Macular Damage in Glaucoma is Associated With Deficits in Facial Recognition

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• PURPOSE: This report examines the relationship between glaucomatous macular damage and facial recognition. In addition, it assesses the role of contrast sensitivity (CS) as an intermediary step in the causal pathway between macular damage and impairment of facial recognition.

• DESIGN: Prospective cross-sectional study.

• METHODS: This study was conducted in a single tertiary care center. The study population included 144 eyes of 72 participants with a diagnosis of open angle glaucoma in one or both eyes and a visual acuity of 20/40 or better in each eye. The presence or absence of macular damage was determined by comparing corresponding regions of the retinal nerve fiber layer and the retinal ganglion cell layer with spectral-domain optical coherence tomography with the 10-2 visual field (VF). Better and worse eye was determined by 10-2 VF mean deviations (MDs). Interventions were 1) macular function as measured by 10-2 VF and 2) CS as measured by the Freiburg Visual Acuity and Contrast Test (FrACT). The primary outcome measure was the Cambridge Face Memory Test (CFMT) score.

• RESULTS: Regardless of eye, there was a significant correlation between facial recognition and 10-2 VF MD ($P < .0001$ better, worse eye). The 10-2 VF MD remained a significant predictor of facial recognition after adjusting for potential confounders including glaucoma severity, CS, age, and visual acuity ($P = .004$ better eye, $P = .019$ worse eye).

• CONCLUSIONS: Even with good central visual acuity, patients with glaucomatous macular damage exhibit diminished facial recognition, which is partly mediated through diminished CS. (Am J Ophthalmol 2020;217:1–9. © 2020 Elsevier Inc. All rights reserved.)

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OLDER DESCRIPTIONS OF GLAUCOMA DISEASE PROgression are classically described as a gradual loss of peripheral vision, with sparing of central vision until late in disease.[1] Recent data, however, suggest that central (macular) visual function (±8 degrees of fixation) is impacted earlier in glaucoma than was previously believed. Up to 80% of patients with early glaucoma have evidence of macular involvement.[2–5] Furthermore, macular damage has a significant negative impact on vision-related quality of life, as measured by the National Eye Institute Visual Function Questionnaire.[6,7]

Along with visual acuity (VA), contrast sensitivity (CS) has been identified as a clinical measure of macular function and is therefore a common outcome measure in literature regarding macular pathology.[8] While VA is the most widely used measure to assess visual function, CS has been identified as a more useful measure in low luminance settings.[9–12] Patients with glaucoma commonly experience visual difficulty with tasks such as driving at night in low luminance environments.[13,14] Given our improved understanding of macular pathology in glaucoma, increasing attention is being focused on the visual symptoms associated with decreased macular function despite normal VA.[7]

Facial recognition is an activity of daily living that is vital for social interaction and forming and maintaining relationships. It is likely that decreased facial recognition skills negatively impacts patient quality of life by affecting patients' ability to interact socially. Facial recognition has been studied in patients with age-related macular degeneration (AMD) and is now included in vision-based quality of life questionnaires.[15,16] The primary purpose of this study is to investigate the association between facial recognition and macular function in patients with glaucoma, as measured by the mean deviation (MD) of the 10-2 visual field (VF). The secondary purpose is to assess the role of CS as a potential intermediary step in the causal pathway between macular damage and impairment of facial recognition. Understanding this relationship will give us a better understanding of visual impairment in patients with glaucoma.

METHODS

• STUDY DESIGN: Patients with glaucoma were enrolled in a prospective cross-sectional study conducted at the Department of Ophthalmology at Columbia University

