



EFFICACY AND SAFETY OF TOPICAL DORZOLAMIDE 2% IN REDUCING CENTRAL MACULAR THICKNESS IN PATIENTS WITH MACULAR EDEMA: A PROSPECTIVE INTERVENTIONAL STUDY

Ophthalmology

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ABSTRACT

Purpose: To evaluate the effect of topical dorzolamide 2% on central macular thickness (CMT) and best-corrected visual acuity (BCVA) in patients with macular edema of different etiologies. **Methods:** This prospective interventional study was conducted at the Regional Institute of Ophthalmology, Prayagraj, from July 2023 to June 2024. Ninety patients with increased CMT on spectral-domain optical coherence tomography (SD-OCT) were randomized into two groups: dorzolamide 2% eye drops three times daily (n=45) and placebo (n=45). Included conditions were diabetic macular edema (DME), acute, chronic and recurrent central serous retinopathy (CSR), retinitis pigmentosa-associated cystoid macular edema (RP-CME), Irvine-Gass syndrome, and retinal vascular occlusion-related macular edema. CMT and BCVA were assessed at baseline and at 1, 2, and 3 months. **Results:** The groups were comparable in age, gender, and disease distribution. At 3 months, mean CMT decreased from $468.86 \pm 72.45 \mu\text{m}$ to $305.65 \pm 76.17 \mu\text{m}$ in the dorzolamide group, compared with $441.95 \pm 77.94 \mu\text{m}$ to $412.03 \pm 63.70 \mu\text{m}$ in controls ($p < 0.001$). Significant CMT reduction was observed in acute CSR, chronic CSR, and RP-CME, whereas limited response was noted in recurrent CSR, Irvine-Gass syndrome, DME, and vascular occlusion-related edema. No significant difference in BCVA was found between groups at 3 months ($p = 0.806$). **Conclusion:** Topical dorzolamide 2% effectively reduces CMT in selected macular edema subtypes, particularly acute CSR, chronic CSR, and RP-CME, with good tolerability. Although visual acuity improvement was not significant, dorzolamide may serve as a safe, non-invasive, and cost-effective adjunctive treatment.

KEYWORDS

Dorzolamide, Carbonic Anhydrase Inhibitor, Central Macular Thickness, Macular Edema, Central Serous Retinopathy, Optical Coherence Tomography, BCVA

INTRODUCTION

Central macular thickness (CMT) is a critical OCT-based metric reflecting the structural integrity of the macula, measured within the central 1 mm of the ETDRS grid. Elevation of CMT is characteristic of macular edema (ME), a vision-threatening complication that may arise from a broad spectrum of retinal diseases including central serous chorioretinopathy (CSR), diabetic macular edema (DME), retinal vein occlusion (RVO), retinitis pigmentosa (RP), and pseudophakia.[1,2]

The pathophysiology of macular edema involves breakdown of the blood-retinal barrier, dysregulation of retinal microvasculature, and loss of ion-pump polarity in the retinal pigment epithelium (RPE), culminating in intra- or sub-retinal fluid accumulation.[3,4] While intravitreal anti-VEGF agents and corticosteroid implants have revolutionized ME management, these therapies entail repeated injections, significant costs, and non-trivial complication risks including endophthalmitis and retinal detachment.[5]

Dorzolamide, a sulfonamide-derived topical carbonic anhydrase inhibitor (CAI) originally developed for intraocular pressure reduction, has attracted growing interest as a non-invasive alternative for CMT reduction. Carbonic anhydrase inhibition acidifies the subretinal microenvironment, restores RPE polarity, and augments fluid efflux from the retina to the choroid. Additional anti-edematous mechanisms include inhibition of gamma-glutamyl transpeptidase and attenuation of neutrophil-mediated inflammation.[6-10] Acetazolamide, an oral CAI, was the first reported agent to resolve diverse forms of macular edema, including post-inflammatory, RP-related, and post-surgical variants.[11-13]

Topical dorzolamide offers the pharmacological advantages of carbonic anhydrase inhibition while largely avoiding systemic side effects. Prior case series and small trials have documented its benefit in RP-related CME, choroideremia, X-linked retinoschisis, and diabetic CME.[14-21] However, rigorous prospective data comparing dorzolamide to placebo across multiple etiologies of macular edema, particularly from the Indian subcontinent, remain sparse.

