of macular edema may be due to inflammation that occur post surgery, this is why bevacizumab may have had only a mild effect in decreasing edema, and because bevacizumab has a short half life in vitrectomized eyes.[14]

In this study, we did not compare treatment outcomes of triamcinolone acetonide with anti-VEGF agents, particularly bevacizumab, because we always give corticosteroids first for these cases. Based on our results, we cannot conclude which is the better treatment between these two classes of ocular therapeutics for post-vitrectomy CME.

Overall, there was a significant anatomical improvement at 3, 6, and 12 months with the initial treatment with steroids. In terms of visual acuity improvement, the significant gain in vision at 1 month was not sustained at 3, 6, and 12 months. Adding anti-VEGF in these eyes resulted in minimal additional benefit because we gave bevacizumab in worse cases. We cannot conclude that this was a failure of therapy, but in these cases, the addition of an anti-VEGF agent did not help anatomically and functionally. We could have tried giving an anti-VEGF agent as first line therapy for post-vitrectomy CME to test their efficacy, but we did not because it theoretically would not have a long duration of action in vitrectomized eyes. The half-life of intravitreally injected bevacizumab in vitrectomized eyes decreases by 54% compared with nonvitrectomized normal eyes[14] compared with the longer-acting triamcinolone in vitrectomized eyes[15]. Only a randomized controlled clinical trial comparing the efficacy of corticosteroids and anti-VEGF agents will be able to answer this question definitively.

Despite the significant favorable anatomic response to TCA up to 1-year follow-up, there was non-significant improvement in visual acuity 3 months onward to 1 year of follow-up. This may be due chronic CME that can possibly cause progressive IS-OS disruption. In other cases such as MH cases, loss of retinal photoreceptors with subsequent thinning, and possible toxicity from the use of indocyanine green in MH eyes,[1] may explain why the visual acuity did not significantly improve after repeated injections. In the univariate analysis, poorer visual acuities and significantly higher values of CFT were observed for more TCA injections, likely due to the fact that more injections were needed for eyes with

greater CFT values or more severe CME. It is difficult to answer the question if post-vitrectomy CME is treatable or not based on our results. The answer is categorical. Anatomically, yes, the macular edema resolves, but functionally, no, the visual acuity gains are not sustained long-term.

In this study, there was no statistically significant difference in IOP at 1-year compared with baseline for both the TCA alone group and the combined TCA + bevacizumab group. Majority of eyes were started on prophylactic anti-glaucoma drops two weeks after a TCA injection[16], and no eye in this cohort needed laser or surgery to control IOP. Direct intravitreal corticosteroid injection bypasses the blood-retinal barrier, leading to high local drug concentrations with no or little systemic adverse event but has potential ophthalmic adverse events such as cataract formation and transient elevation of IOP.[17] The onset of IOP elevation is variable after initiation of corticosteroid therapy, and the magnitude of the steroid response is equally variable, ranging from a rise of a few millimeters of mercury to dramatic rises of 40 mmHg. The eyes are at maximum risk of an IOP elevation from weeks 2 through 5 after an intravitreal TCA injection.[18] Definitive treatment consists of discontinuation of steroid therapy, although this is not always practical or possible given the nature and severity of the underlying disease process requiring corticosteroid therapy. Traditional IOP lowering therapies, including topical, oral medications and laser trabeculoplasty, have all shown efficacy in lowering steroid-induced elevations of IOP.[19–21] It is the opinion of our group that it is advisable to start prophylactic anti-glaucoma drops to eyes that have received a greater number of TCA injections because of the association of greater number of injections with higher IOP values.[16]

The strengths of the present study include a fairly large cohort of eyes with long follow-up given different types of treatment for the chronic CME, and use of FA and high-resolution spectral domain OCT images. Limitations include retrospective study design.

Conclusion

Chronic CME after pars plana vitrectomy is difficult to treat and usually recurs. Intravitreal TCA is effective in reducing the macular edema anatomically, but there was only short-term (1 month) visual acuity improvement even with the continued reduction of retinal thickness. Ongoing or recurrent chronic inflammation, and the underlying retinal pathology, may be contributing to this limited visual potential. Even with repeated injections, IOP did not significantly rise with the use of prophylactic anti-glaucoma drops.