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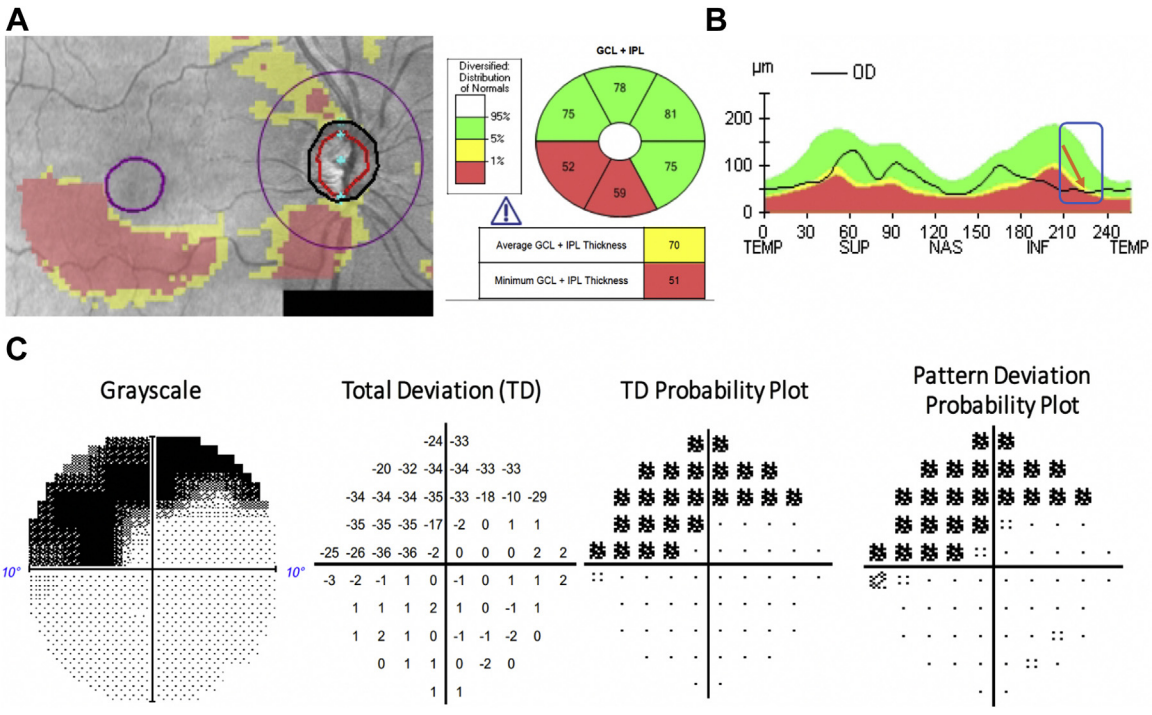

FIGURE 1. Structure–function corroboration of focal macular defect of the inferior disc/superior visual field. (A) Macular OCT demonstrating inferotemporal damage. GCL + IPL = ganglion cell plus inner plexiform layer. (B) Corresponding retinal nerve fiber layer thinning (orange arrow) includes the region corresponding to the inferior macula, as outlined in blue. (C) Corresponding superior paracentral visual field defect.

Irving Medical Center, New York, New York, from March 25, 2019 to April 30, 2019. The Institutional Review Board of Columbia University Medical Center prospectively approved the study and its methods. The study adhered to the regulations of the Health Insurance Portability and Accountability Act. Because the institutional review board determined that the study was of no risk to patients, written consent was not required. Verbal informed consent was obtained from all participants. All study methods adhered to the tenets of the Declaration of Helsinki.

A total of 72 eligible, consecutive patients were recruited for this study. All participants underwent a comprehensive ophthalmologic examination including medical history review, best-corrected VA, slit-lamp biomicroscopy, intraocular pressure measurement, gonioscopy, dilated ophthalmoscopy, and standard automated perimetry (SAP) (24-2 and 10-2 programs using the Swedish Interactive Threshold Algorithm [SITA]; Carl Zeiss Meditec, Inc., Dublin, California, USA). Individuals were included if they had a diagnosis of primary open-angle glaucoma in one or both eyes as determined by a glaucoma specialist. Glaucoma was determined based on characteristic optic nerve damage on stereoscopic examination with localized or diffuse retinal nerve fiber layer (RNFL) thinning on spectral-domain optical coherence tomography (OCT) in addition to open anterior chamber angles on gonioscopy. Glaucomatous eyes both with and without macular damage as determined by 10-2 VF and macular OCT were included. Additional inclusion criteria were a VA ≥20/40 in each eye and a score ≥30 on the short test of mental status (STMS). A score ≥30 on the STMS was required to exclude patients with cognitive impairment. In addition, patients with any ocular or systemic disease that could affect the optic nerve, macula, or VF were excluded. Specifically, patients with epiretinal membranes, drusen, or AMD were eliminated from study entry. Lenses were graded according to the Lens Opacities Classification System III criteria.[17] Patients with ≥2+ nuclear sclerotic changes or any posterior subcapsular or cortical lenticular changes were excluded. Patients with posterior capsule opacification, as assessed by slit-lamp examination and best-corrected VA, were not eligible for study inclusion. Patients undergoing miotic therapy or those whose pupil diameter was <3 mm as measured by the Humphrey VF auto pupil feature were excluded. Patients with severe dry eye or ophthalmic surface disease as determined by the glaucoma specialist or by diminished signal strength (<7) on OCT were also excluded. Eyes were required to be orthophoric for study inclusion. Patients were required to have a reliable VF, defined as <33% fixation losses or false-negative errors and <15% false-positive errors. To increase specificity, the abnormal regions of the RNFL, retinal ganglion cell plus inner plexiform layer (RGC+), spectral-domain OCT, and VF were required to show