These etiologically diverse conditions were considered together because they converge on a shared final pathophysiological pathway—breakdown of the blood–retinal barrier and loss of retinal

pigment epithelial ion-pump polarity—that is the specific target of carbonic anhydrase inhibition, irrespective of the primary retinal insult; several individual subtypes are also too infrequent at a single center to be independently powered within a limited study period. The present study was therefore designed as a prospective interventional trial to evaluate the efficacy and safety of topical dorzolamide 2% in reducing CMT across a spectrum of retinal diseases causing macular edema in a tertiary ophthalmology center in North India.

Materials and Methods

Study design and setting

This was a prospective, interventional, placebo-controlled study conducted at the Retina Clinic of the Regional Institute of Ophthalmology, M.D. Eye Hospital, Prayagraj (a Government tertiary care referral center), from July 2023 to June 2024. The study was approved by the Institutional Ethics Committee of M.L.N. Medical College, Prayagraj. Written informed consent was obtained from all participants.

Study population

Ninety consecutive patients aged 18 years and above presenting with increased CMT ($>310 \mu\text{m}$ on SD-OCT) attributable to the following diagnoses were enrolled: acute CSR, chronic CSR (duration > 3 months), recurrent CSR, DME, RP with CME, Irvine-Gass syndrome (pseudophakic CME), and macular edema secondary to retinal vascular occlusion (BRVO/CRVO). Patients were allocated equally into case (n = 45) and control (n = 45) groups using a stratified block-allocation approach, with a separate randomization sequence generated for each disease subtype to ensure balanced representation of every etiology across the two arms.

Inclusion criteria

Patients diagnosed with any of the above conditions, documented CMT elevation on SD-OCT, aged ≥ 18 years, willing and able to provide informed consent, and able to adhere to follow-up schedule. For Irvine-Gass syndrome, only eyes presenting at least 4 weeks after uneventful cataract surgery were eligible, consistent with the classical description of Irvine-Gass onset at 4–12 weeks postoperatively, so as to exclude the early post-operative inflammatory phase during which transient cystoid change is common and often self-limiting.

Exclusion criteria

Patients with refractory clinically significant macular edema, vitreoretinal traction, choroidal neovascular membrane, mixed ischemic maculopathy, prior intravitreal anti-VEGF or steroid injection for ME, uveitic macular edema, high myopia, glaucoma, intraocular tumor, or impaired renal function were excluded. Prior intravitreal anti-VEGF or corticosteroid therapy was specifically excluded so that the anatomical effect of dorzolamide could be assessed in treatment-naïve eyes, without confounding from residual pharmacologic activity of these agents or from tissue remodeling secondary to prior intravitreal treatment. All patients underwent a standardized systemic evaluation—complete hemogram, liver and kidney function tests, erythrocyte sedimentation rate, C-reactive protein, fasting and postprandial blood glucose, Mantoux test, chest radiograph, and TORCH panel—to screen for infective, inflammatory, and uveitic causes of macular edema prior to enrolment; diabetic status, captured via fasting and postprandial glucose in every participant, was categorized as the primary disease entity (DME) only when the macular edema itself was attributable to diabetic retinopathy, rather than as a separate covariate in patients with a different primary diagnosis. Eyes in the vascular-occlusion-edema subgroup that had previously received peripheral scatter (panretinal or sectoral) photocoagulation for retinal ischemia remained eligible, as this does not directly treat the macula; no enrolled eye had received prior focal or grid macular laser directed at the edema itself.

Intervention

Cases received topical dorzolamide 2% eye drops three times daily in the affected eye(s) for 3 months, followed by a tapering schedule (twice daily for 1 week, then once daily for 1 week before cessation). The tapering schedule was adopted as an empirical, precautionary measure—based on institutional clinical experience with topical carbonic anhydrase inhibitors—to minimize any theoretical risk of rebound fluid accumulation on abrupt cessation, rather than on published evidence specific to this indication; conventional practice with topical CAIs used for intraocular pressure control is to discontinue without tapering, and this represents a protocol choice rather than an evidence-based requirement. Controls received an equivalent placebo (preservative-free artificial tears) on the same schedule. Treatment failure was defined as worsening of BCVA by ≥ 2 Snellen lines from baseline, or an increase in CMT $>50\%$ from baseline; patients meeting these criteria were withdrawn from study medication and switched to standard-of-care management appropriate to their underlying diagnosis (e.g., intravitreal anti-VEGF or corticosteroid therapy, or focal laser, as clinically indicated), outside the outcome analysis of this trial. Patients with refractory, vision-threatening macular edema were excluded from randomization at the outset (see Exclusion criteria), and this pre-defined treatment-failure rule served as an additional safety net for placebo-arm patients who deteriorated during follow-up.