Acknowledgments

Funding/Support:

This study was supported by NIH grants R01EY007366, R01EY016323, R01EY018589, NEI vision core grant P30EY022589, and Research to Prevent Blindness. The funding organizations had no role in the design or conduct of this research.

References

1. Kim SJ, Martin DF, Hubbard GB 3rd, et al. Incidence of postvitrectomy macular edema using optical coherence tomography. Ophthalmology 2009;116(8):1531–7. [PubMed: 19501405]

2. Frisina R, Pinackatt SJ, Sartore M, et al. Cystoid macular edema after pars plana vitrectomy for idiopathic epiretinal membrane. Graefes Arch Clin Exp Ophthalmol 2015;253(1):47–56. [PubMed: 24859385]

3. Furino C, Boscia F, Recchimurzo N, Sborgia C, Alessio G. Intravitreal dexamethasone implant for refractory macular edema secondary to vitrectomy for macular pucker. Retina 2014;34(8):1612–6. [PubMed: 24752008]

4. Chen CH, Wu PC, Liu YC. Intravitreal bevacizumab injection therapy for persistent macular edema after idiopathic macular epiretinal membrane surgery. J Ocul Pharmacol Ther 2011;27(3):287–92. [PubMed: 21428791]

5. Taney LS, Baumal CR, Duker JS. Sustained-release dexamethasone intravitreal implant for persistent macular edema after vitrectomy for epiretinal membrane. Ophthalmic Surg Lasers Imaging Retina 2015;46(2):224–8. [PubMed: 25707048]

6. Nussenblatt RB, Kaufman SC, Palestine AG, Davis MD, Ferris FL 3rd. Macular thickening and visual acuity. Measurement in patients with cystoid macular edema. Ophthalmology 1987;94(9):1134–9. [PubMed: 3684231]

7. Nelson ML, Martidis A. Managing cystoid macular edema after cataract surgery. Curr Opin Ophthalmol 2003;14(1):39–43. [PubMed: 12544809]

8. Mentes J, Erakgun T, Afrashi F, Kerci G. Incidence of cystoid macular edema after uncomplicated phacoemulsification. Ophthalmologica 2003;217(6):408–12. [PubMed: 14573973]

9. Yanyali A, Aytug B, Horozoglu F, Nohutcu AF. Bevacizumab (Avastin) for diabetic macular edema in previously vitrectomized eyes. Am J Ophthalmol 2007;144(1):124–6. [PubMed: 17601433]

10. Kozak I, Cheng L, Freeman WR. Lidocaine gel anesthesia for intravitreal drug administration. Retina 2005;25(8):994–8. [PubMed: 16340529]

11. Cheng L, Banker AS, Martin M, Kozak I, Freeman WR. Triamcinolone acetonide concentration of aqueous humor after decanted 20-mg intravitreal injection. Ophthalmology 2009;116(7):1356–9. [PubMed: 19450879]

12. Leopold I Surgical pharmacology of the eye. New York, NY: Raven Press; 1985.

13. Liu X, Zhou X, Wang Z, Li T, Jiang B. Intravitreal bevacizumab with or without triamcinolone acetonide for diabetic macular edema: a meta-analysis of randomized controlled trials. Chin Med J (Engl) 2014;127(19):3471–6. [PubMed: 25269916]

14. Kakinoki M, Sawada O, Sawada T, Saishin Y, Kawamura H, Ohji M. Effect of vitrectomy on aqueous VEGF concentration and pharmacokinetics of bevacizumab in macaque monkeys. Invest Ophthalmol Vis Sci 2012;53(9):5877–80. [PubMed: 22836776]

15. Kosobucki BR, Freeman WR, Cheng L. Photographic estimation of the duration of high dose intravitreal triamcinolone in the vitrectomised eye. Br J Ophthalmol 2006;90(6):705–8. [PubMed: 16531422]

16. Barteselli G, Amini P, Ezon IC, Nezgoda JT, Cheng L, Freeman WR. Impact on Intraocular Pressure after 20-Mg Decanted Triamcinolone Acetonide (Kenalog) Injection When Using Prophylactic Antiglaucoma Therapy. Retina 2015;35(1):75–81. [PubMed: 25077534]