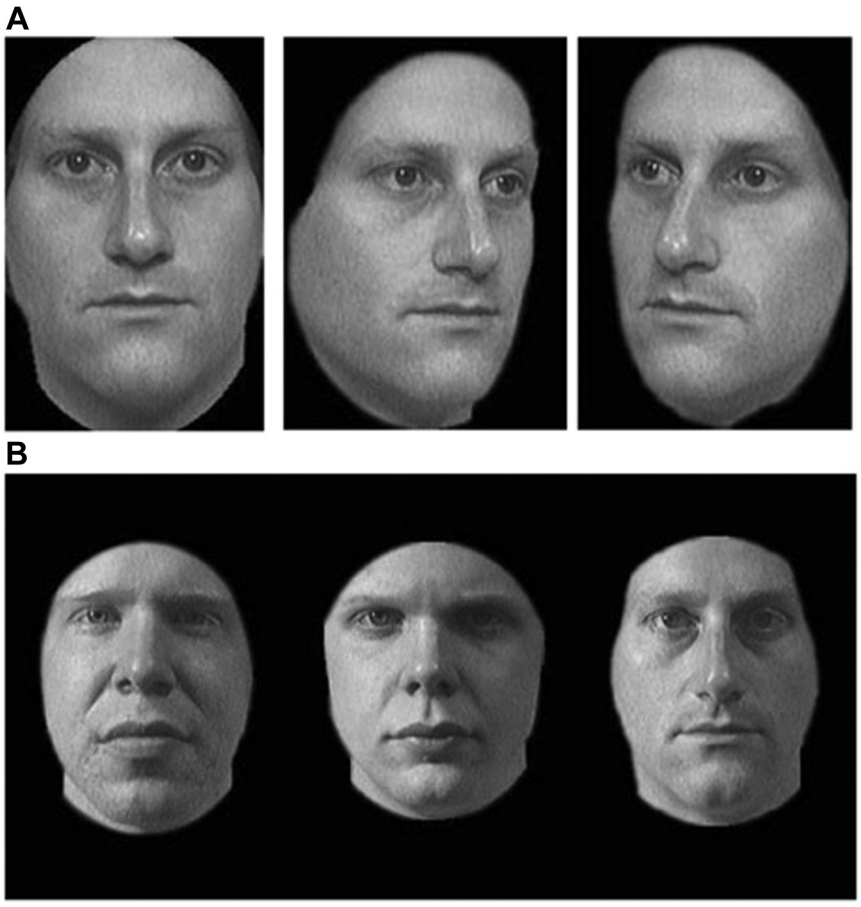

FIGURE 2. (A) Viewing phase in which patients are shown 3 consecutive images of 1 face, each image of a different viewing angle. (B) The forced-choice recognition phase, in which the patient must choose which face he or she has viewed previously.

topographic agreement, as described below. For the purposes of this study, structure–function agreement was required for a classification of "damage." Therefore, eyes without corresponding damage on both spectral-domain OCT and VF were classified as "no damage." Specifically, eyes with RGC+ thinning without corresponding VF loss were classified as no damage.

