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Evaluation of retinal nerve fibre layer, ganglion cell layer and choroidal thickness with optical coherence tomography in migraine patients: a case-control study

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Background: Evaluation of retinal nerve fibre layer (RNFL), ganglion cell layer (GCL) and choroidal thickness (CT) with optical coherence tomography (OCT) in chronic migraine patients, to compare with healthy controls.

Material and Method: Ninety-four eyes of 47 chronic migraine patients (Group 1) and 68 eyes of 34 healthy individuals (Group 2) were included in this prospective case-control study. The right and left eyes were separately evaluated. Mean peripapillary RNFL thicknesses, mean GCL measured from superior and inferior quadrants, and mean CT were measured at three different regions (central, 500 μm nasal and temporal region of the fovea).

Results: There was no statistically significant differences in RNFL between the two groups ($p > 0.05$), while CT values were significantly higher and GCL values were significantly lower in chronic migraine groups ($p < 0.05$). There were no statistically significant differences between migraine duration, frequency and length of attacks, presence of aura, relation to menstrual cycle, white matter lesions in cranial magnetic resonance imaging and RNFL, GCL and CT ($p > 0.05$).

Discussion: In this study, we observed chronic migraine disease does not have any effect on peripapillary RNFL thickness; however, increases in CT and decreases in GCL thickness were observed in migraine patients.

Key words: choroidal thickness, chronic migraine, optical coherence tomography, retinal ganglion cell layer, retinal nerve fibre layer

Migraine is a highly common, chronic and multifactorial neurovascular disease which is characterised by typical headaches triggered with internal and external factors in genetically predisposed individuals. Among neurological diseases, life-time prevalence in adults is reported to be 33 per cent in females and 13 per cent in males.[1] There is no biological marker or imaging finding for the diagnosis of migraine. The diagnosis is solely based on a good patient history and neurological examination. According to 2004 classification of the International Headache Society (IHS),[2] migraine is categorised into subgroups depending on coexistence of neurological or other symptoms along with headache.

Recently, optical coherence tomography (OCT) has begun to be used for various neuro-ophthalmic applications. OCT is a reliable and reproducible technique in retinal nerve fibre layer (RNFL) thickness measurement.[3,4] It is a non-invasive, non-contact imaging technique similar to ultrasound imaging, except that it uses infrared wavelengths of 820 nanometers.[5] Migraine is being studied continually due to its high prevalence and socio-economic effect, and hopes of treatment increase as the underlying pathophysiological mechanisms are better understood.[6]

We aimed to assess with OCT, RNFL, ganglion cell layer (GCL) and choroidal thickness in migraine patients and compare these with healthy controls.

METHODS

This prospective case-control study was performed at Adiyaman University, Training and Research Hospital, Neurology and Ophthalmology Departments. All participants gave their informed consent according to the Declaration of Helsinki and the study was approved by the internal ethics board at Adiyaman University.

Patient selection

Subjects with chronic migraine were diagnosed by a neurologist, according to the diagnostic criteria of IHS Classification 2004[2] after excluding by clinical examination other reasons for headache. Patients with no systemic disease, no retinal or choroidal pathologies, and no refractive errors were included. The study group consisted of chronic migraine patients followed in Adiyaman University Training and Research Hospital Neurology Clinic, while the control group consisted of healthy volunteers who presented to Adiyaman University Training and Research Hospital Ophthalmology Clinic with eye-related complaints and were not found to have

ocular or systemic diseases and headache symptoms after examinations.

Ophthalmic examination

Best corrected visual acuity measurement, slitlamp biomicroscopic examination, intraocular pressure measurement with Goldmann applanation tonometry, gonioscopy with three mirror contact lenses and funduscopic examination were performed for all the participants. RNFL, GCL and choroidal thickness measurements were taken with spectral domain OCT (Heidelberg Engineering, Heidelberg, Germany) (Figures 1–3). Choroidal thickness was measured at three different regions: central fovea, 500 μm nasal and temporal to the fovea; and mean macular corneal thickness value was obtained by taking the average of these three regions. Choroidal thickness was determined as the distance between the posterior edge of the retinal pigment epithelium and choroid-sclera junction (Figure 1). Automated measurements of the macular GCL and RNFL thicknesses were performed by a built-in software program (Heidelberg Engineering) (Figures 2 and 3). The GCL thickness was measured between the surface of the internal limiting membrane and the outer boundary of the inner plexiform layer (Figure 2).