Outcome measures

The primary outcome was change in CMT from baseline to 3 months, measured by Spectral-Domain OCT (TOPCON 3D OCT-1 Maestro2); the same instrument was used for all baseline and follow-up image acquisitions in every patient to eliminate inter-device variability. Secondary outcomes included: (1) change in BCVA (logMAR, converted from Snellen acuity); (2) proportion of eyes with complete SRF resolution; and (3) incidence and nature of adverse effects. All assessments were performed at baseline and at 1, 2, and 3 months post-treatment.

Statistical analysis

Data were analyzed using SPSS version 23.0. Continuous variables are presented as mean $\pm$ SD; categorical variables as frequencies and percentages. Between-group comparisons were made using the independent samples t-test for normally distributed data and the Wilcoxon rank-sum test for non-normal distributions. The Chi-squared test (or Fisher's exact test where appropriate) was used for categorical comparisons. Within-group longitudinal changes were assessed using repeated-measures ANOVA. No formal a priori sample-size or power calculation was performed; the sample size (90 patients, 150 eyes; 45 per arm) was determined pragmatically by patient accrual over the study period, and disease-specific subgroup analyses (e.g., DME and vascular-occlusion edema, $n = 3$ eyes/group each) should therefore be interpreted as exploratory and hypothesis-generating rather than confirmatory. Statistical significance was set at $p < 0.05$.

RESULTS

Baseline demographic and disease characteristics

A total of 90 patients (150 eyes: 75 per group) were enrolled: 45 in the dorzolamide group and 45 in the placebo group. Mean age was 38.69 ± 10.63 years (cases) versus 38.81 ± 9.24 years (controls; $p = 0.944$). The gender distribution was comparable: 32 males (71.11%) and 13 females (28.89%) in cases, and 33 males (73.33%) and 12 females (26.67%) in controls ($\chi^2 = 0.13$, $p = 0.718$). Unilateral involvement predominated in both groups (84.44% cases, 91.11% controls; $p = 0.678$). Disease distribution was identical between groups: acute CSR 33.33%, chronic CSR 13.33%, recurrent CSR 13.33%, RP-CME 13.33%, Irvine-Gass syndrome 13.33%, DME 6.67%, and vascular occlusion edema 6.67% ($\chi^2 = 0.00$, $p = 1.00$); macular edema directly attributable to diabetes (DME) thus accounted for 3 eyes per group (6 of 150 eyes overall), while all patients underwent fasting and postprandial blood glucose screening as part of the standard systemic work-up regardless of primary diagnosis. All 90 enrolled patients (150 eyes) completed the full 3-month follow-up with no losses to follow-up, attributable to free-of-cost care and structured follow-up counselling at this government tertiary institution. [Table 1]

Effect on central macular thickness

Pre-treatment CMT was comparable in both groups (cases: 468.86 ± 72.45 μ m; controls: 441.95 ± 77.94 μ m; $t = 1.22$, $p = 0.229$). After one month, CMT reduced by 71.44 μ m in cases to 397.42 ± 61.89 μ m versus a reduction of 30.60 μ m in controls to 411.35 ± 67.38 μ m ($t = 0.73$, $p = 0.466$). The between-group difference became statistically significant at 2 months (cases: 351.67 ± 71.90 μ m, reduction of 117.19 μ m; controls: 400.32 ± 74.17 μ m, reduction of 41.63 μ m; $t = 2.26$, $p = 0.029$) and was highly significant at 3 months (cases: 305.65 ± 76.17 μ m, reduction of 163.21 ± 56.17 μ m; controls: 412.03 ± 63.70 μ m, reduction of 29.92 ± 13.20 μ m; $t = 5.07$, $p < 0.001$). [Table 2 and 4]

