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Baseline 24-2 Central Visual Field Damage is Predictive of Global Progressive Field Loss

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Abstract

Purpose—Central visual field (VF) damage in glaucoma patients can significantly hinder daily activities. The present study investigates whether the presence of localized baseline damage to the central ten degrees of the VF is predictive of faster global mean deviation (MD) progression.

Design—Prospective, cohort study.

Methods—Eyes from the multicenter African Descent and Glaucoma Evaluation Study (ADAGES) with established glaucoma and VF loss and a minimum of five 24-2 VFs were eligible. Baseline central 24-2 damage was defined as any of the 12 central-most points with total deviation (TD) values at P<0.5% on two consecutive examinations. Progression was determined using trend-based and event-based criteria: (i) rates of MD change significantly faster than zero and (ii) >−5 dB MD loss over the entire follow-up.

Results—827 eyes of 584 patients were studied. Mean rate of MD change of the entire sample was −0.15 dB/year (95% CI: −0.19 to −0.12, P<0.001). Eyes with baseline central damage progressed faster than those without (difference: $\beta_{central}$ = −0.07 dB/year, 95% CI=−0.11 to −0.01, P=0.011) and were more likely to experience MD loss greater than 5 dB [hazard ratio=3.0 (95% CI: 2.1 to 4.1, P<0.001)]. These differences remained significant after adjusting for confounders.

Conclusion—The presence of central VF damage at baseline is significantly associated with more rapid global progression. Detection of central VF damage aids in stratification of high-risk patients who may need intensive surveillance and aggressive treatment.

Keywords

central visual field damage; risk factors; glaucoma; visual fields; progression; nasal step; mean deviation; mean deviation sensitivity; visual field index

INTRODUCTION

The prevalence of glaucoma, the leading worldwide cause of irreversible blindness, will increase to 111.8 million individuals by 2040.[1] Developing strategies to effectively screen and identify individuals at greater risk for more rapid glaucoma progression will be imperative to curtailing the effects of the disease.

Patterns of visual field (VF) loss and their effect on the progression of disease are an area of interest in glaucoma research.[2–4] The central VF is regarded as an important area for glaucoma monitoring, as loss in this region has been associated with a greater degree of morbidity[5, 6] and longitudinal decline in quality of life than more peripheral loss.[7–9] Moreover, there have been reports that central VF loss can cause significant hindrances on daily activities such as reading[10] and driving,[11] so that monitoring central VF loss may be particularly important for patient well-being.

While it has been established that the central VF is an important determinant of vision-related quality of life, the impact of baseline damage in this area on future VF progression has not been fully investigated. The present study aims to investigate the impact of baseline damage to the central field on global VF progression. We hypothesize that baseline damage within the central 10 degrees of the 24-2 VF strategy is predictive of faster mean deviation (MD) rates of progression.

METHODS

Data for this analysis was collected as part of the multi-site, prospective, cohort study, African Descent and Glaucoma Evaluation Study (ADAGES). This collaboration (clinicaltrials.gov Identifier: NCT00221923) includes the Hamilton Glaucoma Center at the Department of Ophthalmology, University of California-San Diego (UCSD) (data coordinating center), Edward S. Harkness Eye Institute at Columbia University Medical Center (site formerly located at New York Eye and Ear Infirmary), and the Department of Ophthalmology, University of Alabama-Birmingham (UAB). The institutional review boards at all sites approved the study methodology, which adheres to the tenets of the Declaration of Helsinki and to the Health Insurance Portability and Accountability Act. All participants gave written informed consent. ADAGES enrollment began in January 2003 and ended in July 2006, and follow-up continued into 2016. Participants of both African and European ancestry were included in this longitudinal cohort study of individuals with healthy eyes, glaucoma suspects, and established glaucoma.