• **SPECTRAL-DOMAIN OCT SCAN AND VF TESTING:** Spectral-domain OCT images were acquired by a trained ophthalmic photographer. All scans were reviewed and images were excluded if they contained motion or blinking artifacts, incorrect placement of the measurement circle, segmentation error, or poor image quality (signal strength <7) (Cirrus SD-OCT Macular Cube 512 × 128 scan; Carl Zeiss Meditec, Inc., Dublin, California, USA). VF testing was performed with the Humphrey Field Analyzer II (Carl Zeiss Meditec, Inc.) with appropriate refractive correction. The 10-2 and 24-2 SITA-SAP programs were used for monocular VF testing. All VF testing was performed by a trained ophthalmic technician. For this study, "better" and "worse" eyes were defined based upon the 10-2 mean deviation (MD). The RNFL and RGC+ spectral-domain OCTs, 10-2, and 24-2 VF data were examined for the presence of corresponding regions of macular damage on VF and OCT probability plots.[2] Figure 1 shows an example of superior macular damage.

• **VA AND CONTRAST TESTING:** Monocular VA was measured using a rear-illuminated Early Treatment Diabetic Retinopathy Study screen. VA was scored as the total number of letters identified correctly and converted to $\log_{10}$ minimum angle resolvable (logMAR). Monocular CS was measured using the Freiburg Visual Acuity and Contrast Test (FrACT).[18] FrACT was administered on a 2015 15.4-inch Macbook Pro laptop at resolution of 1680 × 1050 at 60 Hz. Gamma was set equal to 1.0 using the Spyder calibration tool. Brightness of the screen and surrounding luminance was kept standard for all patients in accordance with the FrACT protocol, with the surrounding luminance at 25% of screen luminance. Luminance was measured using the mobile Light Meter application. Participants completed the FrACT contrast test at a viewing distance of 40 cm, and had their head mounted in a chin rest to minimize head movements and to standardize testing.

TABLE 1. Baseline Ocular Characteristics of Better Eye and Worse Eye

Baseline Ocular Characteristic	Better Eye	Worse Eye
Mean 10-2 MD ± SD	−4.52 ± 5.74	−10.22 ± 7.90
Mean 24-2 MD ± SD	−6.13 ± 7.09	−10.55 ± 8.69
VA (logMAR) ± SD	0.07 ± 0.09	0.11 ± 0.11
Pseudophakic, n = 71 (%)	47/71 (66)	50/71 (70)
Spherical equivalent, phakic eyes, n = 24, ± SD	−1.6 ± 2.9	−1.8 ± 3.0
Axial length ± SD	24.5 ± 1.7	24.0 ± 1.5
Significant astigmatism, n (%)	12/71 (82%)	15/71 (78%)

logMAR = logarithm of minimal angle of resolution; MD = mean deviation; SD = standard deviation; VA = visual acuity; VF = visual field.

• **FACIAL RECOGNITION TESTING:** Facial recognition was assessed using the Cambridge Face Memory Test (CFMT). The CFMT is a freely available, validated test.[19] It has been used to quantify facial recognition defects in a variety of clinical conditions, including macular degeneration and glaucoma.[20,21] A full description of the methodology is outlined in the original paper by Duchaine and Nakayama[19] in which the test validation is described. Briefly, participants binocularly view 6 target faces at three different viewing angles for three seconds each. Their recognition of these faces is subsequently tested in a series of 51 forced-choice recognition trials in which they must identify the previously seen target face from an additional 2 unfamiliar faces. The outcome measure for the test is the percentage of correctly identified faces. Figure 2 shows example images from the viewing and recognition stages of the task. Participants completed the CFMT on a 15.4-inch Macbook Pro laptop at resolution of 1680 × 1050 at 60 Hz. To standardize testing, participants completed the CFMT at a viewing distance of 40 cm and had their head mounted in a chin rest to minimize head movements. Patients performed the testing binocularly. The image size and viewing angle were calculated to mirror viewing a real face at a distance of roughly 1 meter in the real world.

• **STATISTICAL ANALYSES:** Comparison of means and proportions between eyes with and without macular damage was performed using an independent, 2-tailed, unpaired *t* test and χ^2 statistics, respectively. Univariable and multivariable linear regression analyses using ordinary least squares were conducted to determine the association among macular function as measured by 10-2 VF MD in better and worse eyes and facial recognition as measured by CFMT scores. To minimize possible confounding, glaucoma severity, central VA, CS, and presence of an early cataract were included in the multivariable regressions. In addition, variables that were related to CFMT scores ($P \leq .20$) in the univariable analyses were then entered into the linear multivariable regression model. To test if CS had a mediating effect, we used Baron and Kenny's 4-step test,[22] in which 10-2 VF MD was the predictor (X), CFMT was the outcome (Y), and CS was the possible mediator (M). In this test, the significance of the coefficients is tested as follows: 1) X predicting Y; 2) X predicting M; 3) M predicting Y; and 3) X and M predicting Y. Statistical analyses were performed using Stata software (version 15; StataCorp, College Station, Texas, USA).

