Measurement of Retinal Nerve Fiber Layer in Primary Acute Angle Closure Glaucoma by Optical Coherence Tomography

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Abstract-

Objective: To assess the accuracy of quantitative retinal nerve fiber layer (RNFL) thickness assessment using optical coherence tomography (OCT) in the detection of RNFL changes in primary acute angle closure glaucoma (PAACG).

Methods: A cross-sectional study enrolled 60 patients after remission with unilateral PAACG attacks and 60 normal controls with single eyes. Treatment had resolved PAACG in all cases.

RNFLs of both eyes were assessed by Stratus OCT within two weeks and four months after PAACG. The parameters of OCT were compared within two weeks and four months after remission between normal controls, unaffected fellow eyes, and attacked eyes. Twenty PAACG patients underwent five measurements after intraocular pressure was controlled, within 2 weeks and at 1, 2, 3, and 4 months afterward. A comparison of the mean RNFL thickness among affected and fellow eyes was also conducted after remission at various times.

Results: A total of 60 eyes of 60 participants with unilateral PAACG and 60 eyes of 60 normal individuals participated in this study. The PAACG and normal groups both had 9 males and 51 females. Because of treatment, the mean intraocular pressure was 14.3±3.4 mm Hg. PSD and MD values for twenty PAACG patients (33.3%) were excluded from analyzes because of unreliable perimetry results. Statistically significant changes were to start in the average, temporal, superior, nasal, and inferior RNFL thicknesses of 60 acutely attacked eyes (within two weeks of remission), as equated to fellow eyes or normal eyes (P-value <0.001)

Conclusions: After PAACG episodes, RNFL thickness increased in the eyes immediately and decreased (up to 3 months) over time, according to OCT. Because of this detection, more understanding of pathologic retinal changes associated with PAACG can be gained, besides our ability to assess and manage these patients.

Keywords: Optical coherence tomography, acute angle closure glaucoma, retinal nerve fiber.

I. INTRODUCTION

In glaucoma, chronic ocular hypertension damages the optic nerve and other ocular tissues irreversibly. An acute, symptomatic condition that damages the optic nerve irreparably is primary acute angle closure glaucoma (PAACG). After recovery from PAACG attacks,[1] the optic disc may appear pale and uncapped without being hyperemic or swollen. Neuronal damage after PAACG has not been determined, particularly the mechanism and rate of impairment. As a general rule, perimetry documents retinal nerve fiber damage. Because PAACG participants belong to the aging group, and they often have visible cataracts, visual field tests are often difficult and unreliable during and after remission.[2] Observation of the post-PAACG results shows differing degrees of severity and type of visual field defects.[3] Optical coherence tomography (OCT) is a noninvasive, noncontact method of measuring nerve fiber layer thickness by taking cross-sectional images at a resolution of approximately 10mm of the retina and nerve fiber layer (NFL).[4] Both normal and glaucomatous eyes were measured with good reproducibility by several observers.[5–9] The dynamic variations of the retinal nerve fiber layer (RNFL) thickness of PAACG patients that have been followed longitudinally (at diverse time points after the attack) are interesting to quantify objectively. We used optical coherence tomography (OCT) to examine dynamic structural changes in RNFL thickness after PAACG 4 months later. A comparison of the RNFL thickness changes in PAACG eyes with the RNFL thickness changes in the eyes of fellow unaffected individuals and healthy individuals was carried out.

II. MATERIAL AND METHODS

The cross-sectional study involved 60 patients with unilateral PAACG attacks and 60 normal controls with single eyes after remission. Treatment had resolved PAACG in all cases. The study was conducted for six months at Liaquat University of Medical & Health Sciences (LUMHS) Jamshoro. There were three study visits in the current study: a visit two weeks after the PAACG episode, a visit four months later, and a visit five months after the PAACG episode to follow longitudinally RNFL measurements at five different time points. RNFL measurements were taken on the same patients before acute attack, after acute attack, and five times on the same patients. RNFL measurements were performed on 21 patients with unilateral PAACG at 5 diverse time points following the attack (within 2 weeks following the attack, 1, 2, 3, and 4 months afterwards). RNFL thickness measurements were conducted on 60 PAACG subjects within two weeks of the study and after four months. It took two weeks to complete the automated visual fields after the OCT exam.