Participants

Participants were asked to identify their race by self-report using the National Eye Institute inclusion/enrollment system describing ethnicity and race (http://orwh.od.nih.gov/pubs/outreach.pdf [pages 120–121]). Information regarding a family history of glaucoma (biological mother, father, sibling, aunt, uncle, and grandparent) was also obtained. All participants were recruited from the glaucoma clinics and ophthalmic practices at each of the three recruiting sites, by advertisement and community presentations, and by referral from other ophthalmologists and optometrists in the community.

The ocular testing completed for ADAGES has been described elsewhere.[12] In brief, participants underwent a comprehensive ophthalmic examination, including annual review of medical history, best-corrected visual acuity, slit-lamp biomicroscopy, intraocular pressure (IOP), dilated funduscopic examination, pachymetry, simultaneous stereoscopic optic disc photography, and standard automated perimetry visual field (VF) testing with 24-2 Swedish interactive threshold algorithm (Carl Zeiss Meditec, Inc., Dublin, California, USA). VFs were repeated every 6 months and optic disc photographs were performed every 12 months.

Inclusion criteria at baseline

All participants had open angles, a best-corrected visual acuity 20/40, and a refractive error <5.0 diopters sphere and <3.0 diopters cylinder. At least one high-quality stereophotograph and two reliable (<33% false positives, false negatives, and fixation losses) standard automated perimetry Humphrey 24-2 field test results at baseline were required. Both eyes were included, except in cases where only one eye met the study criteria. Diabetic participants without evidence of retinopathy were included. Each participant underwent VF testing using the 24-2 program on the Humphrey Field Analyzer II, with the Swedish Interactive Thresholding Algorithm (SITA), 33 version 4.1 (Carl Zeiss Meditec, Inc., Dublin, California, USA). Only patients with glaucomatous optic neuropathy and abnormal 24-2 VFs (as defined below) were included in the present report.

Exclusion criteria

Participants were excluded if they had a history of intraocular surgery (except for uncomplicated cataract surgery or glaucoma surgery), secondary causes of glaucoma (e.g., iridocyclitis, trauma), other systemic or ocular diseases known to affect the VF (e.g., pituitary lesions, demyelinating diseases, etc.), significant cognitive impairment, history of stroke, Alzheimer disease, or dementia, problems other than glaucoma affecting color vision, an inability to perform VF examinations reliably, or a life-threatening disease that precluded retention in the study.

Glaucomatous Optic Neuropathy

Details of the ADAGES methodology have been described elsewhere.[12] Glaucomatous optic neuropathy was defined as excavation, neuroretinal rim thinning or notching, localized or diffuse retinal nerve fiber layer defect, or vertical cup-disc ratio asymmetry > 0.2 between eyes (not explained by differences in disc size) based on masked grading of

stereophotographs by two graders at the Imaging Data Evaluation and Analysis (IDEA) Reading Center. Only photographs of adequate quality were used for evaluation.

An abnormal 24-2 VF was defined as pattern standard deviation (PSD) had a P<5% or the Glaucoma Hemifield Test (GHT) result was "outside normal limits." Abnormality had to be confirmed with an additional VF test.[12]

Subjects with glaucomatous optic neuropathy, abnormal 24-2 VFs, and at least five 24-2 VFs were included in this study.

Visual Field Progression

Progression was defined using: (i) trend analysis: significant rate of MD change (significantly different from zero) using mixed effects linear models and (ii) event analysis: >5 dB MD loss over the entire follow-up period. This cut-off value was chosen as it has been reported that MD losses >5 dB are associated with significant decline in vision-related quality of life.[13]

Since the 24-2 MD calculation allots greater weight to the central points of the VF, which could potentially overestimate the effect of baseline central damage, we also analyzed models with mean sensitivity deviation (MSD) as the outcome variable. The MSD is calculated after simple averaging of the deviation sensitivities from the TD plots. Of note, given the that these values are in log scale (dB), they were first converted to linear scale, averaged, and then converted back to dB. Additionally, we modeled Visual Field Index (VFI) as the outcome variable. Since VFI gives the central field even more weight than MD, we hypothesized that any potential association between central field loss and global progression would be most strongly demonstrated with this measure.

