MICROPERIMETRIC CHANGES AFTER INTRAVITREAL TRIAMCINOLONE ACETONIDE INJECTION FOR MACULAR EDEMA DUE TO CENTRAL RETINAL VEIN OCCLUSION

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Purpose: The purpose of this study was to evaluate the effect of intravitreal triamcinolone acetonide on macular function in cases of macular edema because of central retinal vein occlusion.

Methods: Twelve eyes of 12 patients with central retinal vein occlusion were included in this study. In each eye, at baseline and 1, 3, and 6 months after intravitreal triamcinolone acetonide injection, logarithm of the minimum angle of resolution visual acuity, macular sensitivity, fixation stability and fixation location by MP-1 microperimetry, and foveal thickness by optical coherence tomography were assessed.

Results: Patients' ages ranged from 50 to 75 years (mean ± SD, 59 ± 8 years). All patients were classified as nonischemic. At 1, 3, and 6 months, the mean foveal thickness had decreased from 453 ± 108 μm to 254 ± 40.3 μm, 297 ± 90 μm, and 320 ± 82 μm and the mean retinal sensitivity had increased from 5.5 ± 3.3 dB to 9.4 ± 3.5 dB, 7.8 ± 3.3 dB, and 7.2 ± 4.2 dB, respectively. At baseline, fixation was stable in one, relatively unstable in six, and unstable in five eyes. However, 6 months after intravitreal triamcinolone acetonide injection, fixation was stable in 8, relatively unstable in 3, and unstable in one. At baseline, in eyes with macular edema, fixation location was predominantly central in 2, poor central in 4, and predominantly eccentric in 6. And 6 months after treatment, fixation location was predominantly central in 8, poor central in 3, and predominantly eccentric in 1.

Conclusion: In eyes with macular edema in central retinal vein occlusion, a short-term improvement in retinal sensitivity and fixation properties can be achieved by intravitreal triamcinolone acetonide injection.

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Central retinal vein occlusion (CRVO) is a retinal vascular disorder often complicated by macular edema (ME). The natural history of the disorder reveals that 47% of eyes with perfused CRVO and initial visual acuity 20/50 or worse have final visual acuity of 20/250 in 3 years.[1] The CRVO Study Group, in a double-masked study of 155 eyes with visual acuity 20/50 or worse, evaluated the efficacy of grid laser photocoagulation in the management of ME caused by CRVO.[2] Intravitreal triamcinolone acetonide injection may stabilize leaky vascular endothelium, reducing the extracellular fluid accumulation that causes ME, perhaps by downregulating vascular endothelial growth factor.[3] Intravitreal triamcinolone acetonide has been used to treat various intraocular neovascular, proliferative, and edematous diseases.[4–7] It has also been shown that intravitreal triamcinolone acetonide injection is effective for treating ME due to

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CRVO.[8–10] In these studies, visual acuity and morphologic features before and after intravitreal triamcinolone acetonide injection were evaluated, but patients' subjective appraisal of their visual function, which may differ, was not comprehensively discussed.

The purpose of this study was to obtain a measure of macular function before and after intravitreal triamcinolone acetonide injection in patients with ME due to CRVO. We used microperimetry to compare the sensitivity of the fovea before and after intravitreal triamcinolone acetonide injection. The fixation stability and location before and after injection were also determined by microperimetry.

Material and Methods

In this clinical trial, 12 eyes of 12 patients with ME due to CRVO (9 men and 3 women) were evaluated. Patients' ages ranged from 50 to 75 years (mean ± SD, 59 ± 8 years). The eligibility criteria included presence of ME due to CRVO identified by fundus examination, presence of angiographically confirmed ME documented by optical coherence tomography (OCT), no evidence of ocular disorders that might potentially result in ME, such as diabetic retinopathy, uveitis, macular pucker, or vitreomacular traction, and no evidence of glaucoma or ocular hypertension. Because several diseases may influence microperimetry and visual acuity, we excluded patients with moderate to dense lens opacity, implanted intraocular lenses, corneal opacities, a history of refractive surgery, a history of intraocular inflammation such as anterior or posterior uveitis, multifocal choroiditis, a history of retinal detachment, a history of ocular trauma, or optic neuropathy. In this consecutive series, no eyes had received previous laser photocoagulation. Each patient was informed of the off-label status of triamcinolone acetonide, and informed consent was obtained. This study was performed in accordance with the ethical standards laid down in the Declaration of Helsinki.