The included patients in this study had best-corrected visual acuity of Z20/40 and a refraction of <75.0D (sphere) and 2.5D (cylinder), with an IOP of <21mm Hg and there is no history of elevated intraocular pressure, past episodes of acute glaucoma, or clinical findings indicative of recent acute attacks, and no experience with intraocular surgery or laser therapy. The following patients were excluded: bilateral attacks; secondary angle closures, PAACGs of the fellow eye in the past or during premeasurement follow-up, and refractions of Z5.0D (sphere) and/or 3D (cylinder).

Eyes with normal visual fields were excluded because of blurry OCT images and opacity of visual media. A normal subject would be a volunteer or a friend of a patient. Every normal subject had one eye randomly selected for the experiment. We defined normal glaucoma hemi field results as being within a 95% confidence interval, and mean deviation and PSD were defined as being within that interval. The results of a visual field test analysis were excluded if fixation loss was >20%, false positives and false negatives were >30%.

Statistical Analysis: In this study, SPSS version 23.0 for Windows was used to perform statistical analyses. To compare OCT parameters, one-way analysis of modification (ANOVA) with Tukey test was applied, and to compare MD, PSD, and normal eye visual field indices, one-way ANOVA was carried out within 2 weeks and at month four. The changes in RNFL thickness after IOP controlled measurements were analyzed using repeated measures ANOVA in both eyes that were affected by acute attack and those that were not. At the level of significance of 0.05, all analyses were conducted.

III. RESULTS

A total of 60 eyes of 60 participants with unilateral PAACG and 60 eyes of 60 normal individuals participated in this study. The PAACG and normal groups both had 9 males and 51 females.

Table 1 defines the study population's characteristics. There was an average duration of 53.6±36.7 hours per attack (range 12 to 120 hours). Because of treatment, the mean intraocular pressure was 14.3±3.4 mm Hg. PSD and MD values for twenty PAACG patients (33.3%) were excluded from analyzes because of unreliable perimetry results. The mean age of PAACG patients, the mean age of normal individuals, and the mean IOP did not differ significantly among these three groups. Although PSD and MD were significantly different between affected eyes and fellow eyes, there was no significant change between the normal eyes and the affected eyes. This table 2 shows the results of a study that measured the thickness of different quadrants of the retina (superior, nasal, inferior, and temporal) in affected eyes, fellow eyes, and normal eyes at two time points: 2 weeks and 4 months.

At 2 weeks, the affected eyes had significantly thicker retinas compared to normal eyes in all quadrants. The fellow eyes, which are the eyes that are not affected, also had thicker retinas compared to normal eyes, but to a lesser extent.

At 4 months, the thickness of the affected eyes had decreased, but it was still significantly different from normal eyes in all quadrants. The fellow eyes were similar in thickness to normal eyes. The p-values are all <0.001, indicating that the differences between affected and normal eyes are statistically significant. When compared with normal and fellow (unaffected) eyes four months after remission from PAACG, the peripapillary NFL thickness was statistically significant (P<0.001). RNFL thickness at diverse time points after an episode of PAACG is shown in Table 3. After the attack, the average thickness of the RNFL significantly varied between time points (within 2 weeks, one month, two months, three months, and four months) (P<0.001). In other words, the RNFL changes in thickness over time. The least significant difference (LSD, P value =0.06) was found to not be significant in months 2, 3 and 4 after episodes, based on a repeated measure ANOVA. In other periods after the episodes, there was a significant change (P<0.001). It was significant to find a change among time periods and groups under the interaction condition (P<0.001). According to our results, there is no difference between the RNFL changes in affected and unaffected eyes.

Table 1: Patients are compared with normal controls after unilateral PAACG.