RESULTS

SEVENTY-THREE PARTICIPANTS WERE RECRUITED FOR THIS study. One participant failed the STMS and was not included in the analyses, leaving 72 participants. Forty-nine patients (68.1%) were European-derived and 41 (56.9%) were women. The participants had a mean age of 67.0 years (SD ± 11.6 years). Age, race, and gender were not found to be significant univariable predictors of facial recognition ($P = .11$, $P = .33$, and $P = .45$, respectively). Eye and VF details are listed in Table 1.

• **BETTER EYES:** Of the 72 better eyes, 44 (61%) had macular damage and 28 (39%) did not. Eyes with macular damage had had more severe glaucoma, as reflected by the 24-2 VF MD ($P < .0001$). Eyes with macular damage required 1.91 ± 1.0 IOP-lowering medications whereas those without macular damage required 1.4 ± 1.0 ($P = .05$). There was no difference in age ($P = .91$) between patients with and without macular damage in the better eye. There was no difference between eyes with and without macular damage in VA ($P = .21$), percent of eyes with early cataract ($P = .19$), axial length ($P = .61$), spherical equivalent (phakic eyes) ($P = .37$), or significant astigmatism ($P = .64$).

The presence of macular damage in the better eye was associated with a significant decline in facial recognition (47.5 ± 7.9 with macular damage vs 54.7 ± 7.3 without macular damage, $P < .0001$). Results of univariable regression of potential determinants of facial recognition are

TABLE 2. Univariable Analyses of Potential Determinants of Facial Recognition

Potential Determinant	*P* Value
Age	.11
Gender	.45
Race	.33
Better eye 10-2 VF MD	<.0001[a]
Better eye 24-2 VF MD	<.0001[a]
Better eye central visual acuity	.016[a]
Better eye presence of early cataract	.52
Better eye contrast sensitivity	.06
Worse eye 10-2 VF MD	<.0001[a]
Worse eye 24-2 VF MD	.001[a]
Worse eye central visual acuity	.09
Worse eye presence of early cataract	.21
Worse eye contrast sensitivity	<.0001[a]
Dry eye requiring prescription drops (n = 10)	.66
No. of IOP-lowering medications	.018[a]
Better eye spherical equivalent if phakic (n = 24)	.60
Worse eye spherical equivalent if phakic (n = 19)	.79
Better eye axial length	.38
Worse eye axial length	46
Better eye significant astigmatism (>1.5 diopters)	.25
Worse eye significant astigmatism (>1.5 diopters)	.15
Better eye location of defect (superior vs inferior)	.46
Worse eye location of defect (superior vs inferior)	.50
Corresponding intereye visual field defects	.34
Use of prostaglandins	.23
Use of beta blocker	.043[a]
Use of alpha agonist	.22
Use of carbonic anhydrase inhibitor	.39
Use of rho kinase inhibitor	.40

AL = axial length; IOP = intraocular pressure; MD = mean deviation; VA = visual acuity; VF = visual field.
[a]Statistically significant ($P < .05$).

listed in Table 2. Worsening macular damage, as measured by 10-2 VF MD, and glaucoma severity, as measured by 24-2 VF MD, were significantly associated with diminished facial recognition in the univariable analyses ($P < .0001$ and $P < .0001$, respectively), as was VA ($P = .016$). The correlation between 10-2 VF MD and facial recognition in the better eye is demonstrated in Figure 3, A.

Regardless of eye, facial recognition was associated with number of IOP-lowering medications ($P = .018$) and use of a beta blocker ($P = .043$). Neither location of central VF defect (superior or inferior) ($P = .46$ better eye, $P = .50$ worse eye) nor intereye correspondence of defects ($P = .34$) was a significant predictor of facial recognition.

Predictors and significance values of the multivariable analyses are listed in Table 3. Specifically, the association between 10-2 VF MD and diminished facial recognition remained significant in the multivariable analyses ($P = .004$, Table 3), whereas 24-2 VF MD no longer remained significant ($P = .89$). Age ($P < .0001$), CS ($P = .047$), and use of beta blocker eye drops ($P = .005$) were also associated with facial recognition in the multivariable model.