All eyes underwent complete ophthalmic examination, including best-corrected visual acuity measurement (with Early Treatment Diabetic Retinopathy Study chart), slit-lamp biomicroscopy, indirect ophthalmoscopy, color fundus photography, fluorescein angiography, and OCT. Best-corrected visual acuity, expressed as logarithm of the minimum angle of resolution (logMAR), was obtained from a distance of 4 m. Fluorescein angiograms were performed on a Heidelberg scanning laser ophthalmoscope (Heidelberg Engineering, Heidelberg, Germany). Optical coherence tomography examinations were performed using the OCT 3,000 scanner (Carl Zeiss Ophthalmic System Inc., Humphrey Division, Dublin, CA). All OCT examinations were done by the same operator, and all scans were done with a scan length of 6 mm. The foveal thickness was defined as the distance between the vitreoretinal interface and the retinal pigment epithelium in the center of the fovea. Macular edema was evident on fluorescein angiography from the typical oval or petaloid hyperfluorescent cystoid spaces radiating from the fovea and on OCT from hyporeflective intraretinal cavities radiating from the center of the macula on cross-sectional scans.

For the injection of triamcinolone acetonide (Kenacort-A, 40 mg/mL; Bristol-Myers Squibb Co, Princeton, NJ), topical proparacaine hydrochloride was applied to the ocular surface followed by preparation with 5% povidone iodine. A cotton-tipped applicator soaked in proparacaine hydrochloride was then applied to the injection site 4 mm posterior to the limbus. The injection consisted of 0.1 mL (4 mg) of a commercially available suspension of triamcinolone acetonide. Indirect ophthalmoscopy was used to confirm proper intravitreal localization of the suspension. Patients were examined on Days 1 and 7 to detect any infection. The response to treatment was monitored functionally by visual acuity and microperimetry assessment and anatomically by OCT foveal thickness after injection. Potential corticosteroid-induced and injection-related complications were also recorded.

Macular sensitivity was evaluated by MP-1 microperimetry (Nidek, Padova, Italy). The MP-1 provides a 45° nonmydriatic view of the fundus with an automated correction for eye movements. Goldmann III stimuli and a 4-2 staircase strategy were used, and a circular test grid with 74 stimulus locations covering an area of 20° was applied. The stimuli were projected on a white background with background illumination set to 1.27 cd/m^2 and a stimulus presentation time of 200 milliseconds. The perimetric strategy of the current software version of the MP-1 starts at an initially defined threshold level for each stimulus. A 4-2 staircase strategy is then carried out, and the last seen threshold value is taken as the final threshold. Although the examiner can define the initial threshold value, the actual threshold of the examined eye remains unaccounted for. The instrument tests the same luminance levels at all test locations before moving on to the next luminance level (i.e., for all locations, one luminance level is projected after the other). Differential light threshold values were compared by calculating 74 points averaged automatically by the MP-1 microperimetry software program of mean sensitivity in a polygon.

All patients had to demonstrate good collaboration in performing a microperimetry test, which means

a prompt and correct understanding of the technique and a good concentration capacity. Each patient underwent a preliminary practice test before the definitive microperimetry test to avoid learning effect.

For assessment of fixation, the fundus movements are tracked during examination while the patient gazes at the fixation target. The autotracking system calculates horizontal and vertical shifts relative to a reference frame and draws a map of the patient's eye movements during the examination. The recorded fixation points are classified into three categories for fixation stability analysis (stable, relatively unstable, and unstable). Fixation is regarded as ''stable'' if more than 75% of the fixation points are inside a 2° diameter circle, as ''relatively unstable'' if less than 75% are inside the 2° diameter circle but more than 75% inside a 4° diameter circle, and as ''unstable'' if less than 75% of the fixation points are inside the 4° diameter circle. To assess the fixation location, a standard, circular, central fixation area 2° in diameter (approximately 700 μm) centered on the fovea is defined. Eyes with more than 50% of the preferred fixation points located within the central area are classified as having predominantly central fixation. Eyes with more than 25% but less than 50% of preferred fixation points located within the central area are classified as having poor central fixation. Eyes with less than 25% of the preferred fixation points located within the central area are classified as having predominantly eccentric fixation. Fixation locations are classified automatically by the MP-1 microperimetry software after a landmark has been positioned in the center of the foveal avascular zone. For statistical analysis, nonparametric data fixation location is graded as: 3 = predominantly central, 2 = poor central, and 1 = predominantly eccentric, and fixation stability graded as: 3 = stable, 2 = relatively unstable, and 1 = unstable. We performed baseline MP-1 microperimetry before treatment and subsequent MP-1 microperimetry assessment 30 days after intravitreal triamcinolone acetonide injection, choosing a follow-up program that uses the same test parameters as in the baseline evaluation. Changes in visual acuity, retinal sensitivity, and foveal thickness (parametric data) in eyes with ME 1, 3, and 6 months after baseline intravitreal triamcinolone acetonide injection were compared with baseline values by the repeated analysis of variance and contrast tests. The MP-1 microperimetry fixation location and stability (nonparametric data) of eyes with ME after treatment were compared with baseline by the nonparametric Friedman analysis of variance and Dunn multiple comparisons tests. Statistical analysis was performed using the SPSS (Version 13.0; SPSS Inc., Chicago, IL). The $P < 0.05$ was considered statistically significant.