Central VF damage and Nasal VF Damage

Central 24-2 damage was defined as any of the 12 central-most points with total deviation (TD) values categorized as abnormal at P<0.5% on two consecutive baseline examinations.

One potential limitation of the proposed analysis is that patients with baseline central VF damage are likely to have a worse overall VF status due to the presence of a greater number of abnormal test locations (P<0.5%). Therefore, we completed an additional analysis in which we evaluated the predictive value of baseline nasal VF damage using GHT sector 3, which consists of 10 points on VF progression. We chose this sector as nasal VF damage is often one of the earliest sites of glaucomatous functional damage.[14–16] We tested whether the presence of any significantly abnormal test location (as defined by TD probabilities at P<0.5%) in the nasal sector and central visual field was associated with VF progression. Figure 1 demonstrates the central and nasal points studied in this report.

Statistical analysis

Measures of center and dispersion are described as means and standard deviation (SD), respectively. Categorical variables were compared using Fisher's exact-test and continuous variables were compared using two-tailed, independent samples t-test. Linear mixed-effects models were used to evaluate rates of change in VF MD over time and their relationship

with the presence of baseline central VF damage (as defined above). In linear mixed models, the average evolution of the outcome variable (MD) is described using a linear function of time, and random intercepts and random slopes introduce subject- and eye-specific deviations from this average evolution. The model accounts for the fact that two eyes from one individual can have different rates of MD loss over time, while also correcting for correlations between two eyes from one individual. Cox proportional hazards models were used to test the relationship between predictors and the event-based outcome.

The following potential confounders were included as covariates in the model: baseline VF MD, age at baseline, central corneal thickness, and mean follow-up IOP. The two-way interaction between "Time" and "Presence of baseline central VF defect" was included in the model in order to evaluate whether there was a significant difference in MD slopes (dB/year) between eyes with and without baseline central VF damage. Additionally, we included the two-way interaction terms for all covariates, particularly the baseline MD, to minimize the effect of a worse baseline severity in patients with central defects on the rates of MD change.

Statistical analyses were performed using commercially available software (STATA, version 14; StataCorp LP, College Station, TX). The α level (type I error) was set at 0.05.

RESULTS

Eight hundred and twenty-seven eyes of 584 patients met our inclusion criteria and are included in this report. Mean (standard deviation, SD) age at baseline was 63 (12) years for those with baseline central damage and 60 (12) years for those without (P<0.001). Eyes with baseline central loss had a mean of 13.7 (5) VFs spanning 8.8 (3) years, while eyes without baseline central loss had a mean of 13.7 (6) VFs spanning 8.8 (4) years (P>0.05). As expected, baseline MD was worse in those eyes with baseline central damage compared to those without (−9.0 (6) dB vs. −2.2 (2) dB, P<0.001). Additionally, baseline MSD was worse in those with baseline central damage compared to those without (−9.0 (6) dB vs. −2.5 (2) dB, P<0.001). Patient characteristics are summarized in Table 1.

The mean rate of MD, MSD, and VFI change of the entire sample was −0.15 dB/yr (95% CI: −0.19 to −0.12, P<0.001), −0.15 dB/yr (95% CI: −0.18 to −0.11, P<0.001), and −0.55%/yr (95% CI: −0.65 to −0.44, P<0.001), respectively. Eyes with baseline central damage had greater average MD rate of progression than those without (difference, $\beta_{central}$: −0.07 dB/year, 95% CI=−0.11 to −0.01, P=0.011). This significant difference in MD rate of progression was also found in those with and without baseline nasal damage (β_{nasal}= −0.05 dB/year, 95% CI= −0.11 to −0.00, P=0.049).

A similar a number of eyes had central or nasal defects at baseline (408 vs. 426, respectively). In addition, there was considerable overlap in the proportion of eyes with simultaneous central and nasal defects (320 of 827 [38%], Table 2).