Characteristic	Normal Controls (n=60)	Fellow Eyes (n=60)	Attacked Eyes (n=60)	P-value
Age in years	57.8±9.42	58.8±9.46	58.8±9.47	0.7
IOP (mm Hg)	14.5±2.8	14.0±3.3	14.2±3.5	0.61
Visual field				
MD (DB)	1.176±0.87	1.85±0.87	6.56±3.56	<0.001
PSD (DB)	1.47±0.56	2.12±1.13	3.85±2.34	<0.001

Table 2: Measurement of RNFL thickness (mm) by status OCT after 2 weeks and after 4 months in PAACG-affected, normal, and fellow-affected eyes

Parameter and Time Point	Affected Eyes Means±SD	Fellow Eyes Means±SD	Normal Eyes Means±SD	P-value
Average thickness				
2 weeks	143.7±23.8	108.4±9.8	107.8±8.7	<0.001
4 Months	84.7±18.9	108.9±11.9	107.8±8.7	<0.001
Superior quadrant				
2 weeks	171.1±31.4	133.2±18.6	128.7±20.6	<0.001
4 months	96.4±27.8	131.8±19.9	—	<0.001
Nasal quadrant				
2 weeks	113.0±36.9	86.8±19.2	84.5±19.4	<0.001
4 months	67.2±25.6	83.8±18.4	—	<0.001
Inferior quadrant				
2 weeks	195.6±35.2	148.7±17.22	144.5±21.6	<0.001
4 months	111.6±31.8	152.2±16.7	—	<0.001
Temporal quadrant				
2 weeks	94.5±36.8	71.4±13.3	73.9±12.6	<0.001
4 months	61.8±15.9	75.1±17.5	—	<0.001

Table 3: In acute attacks and fellow eyes, OCT is used to measure the mean RNFL thickness.

Variable	Means RNFL*	
	Acute Attack Eyes (N=25)	Fellow Eyes (N=25)
Within 2weeks	141.2±19.9	107.5±7.8
Month 1	104.9±21.2	109.2±8.7
Month 2	87.8±22.9	107.5±8.3
Month 3	83.8±21.6	108.9±7.7
Month 4	84.3±24.2	107.5±7.2