Results

The right eye was involved in five cases of ME and the left eye in seven. A history of hypertension was present in seven patients and a history of smoking in five. No patient had diabetes mellitus. All patients were classified as nonischemic. No patient had an afferent pupillary defect, areas of capillary nonperfusion on angiography, iris neovascularization, or vessels in the angle. Before triamcinolone injection,

Table 1. Clinical Characteristics of Cases with ME in CRVO

Case	Age (Years)	Visual Acuity (logMAR)				MP-1 Microperimetry Sensitivity (dB)				MP-1 Microperimetry ''Fixation Location''				MP-1 Microperimetry ''Fixation Stability''				OCT Foveal Thickness (μ)			
		B	1MO	3MO	6MO	B	1MO	3MO	6MO	B	1MO	3MO	6MO	B	1MO	3MO	6MO	B	1MO	3MO	6MO
1	53	1	0.7	0.5	0.7	5.6	8.4	7.1	3.5	1	3	3	3	2	2	3	2	610	352	240	313*
2	55	0.7	0.5	0.5	0.3	2.9	11.4	11.1	11.9	1	3	3	3	1	2	3	3	748	340	332	290
3	65	0.5	0.3	0.4	0.5	9.5	13.1	7.4	9.4	3	3	3	3	2	3	3	3	653	348	442*	400*
4	51	1	0.7	0.6	1	1.2	4.4	5.4	2.7	1	2	2	1	1	2	2	1	610	279	238	385*
5	54	0.4	0.3	0.3	0.4	5.8	11	10.7	6.1	1	3	3	2	1	3	3	3	550	310	210	409*
6	55	0.7	0.3	0.6	0.4	6	12.7	7.1	11.9	1	3	2	3	1	3	2	3	615	254	336*	220
7	50	1	0.7	1	0.7	2.6	7.6	2.5	6.2	1	3	1	3	1	3	2	3	667	315	446*	245
8	69	1	0.5	0.3	0.2	11.7	14.1	13.8	14	3	3	3	3	3	3	3	3	385	210	189	190
9	69	0.7	0.4	0.6	0.4	9.2	10.2	8.5	10.1	2	3	2	3	2	3	2	3	611	211	389*	245
10	52	1	0.7	0.5	0.7	1.3	2.7	3.1	0.7	2	3	2	2	2	3	2	2	550	320	230	434*
11	75	1	0.7	0.5	0.7	4.4	6.7	6.8	5.1	2	3	3	3	2	3	3	3	800	294	271	343*
12	56	1	0.4	0.3	0.3	5.8	10.1	10.5	5.3	2	3	3	2	2	3	3	2	380	263	240	370*

For fixation location 3, predominantly central; 2, poor central; 1, predominantly eccentric; for fixation stability 3, stable; 2, relatively unstable; 1, unstable. Foveal thickness column, *retreatments.

B, baseline (pretreatment); OCT, optical coherence tomography; 1MO, 1 month after treatment.

Table 2. The Visual Acuity, MP-1 Microperimetry Central 20° Retinal Sensitivity, and OCT Foveal Thickness in Eyes with Macular Edema at 1, 3, and 6 Months After Treatment Were Compared with Baseline with Repeated ANOVA and Contrast Tests

	Baseline	1MO	3MO	6MO
Visual acuity (logMAR), mean ± SD	0.83 ± 0.22	0.52 ± 0.17	0.51 ± 0.19	0.53 ± 0.23
Repeated ANOVA $F = 22.9$; $P < 0.001$	—	$P < 0.001$	$P < 0.01$	$P < 0.01$
MP-1 microperimetry retinal sensitivity (dB)	5.50 ± 3.32	9.37 ± 3.51	7.83 ± 3.32	7.24 ± 4.16
Repeated ANOVA $F = 15.5$; $P < 0.001$	—	$P < 0.001$	$P < 0.05$	NS
OCT foveal thickness (μ)	598.25 ± 124.07	291.33 ± 49.00	296.92 ± 89.85	320.33 ± 81.84
Repeated ANOVA $F = 34.7$; $P < 0.001$	—	$P < 0.001$	$P < 0.001$	$P < 0.001$