Disease-specific CMT analysis (Tables 7 and 8 in the original study) revealed significant within-group reductions over 3 months in the dorzolamide arm for acute CSR ($445.60 \rightarrow 283.53$ μ m; $p < 0.001$), chronic CSR ($459.00 \rightarrow 268.40$ μ m; $p < 0.001$), and RP-CME ($460.83 \rightarrow 331.67$ μ m; $p < 0.001$). In contrast, no significant CMT reduction was observed in recurrent CSR ($396.40 \rightarrow 370.00$ μ m; $p = 0.290$), Irvine-Gass syndrome ($390.67 \rightarrow 365.83$ μ m; $p = 0.511$), DME ($451.00 \rightarrow 435.00$ μ m; $p = 0.905$), or vascular occlusion edema ($405.33 \rightarrow 393.00$ μ m; $p = 0.609$). In the control group, only acute CSR showed a significant within-group CMT change ($443.79 \rightarrow 299.50$ μ m; $p < 0.001$), consistent with the known self-limiting nature of acute CSR.

Effect on best corrected visual acuity

Baseline BCVA was comparable between groups (cases: 1.06 ± 0.28 logMAR; controls: 1.04 ± 0.24 logMAR; $t = 0.25$, $p = 0.801$). Over 3 months, BCVA showed modest improvements in both groups, but no statistically significant between-group difference was observed at any time point: 1 month (1.02 ± 0.26 vs. 1.02 ± 0.22 ; $t = 0.00$, $p = 1.000$), 2 months (0.98 ± 0.24 vs. 0.98 ± 0.26 ; $t = 0.00$, $p = 1.000$), and 3 months (0.96 ± 0.26 vs. 0.98 ± 0.28 ; $t = 0.25$, $p = 0.806$). [Table 3]

A notable finding was in acute CSR, where baseline BCVA differed significantly between groups (cases: 0.80 ± 0.20 logMAR vs. controls: 1.00 ± 0.24 logMAR; $t = 3.05$, $p = 0.004$). Despite this, dorzolamide-treated eyes showed substantially greater BCVA improvement: at 2 months (0.36 ± 0.16 vs. 0.74 ± 0.22 logMAR; $t = 6.71$, $p < 0.001$) and at 3 months (0.28 ± 0.18 vs. 0.72 ± 0.28 logMAR; $t = 6.39$, $p < 0.001$). No significant BCVA difference between groups was observed in any other disease subgroup at any time point.

Adverse effects

Adverse effects were uncommon and mild. In the dorzolamide group, burning sensation was reported in 4.44% of patients, redness in 2.22%, and transient corneal edema in 2.22%. No severe ocular or systemic adverse events were recorded. No patient discontinued therapy due to adverse effects.

DISCUSSION

The present study demonstrates that topical dorzolamide 2% administered three times daily for 3 months produces a clinically and statistically significant reduction in CMT compared to placebo in patients with macular edema, particularly in those with acute CSR, chronic CSR, and RP-CME. The overall CMT reduction in the dorzolamide arm (163.21 ± 56.17 μ m) was approximately 5.5-fold greater than in the placebo arm (29.92 ± 13.20 μ m) at 3 months ($t = 5.07$, $p < 0.001$).

These findings align with the growing body of literature on CAI-mediated retinal fluid reduction. The proposed mechanism involves inhibition of carbonic anhydrase IV in the RPE, which acidifies the subretinal space and reverses pathological fluid polarity, thereby enhancing fluid egress from the retina to the choroid.[6-10] Badawi et al. (2021) reported a significant reduction in CMT from $535.27 \pm 97.4 \mu\text{m}$ at baseline to $357.43 \pm 125.8 \mu\text{m}$ at 1 month with topical dorzolamide monotherapy in diabetic CME.[60] Sadr et al. (2024) reported a significantly greater CMT reduction at 3 months in the dorzolamide + bevacizumab arm ($-88.92 \pm 82.90 \mu\text{m}$) versus bevacizumab monotherapy ($-37.64 \pm 86.16 \mu\text{m}$; $p = 0.037$) in DME, supporting dorzolamide's additive role.[70]