17. Haller JA, Bandello F, Belfort R Jr., , et al. Randomized, sham-controlled trial of dexamethasone intravitreal implant in patients with macular edema due to retinal vein occlusion. Ophthalmology 2010;117(6):1134–46 e3. [PubMed: 20417567]

18. Roth DB, Verma V, Realini T, Prenner JL, Feuer WJ, Fechtner RD. Long-term incidence and timing of intraocular hypertension after intravitreal triamcinolone acetonide injection. Ophthalmology 2009;116(3):455–60. [PubMed: 19157561]

19. Ricci F, Missiroli F, Parravano M. Argon laser trabeculoplasty in triamcinolone acetonide induced ocular hypertension refractory to maximal medical treatment. Eur J Ophthalmol 2006;16(5):756–7. [PubMed: 17061231]

20. Pizzimenti JJ, Nickerson MM, Pizzimenti CE, Kasten-Aker AG. Selective laser trabeculoplasty for intraocular pressure elevation after intravitreal triamcinolone acetonide injection. Optom Vis Sci 2006;83(7):421–5. [PubMed: 16840867]

21. Viola F, Morescalchi F, Staurenghi G. Argon laser trabeculoplasty for intractable glaucoma following intravitreal triamcinolone. Arch Ophthalmol 2006;124(1):133–4. [PubMed: 16401800]

Summary Statement:

Chronic CME after PPV is recurrent and difficult to treat. Intravitreal TCA is effective in reducing CME, but there was only short-term VA improvement even with continued reduction of CFT. IOP did not significantly rise with the use of prophylactic anti-glaucoma drops even with repeated injections.