ANOVA, analysis of variance; NS, not significant; OCT, optical coherence tomography; 1MO, one month after treatment.

no eyes had been treated with systemic or local medication or with laser photocoagulation. At the 1-month examination, mean intraocular pressure ± SD had increased from 15.0 ± 1.8 mmHg to 18.0 ± 6.2 mmHg. At the 3- and 6-month examinations, it was 17.0 ± 5.6 mmHg and 15.0 ± 1.6 mmHg, respectively. Six eyes with intraocular pressure of >21 mmHg at an examination were treated with a beta-blocker at the subsequent examination. During follow-up, no cataract progression, endophthalmitis, or injection-related complication was encountered. The clinical characteristics of eyes with ME observed at baseline and 1, 3, and 6 months after treatment are reported in Table 1. At 1, 3, and 6 months, the mean foveal thickness had decreased from 453 ± 108 μm to 254 ± 40.3 μm, 297 ± 90 μm, and 320 ± 82 μm, respectively, the mean retinal sensitivity had increased from 5.5 ± 3.3 dB to 9.4 ± 3.5 dB, 7.8 ± 3.3 dB, and 7.2 ± 4.2 dB, respectively, and the mean visual acuity had increased from 0.83 ± 0.22 logMAR to 0.5 ± 0.2 logMAR, 0.5 ± 0.2 logMAR, and 0.5 ± 0.2 logMAR, respectively. Visual acuity, central 20° retinal sensitivity, and foveal thickness data at baseline and 1, 3, and 6 months after treatment are reported in Table 2. One month after treatment, eyes with ME showed a significant reduction in foveal thickness. There was also a significant increase in logMAR visual acuity and retinal sensitivity. Three months after treatment, eyes with ME showed a significant reduction in foveal thickness. There was also a significant increase in visual acuity and retinal sensitivity. Six months after treatment, eyes with ME showed a significant reduction in foveal thickness. There was also a significant increase in logMAR visual acuity, and none significantly increased but improved retinal sensitivity. Figures 1, 2, and 3 show the fluorescein angiography, MP-1 microperimetry, and OCT cross-sectional images of the fovea for Case 2 at baseline and 6 months after treatment, respectively.

At the 3-month follow-up, Cases 3, 6, 7, and 9 showed recurrence of ME; at the 6-month follow-up,