10-2 VF MD was significantly associated with both diminished facial recognition (as noted above, $P < .0001$) and worsening CS ($P < .0001$) in the univariable models. We therefore tested CS as a potential mediator between 10-2 VF and facial recognition by examining the univariable model where CS was a predictor and facial recognition was the outcome. However, the association between CS and facial recognition only approached significance ($P = .06$). This suggests that the relationship between 10-2 and facial recognition is not fully mediated by CS and that 10-2 is an independent predictor of facial recognition.

• **WORSE EYES:** Of the 72 worse eyes, 63 (87.50%) had macular damage and 9 (12.50%) did not. Eyes with macular damage had worse 24-2 VF MD ($P = .0009$) than those without macular damage. Although more drops were prescribed for those with macular damage in the worse eye, the difference was not significant (1.8 ± 1.0 vs 1.1 ± 1.05, $P = .06$). There was no difference in age ($P = .87$) between patients with and without macular damage in the worse eye. There was no difference between eyes with and without macular damage in VA ($P = .39$), percent of eyes with early cataract ($P = .30$), axial length, spherical equivalent in phakic eyes ($P = .56$), or astigmatism ($P = .40$).

As in the better eye, the presence of macular damage in the worse eye resulted in a significant decline in facial recognition (57.1 ± 3.9 without macular damage vs 49.3 ± 8.5 with macular damage, $P = .008$). Potential univariable determinants of facial recognition in the worse eye are listed in Table 2. Worsening macular damage, as measured by 10-2 VF MD, and glaucoma severity, as measured by 24-2 VF MD, were significantly associated with diminished facial recognition in the univariable analyses ($P < .0001$ better and worse eye), as was CS ($P < .0001$).

Predictors and significance values of the multivariable analyses are listed in Table 4. Specifically, the association between 10-2 VF MD and diminished facial recognition remained significant in the multivariable analyses ($P = .0019$, Table 4), whereas 24-2 VF MD no longer remained significant ($P = .87$). Age ($P = .0001$) was the only other variable associated with facial recognition in the multivariable model. The correlation between 10-2 VF MD and facial recognition in the worse eye is demonstrated in Figure 3, B.

10-2 VF MD was significantly associated with both diminished facial recognition (as noted above, $P < .0001$) and worsening CS ($P < .0001$) in the univariable

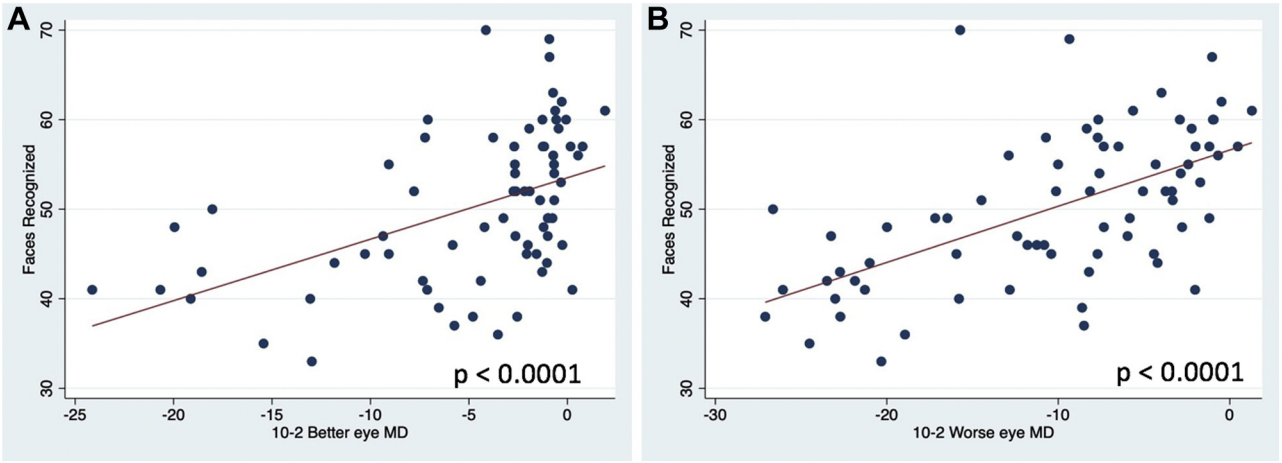

FIGURE 3. (A) Correlation between 10-2 visual field mean deviation and Cambridge Face Memory Test performance in the better eye. (B) Correlation between 10-2 visual field mean deviation and Cambridge Face Memory Test performance in the worse eye.