Optical coherence measurements of all patients were taken by the same physician between 9:00 and 10:00 hours to minimise the effect of diurnal waves. The evaluations of the OCT images were made independently by two observers (AS and ASK). OCT scans of poor quality, defined by low signal strength (<15 dB), and any images with motion artifacts or obvious decentration of the measurement circle were excluded from the analysis. Migraine attacks in the one patient can be on either the right or left side with no distinctive lateralisation, and therefore both eyes of patients were evaluated.

Statistical analysis

The Shapiro–Wilk test was used to test the normality of variables. The variables that were normally distributed were presented as mean ± standard deviation and were compared using Student's t-test. The variables that were not normally distributed were presented as median (minimum-maximum) and were compared with Mann-Whitney U-test. Fisher's exact chi-square test was used to compare categorical variables which were given as 'n' and percentage. Pearson or Spearman's correlation analysis was performed to determine the relationship between migraine duration, frequency and length of attacks, presence of aura, relation to menstrual cycle, white matter lesions in cranial magnetic resonance imaging (MRI) and RNFL, GCL and choroidal thickness. One-way analysis of variance (ANOVA) or Kruskal-Wallis test was used to determine differences between the four groups. The Tukey test was used as a post hoc test, if the ANOVA test was statistically significant. The Dunn's test was used for post hoc test after Kruskal-Wallis test. Significance level was set as $\alpha = 0.05$. The data were analysed using the IBM SPSS Statistics 22 software package (IBM, Armonk, New York, USA).

RESULTS

This study was performed at Adiyaman University, Training and Research Hospital, Neurology and Ophthalmology Departments on 162 eyes of 81 patients. Ninety-four eyes of 47 chronic migraine patients who were followed in our Neurology outpatient clinic (Group 1) and 68 eyes of 34 healthy individuals (Group 2) were included in this prospective case-control study. The median age of the control group was 34 (21–59) years, while the median age of the migraine group was 37 (18–53) years. In the migraine group 13 of the patients were male (27.6 per cent), while in the control group 13 of the healthy volunteers were male (38.2 per cent). There were no statistically significant differences in age and sex distribution between the two groups ($p = 0.784$ and $p = 0.342$, respectively) (Table 1).

The RNFL thicknesses of all quadrants were not significantly different between the two groups ($p > 0.05$) (Tables 2 and 3).

Choroidal thickness was significantly higher in migraine patients on both right and left sides than in the control group ($p < 0.001$ for both eyes), and GCL thickness was significantly lower in migraine