In the multivariable analysis using mixed effects modeling, eyes with baseline central defects progressed faster than those without after adjusting for covariates (difference, $\beta_{central}$ = −0.08 dB/yr, 95% CI: −0.15 to −0.02, P=0.008). Those with nasal defects also progressed

faster (β_{nasal} = −0.07 dB/yr, 95% CI: −0.14 to −0.01, P= 0.021). However, when we added central defects and nasal defects together in the same multivariable model, only those with central defects remained significant (P=0.030 vs. 0.090) (Table 3). Similar results were seen when looking at MSD (P= 0.022 vs. 0.124) and VFI rates of progression (P= 0.007 vs. 0.040).

In the multivariable model with event analysis, the hazard ratio for MD loss >5 dB in eyes with baseline central damage was 3.0 (95% CI: 2.1 to 4.1, P<0.001). (Table 4 and Figure 2)

DISCUSSION

This prospective cohort study demonstrates that baseline central VF loss as measured with the 24-2 VF testing strategy was predictive of more rapid and significant progression of global field damage in eyes with manifest glaucoma. Our findings may have significant clinical implications for closer surveillance as well as more aggressive treatment of glaucoma patients with central VF loss. To our knowledge, the present analysis is the first to prospectively study the effects of central VF damage on global field progression over time in patients with manifest glaucoma.

The main goal of glaucoma therapy is to preserve visual function and quality of life. We found that the presence of baseline central 24-2 damage increases the likelihood of experiencing MD loss > 5 dB (an amount of change known to significantly affect quality-of-life[13]) by three-fold compared to those without baseline central damage. The strong association between baseline central visual damage and progression of global VF damage demonstrates that these eyes are at greater risk of more rapid global progression. Furthermore, Abe et al recently demonstrated greater declines in NEI VFQ-25 scores in those patients who had worse central VF sensitivity at baseline.[9]

We employed several strategies to overcome the potential bias of trying to analyze the effect of central VF damage on VF progression as central and global VF damage are strongly correlated. First, as a stronger weighting of central field points on global indices could bias our results, we also assessed rates of MSD change. We also found statistically significant effects in both MD and MSD. These differences between the various global indices should be taken into consideration in future studies assessing VF outcomes. Second, we also tried to minimize the effect of differences in baseline MD as a confounder on MD rates of change by performing a multivariable analysis that adjusts for baseline MD and differences in slopes between eyes with more versus less severe MD (represented by the variable "Baseline MD" and its interaction "Baseline MD*Time", respectively). Finally, we investigated whether eyes with nasal field defects (meeting the same criteria of abnormality as those with central defects) progressed faster than those without nasal defects. Our rationale for this analysis was that the location of the defect – and not only the overall severity of the VF defect predicts future progression. This type of analysis is also important as many eyes have both nasal and central defects (Table 2). Even though eyes with nasal defects progressed faster than those without it, when both groups (nasal and central) were entered together in the same model, only central defects remained significant suggesting not only the importance of VF severity but, more importantly, the location of the defect. The results of all the analyses

described above provide consistent evidence that baseline central damage predicts global VF MD progression independent of worse overall VF damage found in patients with central loss.

Progression of global VF loss associated with baseline central damage has also been evaluated in a retrospective study of normal tension glaucoma by Membrey and colleauges.[4] In this study, central field loss was defined as presence of a cluster of at least three points in one hemifield depressed by −5 dB, with at least one point depressed by >10 dB in the four paracentral locations. Progression was defined as −1 dB per year or more of sensitivity loss at the same test location. The authors found that eyes with baseline threats to fixation tended to progress more rapidly globally although this was not statistically significant (P=0.18).[4] Additionally, in a retrospective study with normal tension glaucoma patients, Cho et al compared the rates of sensitivity change between eyes with initial paracentral defects and initial peripheral defects. No significant differences were found in testing sectoral and global rates of change between the two groups.[19] Furthermore, Nassiri et al used pointwise event analysis to study glaucomatous eyes with a mean baseline MD of −4.2 ± 4.5 dB and found that rates of decay were faster in the central 10° surrounding fixation compared to 20° and 30° regions.[20] Differences in study design, samples, and definitions of central VF and progression may explain at least in part inconsistencies between our findings and those cited above.