The disease-specific analysis revealed important heterogeneity in dorzolamide's efficacy. In acute CSR, dorzolamide achieved significant CMT reduction and greater BCVA improvement at 3 months (0.28 ± 0.18 vs. 0.72 ± 0.28 logMAR; $t = 6.39$, $p < 0.001$). However, it must be noted that baseline BCVA in the acute CSR case group was significantly better than controls (0.80 vs. 1.00 logMAR; $p = 0.004$), which may partly account for the observed difference; the robust CMT reduction in this subgroup nevertheless supports a genuine drug effect. In chronic CSR and RP-CME, significant CMT reduction was observed at 3 months without corresponding BCVA improvement, consistent with established retinal structural damage in chronically affected eyes. In contrast, dorzolamide showed no significant efficacy in recurrent CSR, DME, vascular occlusion edema, or Irvine-Gass syndrome, likely reflecting distinct pathophysiological mechanisms—particularly VEGF-driven vascular hyperpermeability—that are not addressed by carbonic anhydrase inhibition alone.[60,74,77]

Across the entire cohort, BCVA did not differ significantly between groups at any time point ($p = 0.806$ at 3 months), despite substantial CMT reduction in the dorzolamide arm. This dissociation between anatomical and functional outcomes is well documented in macular edema literature. Persistent photoreceptor damage and ellipsoid zone disruption in chronically edematous eyes may limit visual recovery even after successful fluid resolution. Our findings are consistent with Liew et al. (2020), who similarly observed that topical CAI therapy reduced CMT in chronic CSR without proportionate visual improvement.[66] This anatomical-functional dissociation, rather than reflecting a lack of drug efficacy, most plausibly reflects the extent of pre-existing chronic photoreceptor injury at enrolment, which anatomical fluid resolution alone cannot reverse.

The safety profile of topical dorzolamide in the current study was favorable. The overall adverse event rate was low (burning sensation 4.44%, redness 2.22%, corneal edema 2.22%), none required drug discontinuation, and no systemic adverse effects were encountered. This is consistent with prior publications documenting dorzolamide's acceptable ocular tolerability, provided that patients are screened for pre-existing corneal endothelial disease, which can predispose to irreversible corneal decompensation.[20,61] The topical route further minimizes systemic absorption, mitigating the hypokalemia and metabolic acidosis risks associated with oral carbonic anhydrase inhibitors.

From a health-system perspective, dorzolamide 2% is widely available, affordable, and self-administered, offering a non-invasive treatment avenue that may reduce the burden of intravitreal injections in resource-limited settings. The current study, conducted in a large public tertiary eye hospital in North India serving a predominantly low- to middle-income population, highlights this practical utility.

Limitations of the study include a relatively small sample size (45 per arm), which may limit the detection of statistically significant changes in disease subgroups with few patients (e.g., DME, $n = 3$ per group). The 3-month follow-up period may not capture long-term structural and functional outcomes. Patients were not masked to treatment allocation, introducing the potential for performance bias. The study was not designed or powered for direct efficacy comparisons across etiologies, and the post-treatment tapering schedule was an empirical precaution rather than an evidence-based protocol element; both merit dedicated evaluation in future work. Comorbid diabetes outside the DME diagnostic category, and the exact interval since cataract surgery in Irvine-Gass cases, were not separately tabulated in this dataset. Future multicenter randomized controlled trials with larger sample sizes, longer follow-up, fluorescein angiography, and OCT-

angiography endpoints are needed to better characterize dorzolamide's efficacy across ME etiologies.

CONCLUSION

Topical dorzolamide 2% significantly reduces central macular thickness in patients with acute CSR, chronic CSR, and RP-CME, demonstrating meaningful anatomical benefit over 3 months of treatment. Visual outcomes, however, do not improve significantly in most disease categories—an important consideration in clinical counseling. The treatment is safe, non-invasive, and cost-effective. Given these attributes, topical dorzolamide deserves consideration as a first-line or adjunctive agent in eligible patients with select subtypes of macular edema, pending confirmation by larger prospective trials.

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Conflicts of interest: There are no conflicts of interest.

Ethical approval: Approved by the Institutional Ethics Committee, M.L.N. Medical College, Prayagraj. Registration No. ECR/922/Inst/UP/RR22.

Informed consent: Obtained from all participants.