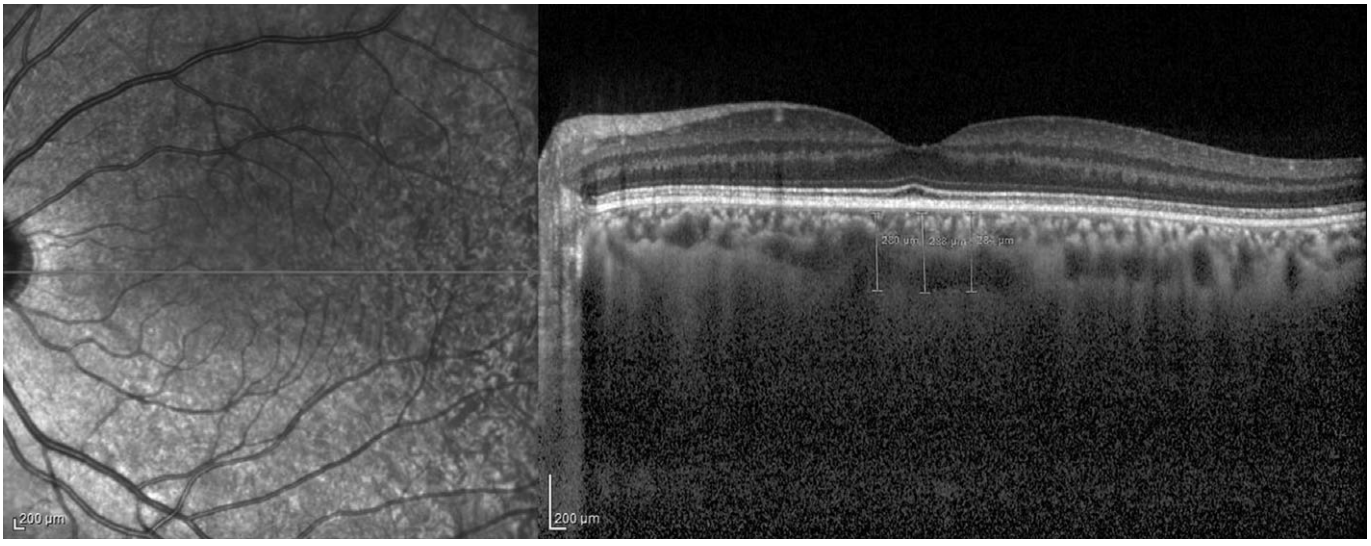

Figure 1. Choroidal thickness measurement with optical coherence tomography

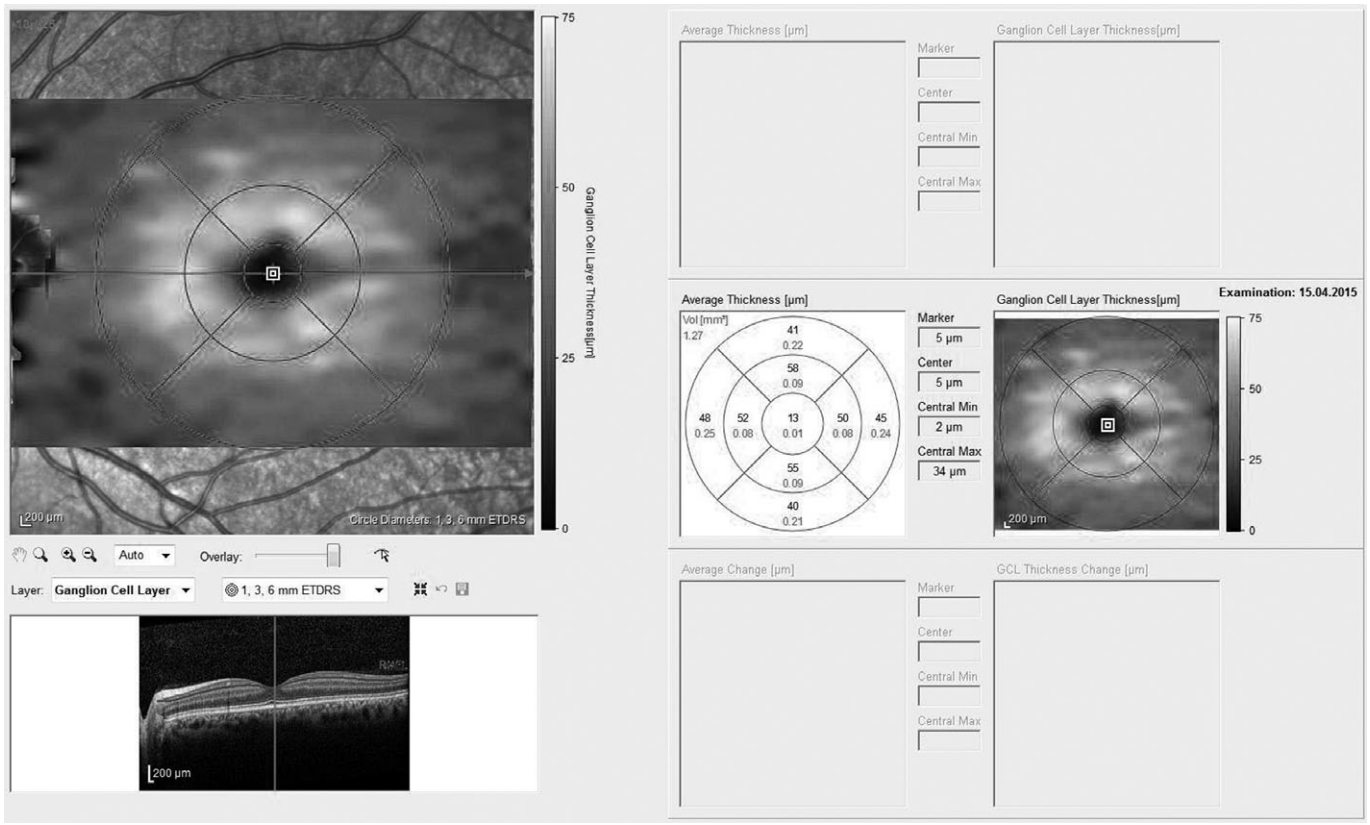

Figure 2. The ganglion cell layer thickness was measured between the surface of the internal limiting membrane and the outer boundary of the inner plexiform layer

patients on both right and left sides ($p = 0.002$ and $p = 0.001$, respectively).

Migraine patients were divided into two subgroups depending on whether the patients presented with a migraine attack. There was no statistical difference in RNFL and GCL measurements between the two subgroups ($p > 0.05$) (Table 4). Mean right choroidal thickness value in patients in the migraine attack subgroup was 320 µm (247–460 µm), while it was 256 µm (192–428 µm) in patients with migraine in the stable phase subgroup. Mean left choroidal thickness value in the migraine attack subgroup was 319 µm (223–466 µm), while it was 241 µm (207–385 µm) in the stable migraine subgroup. Choroidal thickness measurements were significantly higher in the migraine attack subgroup compared to the stable migraine subgroup (right eye, $p = 0.041$; left eye, $p = 0.022$) (Table 4).

There were no statistically significant differences among the migraine subgroups and control group with regard to the RNFL thicknesses ($p > 0.05$); however, significant differences among the three groups were evident with respect to the corneal thickness and GCL values ($p < 0.05$). Corneal thickness values were significantly higher in the attack subgroup compared