Other reports have explored patterns of damage in the VF and their association with more rapid progression. Shon et al proposed that superior arcuate as well as superior and inferior nasal areas may be best able to predict future VF deterioration as these regions demonstrated the most consistent rate of reduction in retinal sensitivity. In this study, reduction of central sensitivity was found to be variable between early and late follow-up period.[21] While this report offers useful insight into VF progression, we cannot definitively conclude that a consistent rate of progression in any given area makes it a solid predictor for overall progression. Additionally, this study used 24-2 VFs to study the macular region; inconsistent results in this region may be due to variable sampling of the damage in this region.

Our study found that central VF defects are linked to a faster decline in global progression of the field. It is known that decline in visual function has a significant effect on quality of life.[13, 22–24] Abe et al reported that reduction of sensitivity in the central inferior region of the 24-2 VF was found to have the highest association with decline in vision-related quality of life.[9] This association between central damage, decline in quality of life, and faster global progression can have significant ramifications for patients and requires physicians to pay special attention to changes in the central VF. Furthermore, the vulnerability of the central field to elevated pressures[25] calls for a more aggressive treatment regimen in the setting of central loss.

Our analysis used only the 24-2 strategy to study the central VF; however previous studies have found that central visual damage can be missed with 24-2 fields.[26] Despite this potential disadvantage of 24-2 tests to detect central VF loss, clinicians more commonly use 24-2 over 10-2 testing to monitor progression in glaucoma patients. Therefore, our findings using 24-2 fields have more direct clinical applicability. Nonetheless, our results need to be

further validated with a long term study using both 24-2 and 10-2 testing strategies. Additionally, proposed reasons for macular loss in glaucoma include nasalization of the central retinal vessel trunk,[27] axonal crowding at the inferior and superior poles of the nerve, [28] and the enlargement of lamina cribrosa pores and associated loss of structural support in these regions.[29] Further investigations to corroborate these anatomical changes with functional testing would be useful. Of note, our results suggest an association between central functional loss and rates of global visual field progression. A causal relationship cannot be inferred from our results and future studies ought to address this matter

In conclusion, the presence of central VF damage is significantly associated with increased velocity of global VF progression. Detection of central VF damage aids in stratification of high-risk patients who may need more intensive surveillance and aggressive treatment.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Biography

Aakriti Garg MD is currently a third-year ophthalmology resident at Columbia University Medical Center in New York, New York. Dr. Garg obtained her medical degree from Columbia University College of Physicians and Surgeons. Her primary areas of research interest include identifying risk factors for visual field progression, imaging in glaucoma, as well as glaucoma's impact on vision-related quality of life.

FIGURE 1.
The central visual field was defined as the 12 points outlined in blue on the 24-2 field, while the nasal visual field was defined as the 10 points outlined in orange on the 24-2 field.
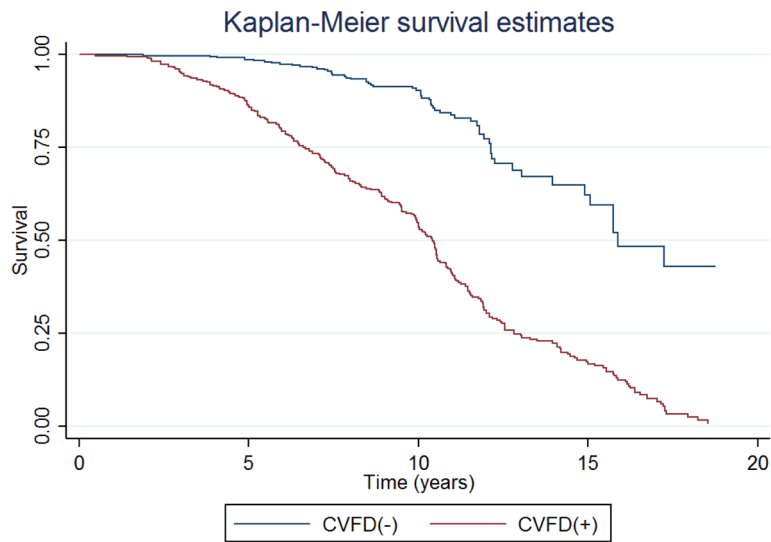
FIGURE 2.
Cox proportional hazards survival analysis comparing the probability of progression (> 5 dB change from baseline) between eyes with and without baseline central visual field damage (CVFD).