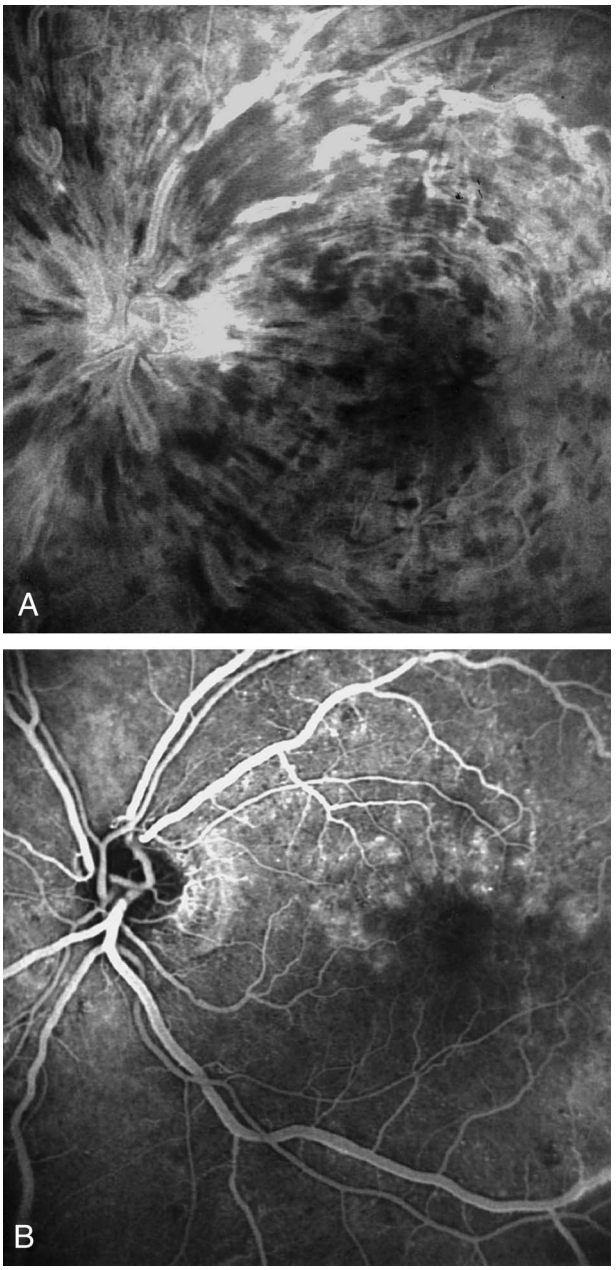

Fig. 1. Fluorescein angiography of Case 2 at baseline (**A**) and 6 months after treatment (**B**).