with the control and stable migraine subgroup ($p < 0.05$); however, there was no significant difference between the control group and stable migraine subgroups in respect to choroidal thicknesses ($p > 0.05$). GCL values were significantly higher in the control group compared with the migraine subgroups ($p < 0.05$).

There were no statistically significant differences between migraine duration, frequency and length of attacks, presence of aura, relation to menstrual cycle, white matter lesions in cranial MRI and RNFL, GCL and choroidal thickness ($p > 0.05$).

DISCUSSION

Focal reduction of the regional cerebral blood flow is detected in migraine attacks, especially in migraine with aura, which starts in the posterior circulation. Reduced blood flow and vasospasm are frequently identified to one hemisphere.[7] On rare occasions, hypoperfusion starts from other parts of the brain or even retina.[8,9] Retinal infarctions resulting from retinal artery occlusions have been reported in migraine patients.[8,10] In addition, cerebral vasospasm could cause structural distortions even in temporary recurrent migraine attacks. Whether similar changes occur in the retina in migraine with or without aura is a subject of uncertainty and only a few studies have mentioned the issue. Changes in the quality of perfusion in optic nerve head microcirculation or even the retina may cause ganglion cell damage in migraine patients.[11] RNFL thickness measurements can be used as an index to assess ganglion cell and retinal nerve fibre injuries.