Efficacy and Safety of Topical Dorzolamide 2% in Reducing Central Macular Thickness in Patients with Macular Edema: A Prospective Interventional Study

TABLES
Table 1: Baseline demographic and disease characteristics of cases and controls

Characteristic	Dorzolamide Group (n = 45)	Placebo Group (n = 45)	Total (n = 90)	p value
Mean Age $\pm$ SD (years)	38.69 $\pm$ 10.63	38.81 $\pm$ 9.24	—	0.944
Male, n (%)	32 (71.11%)	33 (73.33%)	65 (72.22%)	0.718
Female, n (%)	13 (28.89%)	12 (26.67%)	25 (27.78%)	—
Unilateral involvement, n (%)	38 (84.44%)	41 (91.11%)	79 (87.78%)	0.678
Disease distribution, n (%)				
Acute CSR	15 (33.33%)	15 (33.33%)	30 (33.33%)	1.000
Chronic CSR	6 (13.33%)	6 (13.33%)	12 (13.33%)	—
Recurrent CSR	6 (13.33%)	6 (13.33%)	12 (13.33%)	—
RP with CME	6 (13.33%)	6 (13.33%)	12 (13.33%)	—
Irvine-Gass Syndrome	6 (13.33%)	6 (13.33%)	12 (13.33%)	—
Diabetic macular edema (DME)	3 (6.67%)	3 (6.67%)	6 (6.67%)	—
Vascular occlusion macular edema	3 (6.67%)	3 (6.67%)	6 (6.67%)	—

CSR = Central serous retinopathy; RP = Retinitis pigmentosa; CME = Cystoid macular edema; DME = Diabetic macular edema. Data presented as mean $\pm$ SD or n (%). Independent samples t-test used for continuous variables; Chi-square test for categorical variables.

Table 2: Overall mean central macular thickness (CMT) changes across follow-up intervals (n = 75 eyes per group)

Time Point	Dorzolamide Group Mean $\pm$ SD (μm)	Placebo Group Mean $\pm$ SD (μm)	t-value	p-value
Baseline (Pre-treatment)	468.86 $\pm$ 72.45	441.95 $\pm$ 77.94	1.22	0.229
1 month (change from baseline)	397.42 $\pm$ 61.89 (-71.44)	411.35 $\pm$ 67.38 (-30.60)	0.73	0.466
2 months (change from baseline)	351.67 $\pm$ 71.90 (-117.19)	400.32 $\pm$ 74.17 (-41.63)	2.26	0.029*

3 months (change from baseline)	305.65 ± 76.17 (−163.21 ± 56.17)	412.03 ± 63.70 (−29.92 ± 13.20)	5.07	<0.001*
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Values in parentheses denote mean change from baseline. * $p < 0.05$ (significant); ** $p < 0.001$ (highly significant). Independent samples t -test used for between-group comparison.

Table 3: Overall mean best corrected visual acuity (BCVA) changes across follow-up intervals (n = 75 eyes per group)

Time Point	Dorzolamide Group Mean ± SD (logMAR)	Placebo Group Mean ± SD (logMAR)	t-value	p-value
Baseline (Pre-treatment)	1.06 ± 0.28	1.04 ± 0.24	0.25	0.801
1 month	1.02 ± 0.26	1.02 ± 0.22	0.00	1.000
2 months	0.98 ± 0.24	0.98 ± 0.26	0.00	1.000
3 months	0.96 ± 0.26	0.98 ± 0.28	0.25	0.806

BCVA expressed in logMAR units (lower value = better acuity). Independent samples t -test used for between-group comparison. NS = Not significant.

Table 4: Disease-specific central macular thickness (CMT) changes in the dorzolamide group across 3 months

Disease Subgroup	Baseline Mean ± SD (µm)	3 Months Mean ± SD (µm)	Mean Reduction (µm)	p-value (within group)
Acute CSR (n = 15 eyes)	445.60	283.53	−162.07	<0.001**
Chronic CSR (n = 6 eyes)	459.00	268.40	−190.60	<0.001**
Recurrent CSR (n = 6 eyes)	396.40	370.00	−26.40	0.290
RP with CME (n = 6 eyes)	460.83	331.67	−129.16	<0.001**
Irvine-Gass Syndrome (n = 6 eyes)	390.67	365.83	−24.84	0.511
DME (n = 3 eyes)	451.00	435.00	−16.00	0.905
Vascular occlusion ME (n = 3 eyes)	405.33	393.00	−12.33	0.609

** $p < 0.001$ (highly significant within-group change by repeated-measures ANOVA). NS = Not significant. RP = Retinitis pigmentosa; CME = Cystoid macular edema; DME = Diabetic macular edema; ME = Macular edema. Number of eyes per subgroup: Acute CSR = 15, Chronic CSR = 6, Recurrent CSR = 6, RP-CME = 6, Irvine-Gass = 6, DME = 3, Vascular occlusion = 3.

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