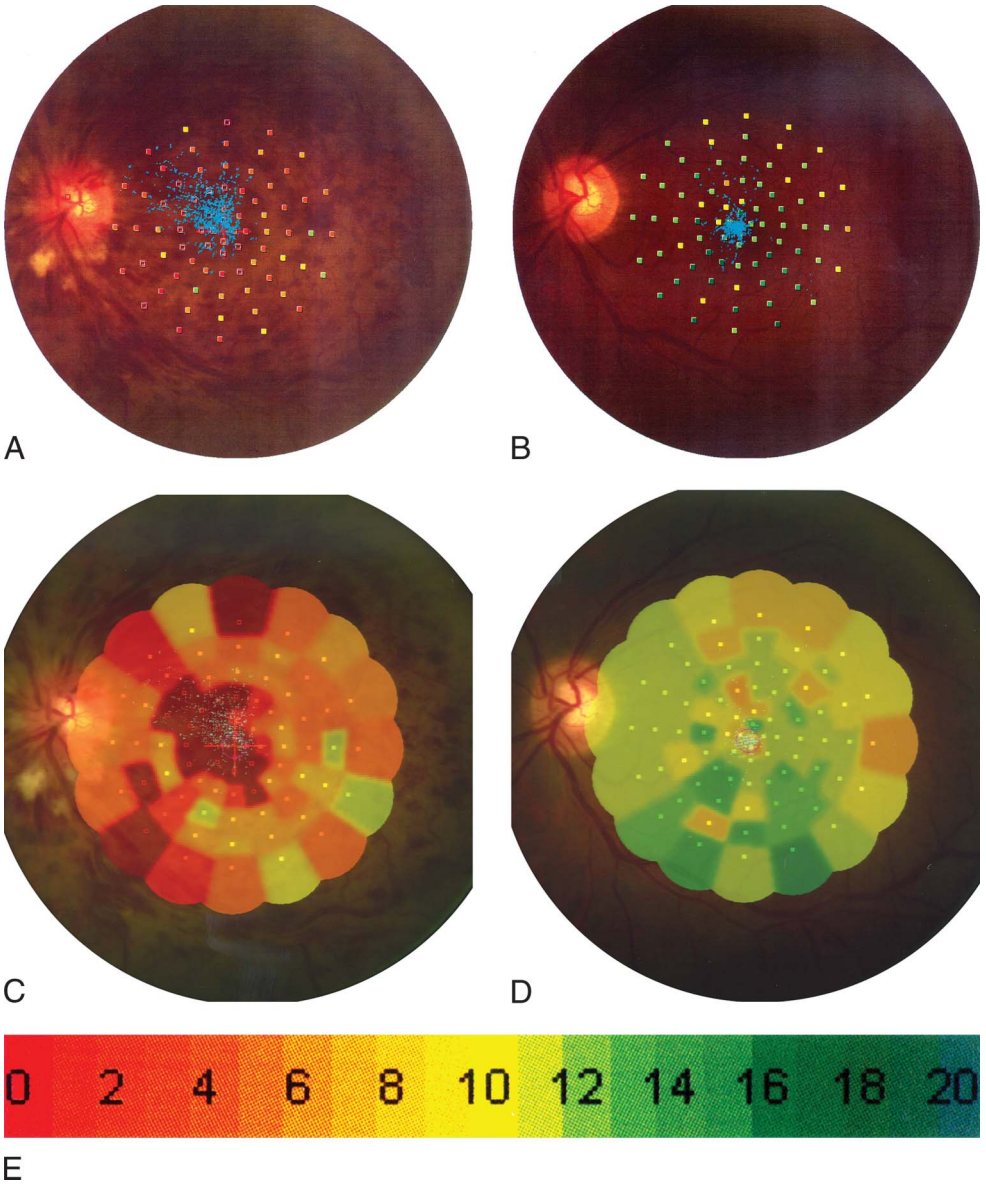

Fig. 2. MP-1 microperimetry images of Case 2. MP-1 image shows that reduced retinal sensitivity at the baseline examination (**A**), and MP-1 image shows that improved retinal sensitivity at the examination 6 months after IVTA injection (**B**). MP-1 color mapping image at the baseline examination (**C**) and at the 6 months after IVTA injection (**D**). E, Color scale. Blue data points represent the locations used for fixation during microperimetric test in the all MP-1 images. IVTA, intravitreal triamcinolone acetonide.

Cases 1, 3, 4, 5, 10, 11, and 12 showed recurrence of ME. These cases were successfully retreated.

At baseline, in eyes with ME, fixation location was predominantly central in two, poor central in four, and predominantly eccentric in six. Ten of 12 eyes with ME showed improvement of fixation location 1 month after treatment. Eyes with ME showed improvement of fixation location 1 month after treatment when compared with baseline. At the 3-month follow-up, 3 of 4 cases with recurrence of ME showed deterioration of fixation location, and after retreatment, fixation location improved again. Eyes with ME showed improvement of fixation location but not significantly 3 months after treatment when compared with baseline. At the 6-month follow-up, in 3 of 7 cases with recurrence of ME, fixation location had deteriorated. In eyes with ME, fixation location had improved but not significantly 6 months after treatment when compared with baseline. At the 6-month follow-up, in eyes with ME, fixation location was predominantly central in 8, poor central in 3, and predominantly eccentric in 1.

At baseline, fixation was stable in one, relatively unstable in six, and unstable in five. In 10 of 12 eyes with ME, fixation stability had improved 1 month after treatment. In eyes with ME, fixation stability had improved 1 month after treatment when compared with baseline. At the 3-month follow-up, in 3 of 4 eyes with recurrent ME, fixation stability had deteriorated and, after retreatment, had improved again at the

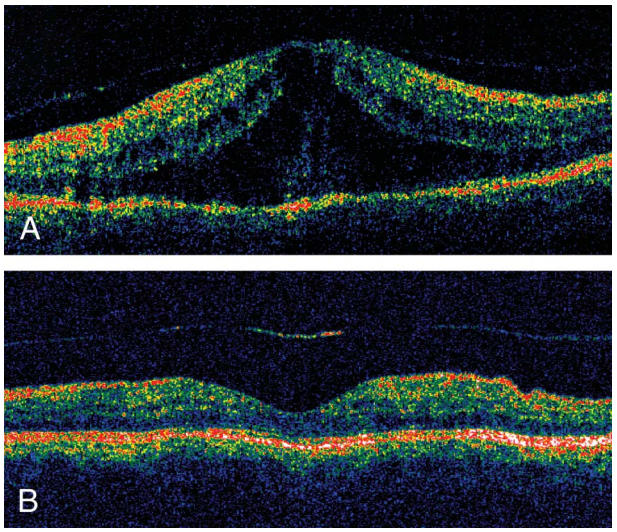

Fig. 3. Optical coherence tomography cross-sectional image of Case 2 at baseline (**A**) and 6 months after treatment (**B**).

6-month follow-up. Two eyes with improved fixation stability 1 month after treatment also showed much more improvement 3 months after treatment without recurrence of ME, but in 1 case with improved fixation stability 1 month after treatment, stability had deteriorated 3 months after treatment without recurrence, and 1 case showed no change of fixation stability even though ME recurred. In eyes with ME, fixation stability had improved 3 months after treatment when compared with baseline. At the 6-month follow-up, in 3 of 7 cases with recurrence of ME, fixation stability had deteriorated. In eyes with ME, fixation stability had improved 6 months after treatment when compared with baseline. At 6 months, fixation was stable in 8, relatively unstable in 3, and unstable in 1. Fixation location and stability data at baseline and 1, 3, and 6 months after treatment are reported in Table 3. Fixation location and stability changes after treatment of Case 2 are shown in Figure 4.