IV. DISCUSSION

There is a possibility that acute primary angle closure (APAC) may permanently damage the optic nerve after it has recovered from acute treatment without obvious cupping. As reported by Marraffa et al.,[10] in 75% of the cases of acute ACG that resolve, the disks are barely cupped (C/Dr0.3), while in 15% of the cases, there is disk atrophy without clear cupping. With an acute ACG that has been resolved in 55% of cases, the visual field may be normal.[11] The impact of an AACG attack on visual field damage was assessed by Bonomi et al.,[11] using automated perimetry 36 to 48 hours after remission. There was a wide range of MD between 0.35 and 26.4 dB. There were some forms of visual field defects in 85%, with 15% having a normal visual field. Different studies have reported different findings regarding post-APAC visual field defects.[11–13] Perimetry may not be uniformly effective in elderly participants with slow sensory-motor coordination, which may explain the inconsistent results. There was also a possibility that mydriasis and post-APAC cataract might have affected the visual field test.[14,15] Despite clear and repeated instructions, 53.3% of the participants failed to produce a reliable automated perimetry result in the current study.[2] We previously described OCT measurements of RNFL loss in POAG.[8,17,18] Would it be possible to examine PAACG with an OCT? Four months after the episode, we studied the affected eyes; we institute a statistically significant decrease in average peripapillary NFL thickness but no decrease in superior, inferior, or temporal quadrants. It is like primary open angle glaucoma (POAG) when the RNFL of the attacked eyes becomes attenuated either locally or diffusely.[17] Because of short-duration episodes of PAACG, Tsai and Chang et al.,[16] observed significant changes in various GDx parameters in patients. According to this study, the elevated local attenuation rate (28/41) and smaller average thickness reductions show that RNFL losses after APAC are more localized than diffuse.[19] GDx measurements showed significant reductions in superior RNFL thickness after PAACG episodes, according to Aung et al.[19] In APAC, the RNFL changes suggest a preferential loss of nerve fibers along the superior and inferior peripapillaries caused by IOP, similar to the POAG report of nerve fiber loss along the upper and lower poles.[20,21] In table 2, the average, superior, nasal, and inferior quadrant thicknesses of PAACG-attacked eyes were significantly increased than those of control eyes two weeks after remission (P-value <0.001). According to Aung et al., GDx measurements after PAACG revealed that average RNFL thickness decreased significantly between weeks 2 and 16 after the episode. Hydropic degeneration and impaired axoplasm may cause an enlargement of the optic disc during an acute attack of PAACG.[1] In the event of an episode of PAACG, swollen RNFL can lead to an increase in the thickness of the peripapillary RNFL. Acutely attacked eyes may have a thicker RNFL than a normal eye if swelling increases RNFL thickness more than glaucoma decreases RNFL thickness. Most PAACG eyes increased in RNFL thickness within 2 weeks of the episode, except for 5/60. There were two patients with a previous episode of acute attack among the five patients whose RNFL did not increase. Additionally, after IOP was controlled, the average RNFL thickness in the 5 unchanged eyes also gradually decreased, eventually falling to a level lower than in their peers after 2 months. Therefore, it is possible that the RNFL atrophy may not be shown in patients whose RNFL thickness increases less than those whose RNFL thickness decreases due to glaucoma, since the increase due to swell is less than the reduction caused by glaucoma. After an acute episode, RNFL thickness may be normal, especially in patients whose IOP was elevated and the RNFL was damaged before the acute attack. It is possible that OCT did not detect structural damage due to a mild attack or because the damage was not severe enough for structural damage to occur. Due to the small sample size, this may have been due to inter-individual differences in coping with acute IOP elevations.[22] The RNFL must be allowed time to decrease in swelling. Thin (atrophy) and thick (edema) are dynamic processes of the RNFL. The OCT measurements can provide information on the process of edema decreasing in vivo. The pathogenesis of raided IOP in this condition may be elucidated by this finding. Using OCT, we observed that, immediately (within 2 weeks) following an acute angle closure episode, RNFL thickness increased in eyes, followed by a decrease (up to 3 months) in RNFL thickness over time in eyes that were longitudinally followed. The reduction in edema was apparent from two weeks (141.2 mm) to month one (104.9 mm) and from month one to month two (87.8 mm), then gradually decreased after two months. A significant difference was not found between months 2 and 3 and month 4 following an episode. (P-value=0.07). RNFL thickness fluctuated in accordance with the unaffected eyes among month 3 and 4 following IOP control. Three months after IOP control, the RNFL thickness was considered to be stable. After controlling IOP for 3 months, it is the best time to detect RNFL defects in acutely attacked eyes. RNFL thickness increased or was normal a month after an acute episode when RNFL swelling was not reduced. At one month following IOP control, the mean average RNFL thickness of acute attack eyes measured by OCT was 104.9 mm, which is very close to normal. It is likely that PAACG patients would have a negative result at this time, however we cannot ensure that structural damage did not exist or that OCT was not sensitive. Therefore, examination time should be considered. In an investigation conducted by Tsai and Chang et al.,16 the average RNFL thickness, ellipse average, superior, inferior, temporal, and nasal thickness parameters did not differ significantly after PAACG remission. One month after the PAACG remission, GDx was used. Due to PAACG remission, the attacks were less severe. This may be due to the use of GDx one month after remission from PAACG.

As a limitation of the study, RNFL thickness change in a clinical population was too small to be accurately reflected in the longitudinally followed sample. Based on their study, Lai et al.,2 Concluded that whether NFL damage is severe depends on the duration of the attack. Acute angle closure may have resulted in elevated IOP for some individuals who had ongoing RNFL damage prior to the acute attack. In light of the magnitude of RNFL thickness escalation instantly following an acute angle closure episode, we are unable to analyze the factors that may influence RNFL outcome. We are also unable to predict how the affected eyes will function. PSD and MD were significantly different among the affected eyes and their fellows, as well as between the affected eyes and the normal eyes. Functional changes were also affected by structural changes. As a result of an APAC episode related with normal vision, Tsai et al.,[3] noted that the thickness of the inferior region RNFLs was significantly reduced in the attacked eyes. This indicates that functional loss is preceded by RNFL changes in PAACG as measured by perimetry like POAG. With OCT providing objective and quantitative assessment of RNFL, can it be used as a physical index or markers for diagnosing and classifying PAACG? In other words, changes in the thickness of RNFL measured by OCT within 2 weeks of an acute angle-closed attack or decreases in RNFL 2 months after IOP is controlled are indicators that there have been previous episodes.

V. CONCLUSION

In our study, we found that the use of OCT to detect objective structural changes caused by elevated IOP attacks can be beneficial for better sympathetic the pathologic retinal variations intricate in PAACG, and for the valuation and administration of the condition.

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