models. As in the better eye, we tested CS as a potential mediator between 10-2 VF and facial recognition by examining the univariable model where CS was a predictor and facial recognition was the outcome. We found a significant association between CS and facial recognition ($P < .0001$). Since the first 3 steps of the Baron and Kenny model were satisfied, a multiple regression analysis using X (10-2 VF MD) and M (CS) to predict Y (facial recognition score) was performed. In this model, 10-2 VF MD remained significant ($P < .0001$) but CS did not ($P = .11$), suggesting that CS does not fully mediate the relationship between 10-2 VF MD and CS and that 10-2 has a significant independent contribution to facial recognition.

DISCUSSION

OUR ANALYSIS SHOWS THAT PATIENTS WITH GLAUCOMAtous macular damage have impaired ability to recognize faces, despite having good central VA. This impairment appears to be partially mediated by CS, although the 10-2 VF MD remained a significant independent predictor of facial recognition. Instead, we hypothesize that the 10-2 VF encompasses several dimensions of visual function required for facial recognition. It is possible that facial recognition may be impaired by diminished CS or paracentral VF loss, both of which are functions of macular damage that are captured by the 10-2 VF.

Our results are consistent with previous reports demonstrating a relationship between glaucomatous damage and diminished facial recognition. In a sample of patients with advanced VF loss, Glen and associates[20] demonstrated that patients with glaucoma performed worse on the CFMT than control subjects. In addition, in a small case-control study, patients with glaucoma were found to need a significantly shorter viewing distance to recognize gender and expression of target faces.[23] Although multivariable regression analysis of their data identified CS as the most important predictor of performance on the CFMT, this study was limited by absence of 10-2 VF testing and macular OCT images.

Facial recognition is a visual task that is central to social interaction. It fosters relationship forming and trust.[24] As such, loss of facial recognition is likely to have a significant impact on patients' quality of life. In the AMD literature, facial recognition has been recognized as an important quality of life measure and therefore included in visual disability questionnaires.[25] It has been suggested that, after reading impairment, it is the main visual complaint in this patient population.[26] In addition, the loss of CS has been identified as an early and reliable ophthalmologic sign of macular function loss in patients with AMD.[27] In some cases, a decrease in CS may precede a decrease in VA, a phenomenon previously termed as "hidden vision loss."[28] By extension, CS impairment is associated with higher disability in vision-related activities of daily living, including impairment of facial recogntion.[21,29–32]

In the glaucoma literature, CS studies have largely examined the correlation between CS and overall glaucoma severity. Older results suggest that patients with glaucoma have decreased CS.[33] Atkin and associates[34] showed that CS impairment was more significant in eyes with severe glaucomatous damage when compared with less affected eyes. Another early study demonstrated that CS is the most sensitive test for measuring visual defects in patients with glaucoma, and it is an important predictor of difficulty experienced by patients when performing visually dependent activities of daily living.[35] Newer results using macular OCT and 24-2 VF suggest that both structural and functional glaucoma testing show a fair relationship with CS. However, Fatehi and associates[36] were not able to demonstrate a correlation between CS and overall

TABLE 3. Multivariable Analyses of Potential Determinants of Facial Recognition (Better Eye)

Potential Determinant	*P* Value
Better eye 10-2 VF MD	.004[a]
Better eye 24-2 VF MD	.89
Age	<.0001[a]
Better eye central visual acuity	.90
Better eye cataract status	.85
Better eye contrast sensitivity	.047[a]
No. of drops	.56
Use of beta blocker eye drops	.005[a]

MD = mean deviation; VF = visual field.
[a]Statistically significant ($P < .05$).