Discussion

Macular edema after CRVO is because of a disruption of the inner blood–retina barrier and is a common cause of visual loss. Triamcinolone acetonide is a corticosteroid suspension with no known retinal toxicity when injected intravitreally and has been shown to reduce breakdown of the inner blood–retina barrier.[11] A limited number of studies evaluating the efficacy of triamcinolone on ME in CRVO have had favorable results. Karacorlu et al[8] injected 4-mg triamcinolone acetonide in eyes of cases with ME and serous macular detachment in CRVO. Initially, the results for visual acuity and OCT findings were impressive, but 6 months later, some recurrences, with decrease of visual acuity and increase of foveal thickness, were observed.[8] Park et al[10] injected 4-mg triamcinolone acetonide in 10 eyes with nonischemic CRVO. After an average 4.8 months of follow-up, ME, on OCT findings, had significantly improved.

Gregori et al[12] reported their results of intravitreal injection of triamcinolone acetonide in eyes with ME in CRVO. Vision improved by 3 or more lines in 21% of eyes at 1 month, 27% at 3 months, 14% at 6 months, and 12% at 1 year.[12] In these studies, visual acuity is the standard measurement of vision. However, high-contrast visual acuity measurement is often a poor predictor of general visual performance. Important daily tasks, such as recognition of faces and symbols, orientation, and reading, are strongly dependent on the preservation of the central visual field.

The purpose of our study was to obtain a measure of macular function before and after intravitreal triamcinolone acetonide injection in cases with ME in CRVO. To accomplish this, microperimetry was performed on eyes with ME in CRVO before and after intravitreal triamcinolone acetonide injection and the sensitivity of the fovea and fixation alterations was determined from the result of microperimetry.

In the previous studies, visual acuity and morphologic features before and after intravitreal

Table 3. The MP-1 Microperimetry Fixation Location and Fixation Stability of Eyes with Macular Edema at 1, 3, and 6 Months After Treatment Were Compared with Baseline with Nonparametric Friedman ANOVA and Dunn Multiple Comparisons Tests

	Baseline	1MO	3MO	6MO
Fixation stability, mean ± SD (median)*	1.67 ± 0.65 (2)	2.75 ± 0.45 (3)	2.58 ± 0.51 (3)	2.58 ± 0.67 (3)
Friedman ANOVA chi-square = 17.52; $P < 0.01$	—	$P < 0.01$	$P < 0.05$	$P < 0.05$
Fixation location†	1.67 ± 0.77 (2)	2.92 ± 0.29 (3)	2.50 ± 0.67 (3)	2.58 ± 0.67 (3)
Friedman ANOVA chi-square = 18.61; $P < 0.001$	—	$P < 0.01$	NS	NS

*For fixation stability 3, stable; 2, relatively unstable; 1, unstable.
†For fixation location 3, predominantly central; 2, poor central; 1, predominantly eccentric.
ANOVA, analysis of variance; NS, not significant; 1MO, one month after treatment.