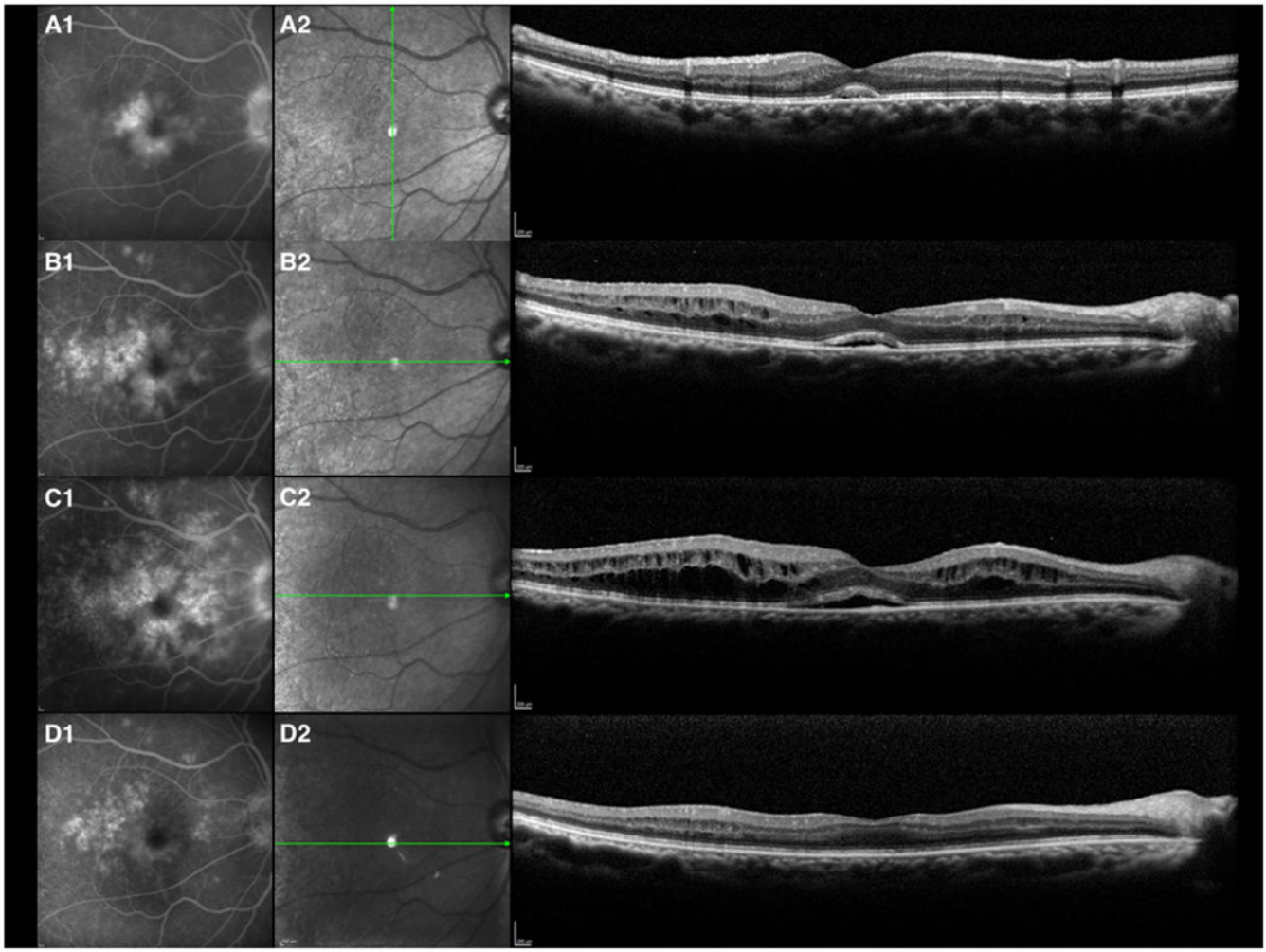

Figure 1.

Fluorescein angiogram (FA) and optical coherence tomography (OCT) of an eye treated with combined intravitreal triamcinolone acetonide (TCA) and bevacizumab. **A1**. Baseline FA shows petalloid macular leakage corresponding to CME and a hot disc. **A2**. Baseline OCT passing through the fovea shows subretinal fluid (SRF) and minimal intraretinal cysts inferior to the fovea. **B1 and B2**. FA and OCT at 3 months showing increased dye leakage, and increased SRF and intraretinal cysts, respectively. **C1 and C2**. FA and OCT at 6 months shows more diffuse leakage of dye during the late phase on FA and greatly increased SRF and intraretinal fluid (IRF) on OCT. **D1 and D2**. FA and OCT at 12 months shows decreased leakage on FA, and resorbed SRF and minimal IRF on OCT.