TABLE 4. Multivariable Analyses of Potential Determinants of Facial Recognition (Worse Eye)

Potential Determinant	*P* Value
Worse eye 10-2 VF MD	.019[a]
Worse eye 24-2 VF MD	.87
Age	.001[a]
Worse eye central visual acuity	.93
Worse eye cataract status	.21
Worse eye contrast sensitivity	.50
No. of drops	.61
Use of beta blocker eye drops	.12
Worse eye significant astigmatism (>1.5 diopters)	.23

MD = mean deviation; VF = visual field.
[a]Statistically significant ($P < .05$).

glaucoma severity using OCT or VF summary measures. One possible explanation for this is that existing summary measures appear to be suboptimal in distinguishing between glaucomatous and healthy eyes.[37] In contradistinction, our study specifically tested the relationship of CS to macular damage as determined by retinotopic matching of RNFL, RGC+, and VF loss.

Facial recognition is a low-contrast task that relies on good central visual function. Aging, nuclear sclerosis, refractive error, pupillary size, dry eyes, and macular disease may all impact CS and potentially facial recognition.[8,38–41] For this reason, we had strict inclusion criteria and ran multivariable regressions to adjust for potential confounding. Because our study criteria prevented patients with diminished VA, visually significant cataract, posterior capsule opacification, severe dry eye, macular disease, and miotic pupils from study eligibility, we did not find any of these potential confounders to be associated with facial recognition in our sample. However, in a larger population-based sample, it is likely that some or all of these factors would contribute to diminished facial recognition. In addition to 10-2 VF MD, we found that age, CS, and the use of beta blocker eye drops were significant predictors of facial recognition in our multivariable model. The relationship between aging and CS is well established in the literature. Aging appears to diminish CS, presumably because of the disproportionate loss of rod receptors over time.[42]

Our findings on topical beta blockers, however, revisits an ongoing debate about their potential visual consequences. In a small cohort of newly diagnosed patients with glaucoma, beta blockers were shown to decrease CS relative to baseline when tested 12 weeks after initiation.[43] Although the exact mechanism is not well understood, one hypothesis relates contrast changes to IOP-independent perfusion changes.[43] In the Low Pressure Glaucoma Treatment Study, patients taking timolol were reported to have faster VF progression than those on brominidine.[44] Although the generalizability of the study is limited due to the high brominidine dropout rate, it is possible that timolol does impact central visual function in ways that are not well understood to date.

Despite the correlation we found between loss of CS and macular damage, CS does not appear to fully mediate the relationship between 10-2 VF and facial recognition. These findings may be related to study design. For example, luminance conditions are important in the assessment of CS. In this study we followed the guidelines for ambient lighting as recommended by the FrACT protocol,[18] measured by the mobile Light Meter application. Future studies should assess the relationship between CS and facial recognition in low luminance conditions. In addition, although we required structure–function correlation as an inclusion criterion, we did not differentiate between patients with focal and diffuse macular damage. It is possible that those with subtle diffuse defects have relatively greater impairment in CS, whereas those with focal defects have more disability because of the presence of local paracentral VF defects.

Additional potential limitations of our study are related to the nature of the facial recognition and the categorization of eye-level testing into patient level data. To assess facial recognition, we used the CFMT. We chose to administer the CFMT as it is well-cited in multiple disciplines and has previous precedent in ophthalmic literature.[20,21] Although previous work has shown the test to be accurate independent of baseline intelligence quotient,[45,46] we believe that it is possible that cognition and memory may be confounders, because patients must recall facial images seen a few minutes earlier. To address this concern, we administered the STMS to all patients recruited. The STMS was chosen because of its focus on learning and recall.[47] Patients with a score <30 on the STMS were not included in the study. This cutoff excludes patients with cognitive impairment and neurodegenerative disease.[47]

Furthermore, translating an eye-based test into a person's visual ability poses inherent difficulty. It is unclear how better and worse eyes interact in facial recognition. While studies regarding most visual outcomes, such as binocular VA, demonstrate that performance often relies on the better eye, much of the contrast literature uses worse eye CS as the predictor in analyses.[48] To account for this, we ran regression analysis separately using better eye and worse eye as categorized by 10-2 VF MD. Lastly, these results are not generalizable to all patients with glaucoma, but are limited to patients with good central VA and those without visually significant cataracts.

In summary, this work increases our understanding of the visual disability experienced by patients with glaucoma. In particular, glaucomatous patients with macular damage in either eye experience impaired facial recognition. This impairment is driven in part, but not entirely, by diminished CS.

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