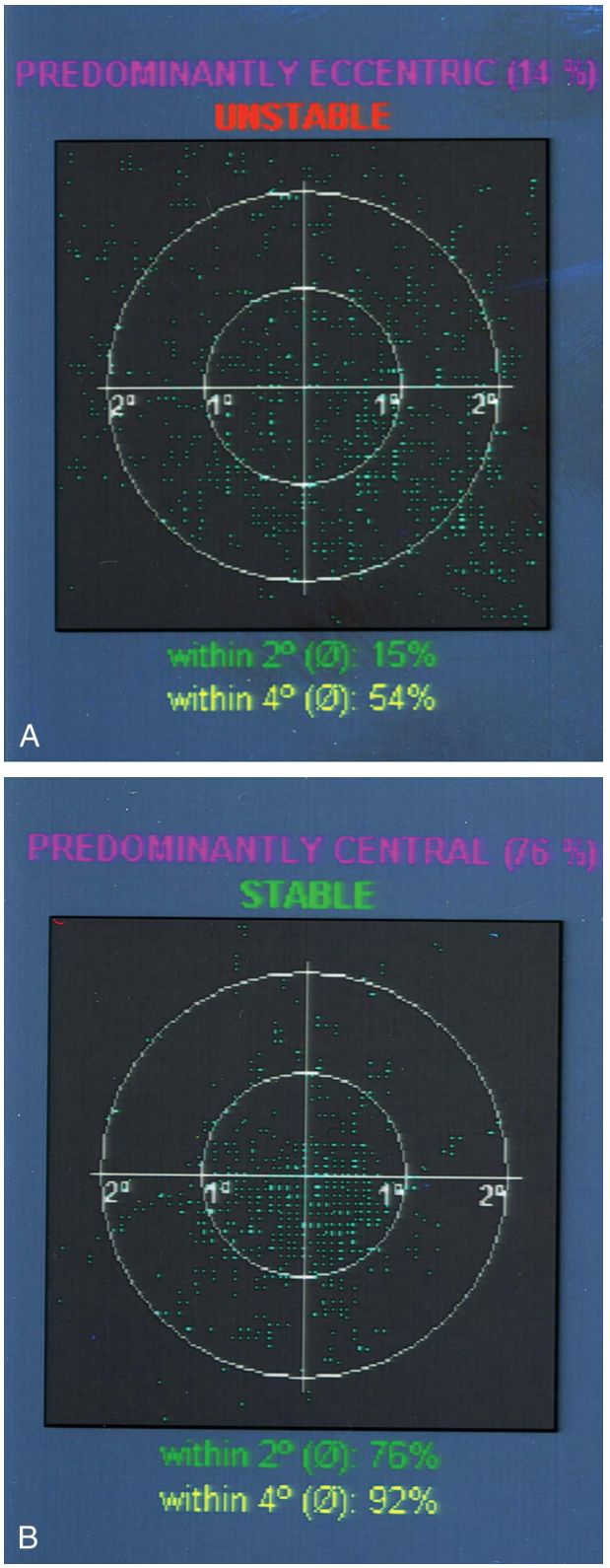

Fig. 4. Fixation location and stability changes after treatment of Case 2. Fixation location was predominantly eccentric, and fixation stability was relatively unstable at baseline (**A**) and becomes central and stable 6 months after treatment (**B**).

triamcinolone acetonide injection were evaluated, whereas patients' subjective appraisal of visual function, which may differ, was not comprehensively discussed.[8–10,12] The results of our study show similarities. On 6-month follow-up, eyes with ME showed a significant increase in logMAR visual acuity. One, 3, and 6 months after intravitreal triamcinolone acetonide injection, the mean visual acuity had increased from 0.8 ± 0.2 to 0.5 ± 0.2, 0.5 ± 0.2, and 0.5 ± 0.2, respectively. There was also a significant reduction in the foveal thickness. At 1, 3, and 6 months, the mean foveal thickness had decreased from 453 ± 108 μm to 254 ± 40.3 μm, 297 ± 90 μm, and 320 ± 82 μm, respectively. At the 3- and 6-month follow-up examinations, 4 of 7 cases, respectively, showed recurrence of ME and were retreated. Also, retinal sensitivity changed after treatment, as shown by microperimetric data. One, 3, and 6 months after triamcinolone acetonide injection, the mean retinal sensitivity had increased from 5.5 ± 3.3 dB to 9.4 ± 3.5 dB, 7.8 ± 3.3 dB, and 7.2 ± 4.2 dB, respectively. Similarly, fixation location had improved significantly 1 month after injection and had also improved, but not significantly, 3 and 6 months after treatment. At baseline, it was predominantly central in 2, poor central in 4, and predominantly eccentric in 6; 6 months after treatment, it was predominantly central in 8, poor central in 3, and predominantly eccentric in 1.

Treated eyes also showed significant improvement in fixation stability at one, 1, 3, and 6 months. At baseline, in eyes with ME, fixation was stable in one, relatively unstable in six, and unstable in five. However, 6 months after intravitreal triamcinolone acetonide injection, fixation was stable in 8, relatively unstable in 3, and unstable in 1.

We also determined not only that visual acuity decreased but also that foveal thickness increased with recurrence. With recurrence, fixation location and stability deteriorated and retinal sensitivity decreased in most of the eyes. Microperimetric parameters may also be good predictors of recurrence. The improvement of retinal sensitivity and fixation stability, which are closely related to the central visual field, positively affects the patient's daily activities. It is well known that important daily tasks such as recognition of faces and symbols, orientation, and reading are strongly dependent on the preservation of the central visual field.[13,14] For example, it has been shown that stability of fixation is directly related to reading ability.[15] High-contrast visual acuity measurement, the standard measurement of vision in both clinical practice and many studies, is a poor predictor of general visual performance. For these reasons, our data are important from the point of view of improvement of retinal

sensitivity and fixation properties after intravitreal triamcinolone acetonide injection in eyes with ME in CRVO. Because of the limitations of our pilot study—relatively short follow-up, a small study sample, and lack of a control group—it was not possible to assess the changes in the central visual field in the long term. Besides showing short-term improvements in retinal sensitivity and fixation properties after intravitreal triamcinolone acetonide injection in eyes with ME in CRVO, our study also shows that further study with longer follow-up and a large series is needed.

Key words: central retinal vein occlusion, macular edema, intravitreal triamcinolone acetonide, microperimetry, fixation, visual acuity.

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