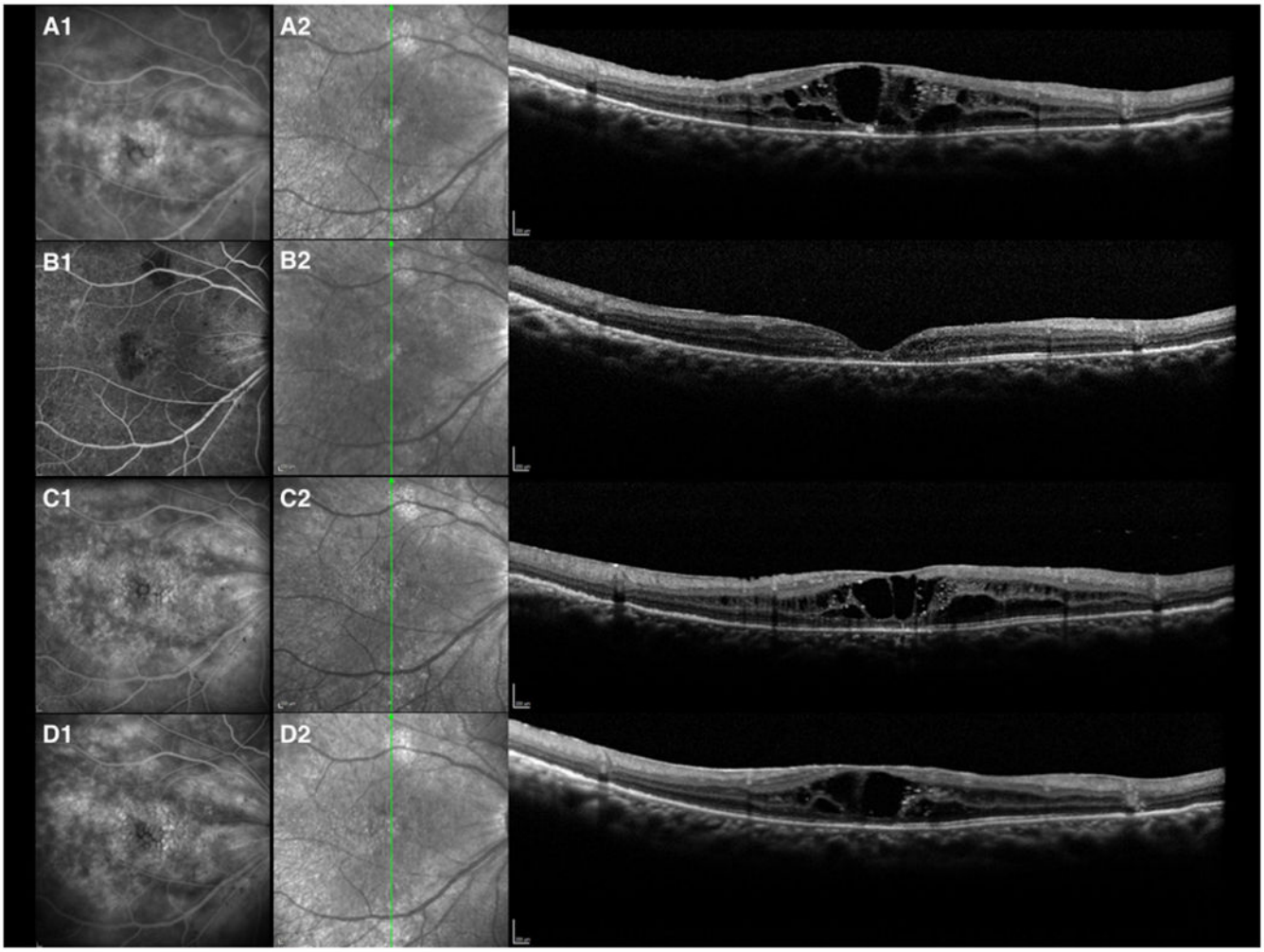

Figure 2.

Fluorescein angiogram (FA) and optical coherence tomography (OCT) of an eye treated with intravitreal triamcinolone acetonide (TCA) alone. **A1**. Baseline FA shows diffuse macular leakage and petalloid leakage in the fovea corresponding to diffuse CME. **A2**. Baseline OCT passing through the fovea shows large intraretinal cysts and an evaginated foveal contour. **B1 and B2**. FA and OCT at 3 months shows no dye leakage, and minimal intraretinal cysts with restoration of the foveal contour, respectively. **C1 and C2**. FA and OCT at 6 months shows recurrence of diffuse leakage of dye during the late phase on FA and recurrence of intraretinal fluid (IRF) on OCT. **D1 and D2**. FA and OCT at 12 months showing essentially unchanged FA and OCT findings from 6 months.