TABLE 1

Characteristics of the study sample by central visual field damage. Data shown as mean (standard deviation, SD) unless otherwise indicated. N = number of eyes.

	Central Visual Field Defect (+) (N=408)	Central Visual Field Defect (−) (N=419)	P-value*
Age (years)	62.9 (12)	60 (12)	<0.001
Baseline 24-2 Mean Deviation (dB)	−9.0 (6)	−2.2 (2)	<0.001
Baseline 24-2 Mean Sensitivity Deviation (dB)	−9.0 (6)	−2.5 (2)	<0.001
Baseline 24-2 Visual Field Index (%)	75.8 (20)	95.5 (3)	<0.001
Number of Visual Field Tests	13.7 (6)	13.7 (6)	0.978
Follow-up Time (years)	8.8 (3)	8.8 (4)	0.979
Central Corneal Thickness (microns)	532.8 (39)	534.4 (39)	0.554
Mean Intraocular Pressure (mmHg)	14.5 (3)	15.8 (3)	<0.001

*Independent t-test with unequal variances

TABLE 2

Proportion of sample (number of eyes) with central versus nasal defects at baseline.

		Central Defect		
		No	Yes	Total
Nasal Defect	**No**	313	88	401
	Yes	106	320	426
	Total	419	408	827

TABLE 3

Multivariable analysis of the rate of mean deviation (MD, in dB) change over time (in years).

Variable	β-coefficient	95% Confidence Interval	P-value
Time 24-2 (years)	0.68	0.32 to 1.04	<0.001
Baseline Central Visual Field Defect	−0.33	−0.57 to −0.08	0.007
Baseline Central Visual Field *Time	−0.07	−0.140 −0.006	0.030
Baseline Nasal Defect	−0.06	−0.30 to 0.18	0.610
Baseline Nasal Defect *Time	−0.06	−0.129 to 0.009	0.090
Baseline Mean Deviation (per dB)	0.94	0.919 to 0.963	<0.001
Baseline Mean Deviation*Time	−0.001	−0.007 to 0.004	0.675
Intraocular Pressure (per mmHg)	0.02	−0.002 to 0.056	0.068
Intraocular Pressure*Time	−0.018	−0.027 to −0.009	<0.001
Central Corneal Thickness (per 40 microns)	0.039	−0.056 to 0.136	0.430
Central Corneal Thickness*Time	0.004	−0.015 to 0.026	0.600
Age (per decade)	0.038	−0.046 to 0.12	0.377
Age*Time	−0.095	−0.124 to −0.066	<0.001

TABLE 4

Results of the multivariable survival analysis of a > 5 dB MD change from baseline.

	Hazard Ratio	95% Confidence Interval	P-value
Baseline Central Field Defect	3.00	2.18 to 4.14	<0.001
Mean Intraocular Pressure (per mmHg)	1.03	1.005 to 1.06	0.021
Central Corneal Thickness (per 40 μm)	1.04	0.92 to 1.17	0.460
Age (per decade)	1.35	1.22 to 1.50	<0.001
Baseline Mean Deviation (per dB)	0.93	0.92 to 0.95	<0.001