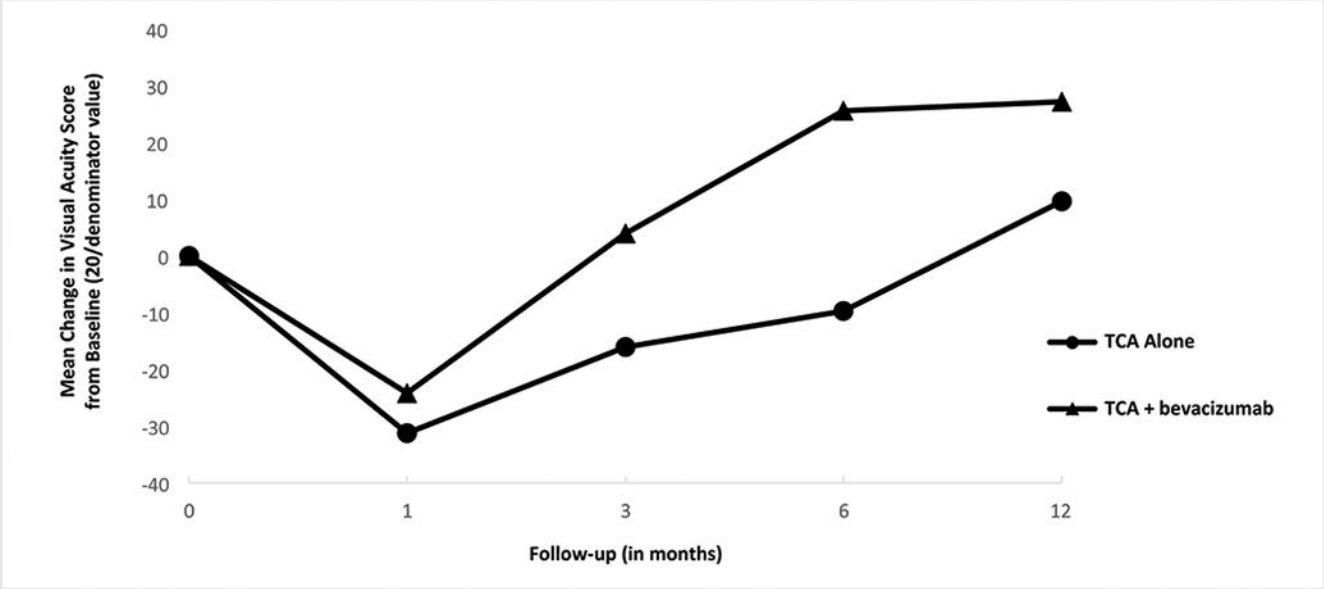

Figure 3.
Graph showing the mean change in visual acuity score during the first year of follow-up.

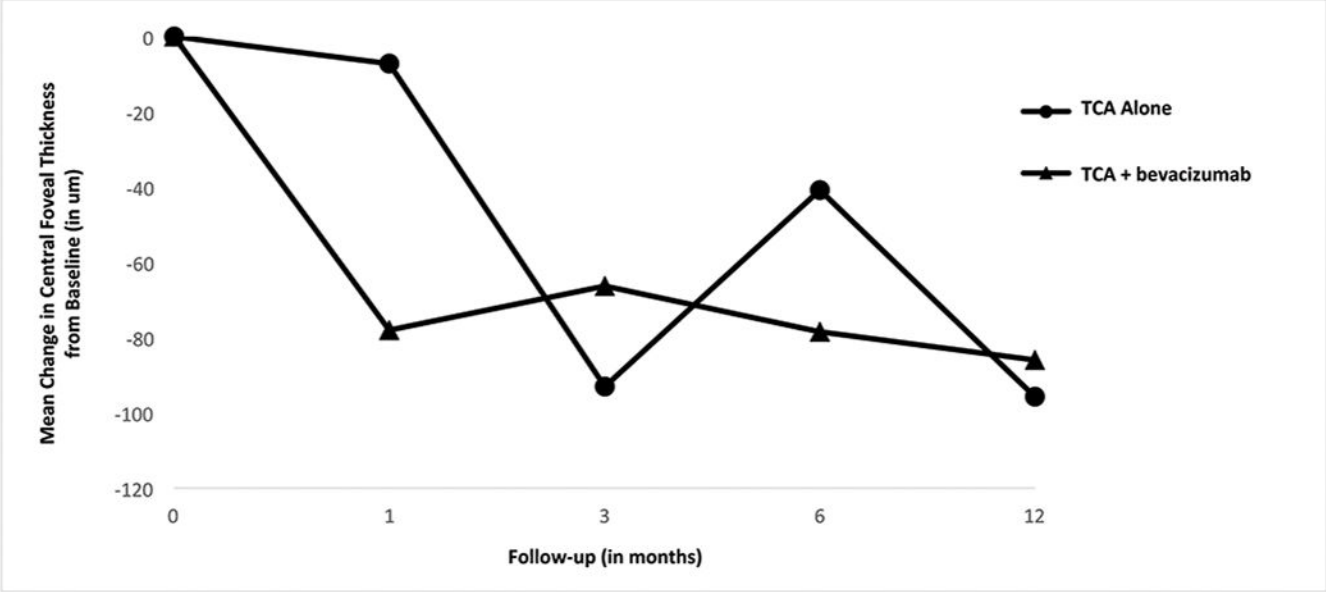

Figure 4.
Graph showing the mean change in central foveal thickness during the first year of follow-up.

Table 1.

Baseline demographic characteristics of eyes with post-vitrectomy CME

	All eyes (n=39)	P value
Male sex, n (%)	20 (51.3)	0.1705
Duration between PPV and onset of CME (in months), median (IQR)	5 (3-12)	0.4118
Central foveal thickness (CFT) (in um), mean (range)	433.95 (216-739)	
Indication for surgery, n (%)		0.6448
ERM, VMT, MH	26 (66.7)	
Retinal detachment	11 (28.2)	
Dislocated lens	2 (5.1)	
Treatment for CME, n (%)		
Triamcinolone acetonide (TCA)	25 (64.1)	
Bevacizumab (Avastin)	3 (7.7)	
TCA + bevacizumab	11 (28.2)	

IQR – interquartile range; PPV – pars plana vitrectomy; CME – cystoid macular edema; ERM – epiretinal membrane; VMT – vitreomacular traction; MH – macular hole

Table 2.

Treatment outcomes for all eyes that received TCA for post-vitrectomy CME

	Mean (± Standard Deviation) (n=36)	P value*
Central foveal thickness (in um)		
Baseline	444.38 ± 144.72	
1 month	398.50 ± 157.54	0.2950
3 months	382.70 ± 133.10	**0.0171**
6 months	389.93 ± 127.67	**0.0401**
12 months	346.93 ± 136.27	**0.0024**
ETDRS visual acuity (20/denominator shown)		
Baseline	119.86 ± 82.19	
1 month	93.85 ± 62.07	**0.0169**
3 months	112.84 ± 81.90	0.4862
6 months	124.97 ± 110.70	0.9098
12 months	139.49 ± 135.93	0.4312
Intraocular Pressure (in mmHg)		
Baseline	15.28 ± 4.57	
1 month	18.93 ± 8.27	0.1185
3 months	16.06 ± 6.05	0.4344
6 months	14.71 ± 5.01	0.5267
12 months	16.12 ± 7.08	0.3925

* paired T-test comparing baseline to 1, 3, 6, 12 months; TCA – triamcinolone acetonide; CME – cystoid macular edema; ETDRS – Early Treatment Diabetic Retinopathy Study

Table 3.

Treatment outcomes of TCA alone vs. combined TCA and bevacizumab for post-vitrectomy CME

	TCA alone Mean (±SD) (n=25)	P value*	Combined TCA + bevacizumab Mean (±SD) (n=11)	P value*
Central foveal thickness (in um)				
Baseline	455.96 (±148.15)		420.18 (±140.98)	
1 month	426.40 (±185.62)	0.8211	352.00 (±91.01)	0.3009
3 months	398.59 (±143.08)	**0.0416**	355.70 (±116.16)	0.2519
6 months	421.26 (±122.91)	0.1120	330.40 (±120.37)	0.2028
12 months	368.68 (±144.08)	**0.0104**	301.00 (±111.67)	0.1334
ETDRS visual acuity (20/denominator shown)				
Baseline	132.56 (±90.78)		91.00 (±50.44)	
1 month	100.16 (±63.85)	**0.0537**	76.71 (±57.91)	0.1041
3 months	122.65 (±89.18)	0.3529	95.00 (±66.86)	0.8352
6 months	128.96 (±104.61)	0.5626	116.64 (±127.49)	0.4601
12 months	150.14 (±140.01)	0.7064	118.18 (±131.19)	0.3930
Intraocular Pressure (in mmHg)				
Baseline	14.96 (±3.61)		16.00 (±6.39)	
1 month	17.37 (±6.26)	0.2350	22.22 (±11.18)	0.3040
3 months	17.00 (±6.22)	0.1051	14.27 (±5.55)	0.3323
6 months	15.78 (±5.16)	0.4173	12.46 (±4.01)	0.0576
12 months	15.27 (±5.29)	0.7123	17.82 (±9.85)	0.4215

*paired T-test comparing baseline to 1, 3, 6, 12 months within each group; TCA – triamcinolone acetonide; CME – cystoid macular edema; ETDRS – Early Treatment Diabetic Retinopathy Study; SD – standard deviation