The retina with its receptors, ganglion cells, glial support cells and axons is considered by many anatomists to be an extension of the brain. RNFL is similar to

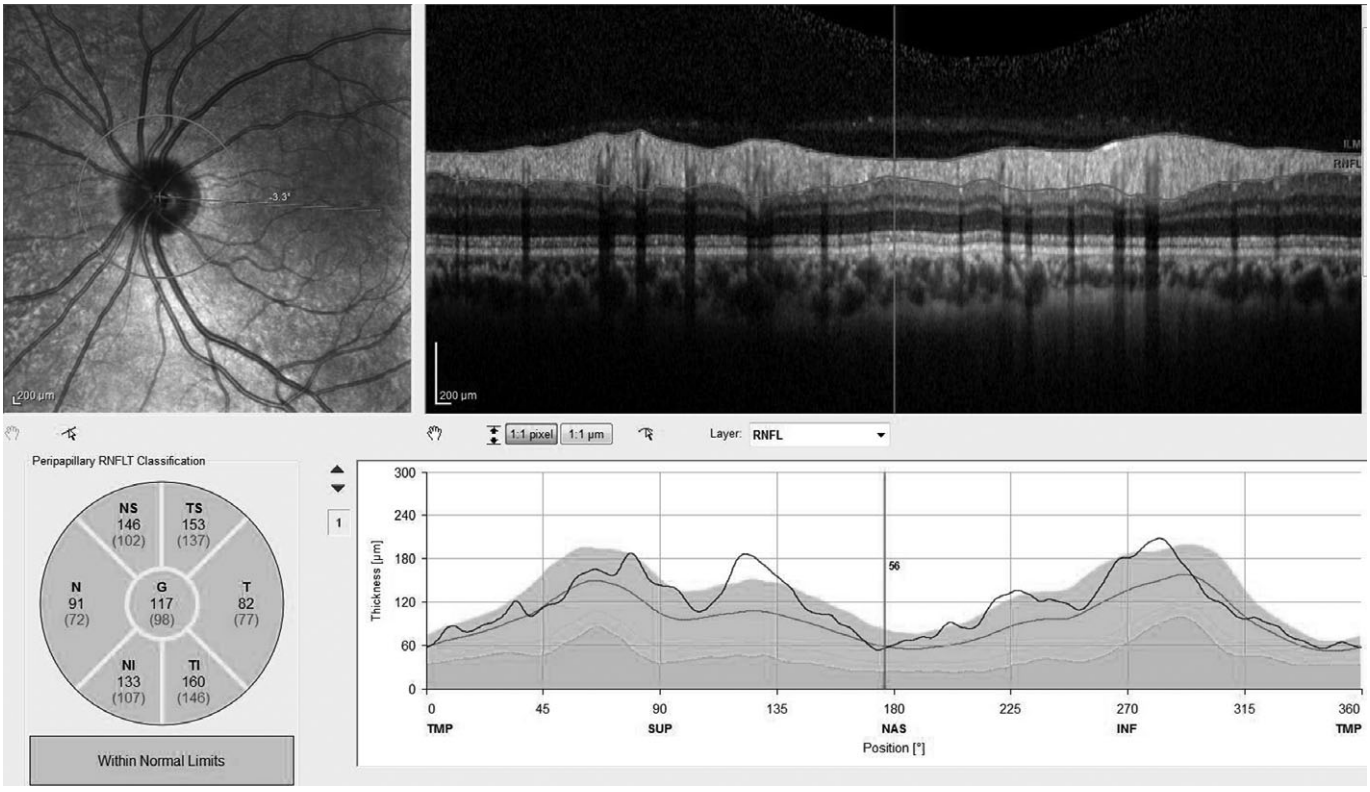

Figure 3. Retinal nerve fibre layer analysis with optical coherence tomography

grey matter of the brain and the changes in its thickness are merely due to axon damage. From this point of view, the retina is considered to be an easily traceable piece of brain. Thus, RNFL thickness follow-up with achromatic photographs has been used for a long time for the diagnosis of clinical and subclinical optic neuropathy in degenerative neurological diseases, primarily multiple sclerosis.[12,13]

RNFL and RNFL + GCL thickness is decreased in Alzheimer's disease patients compared to healthy individuals. OCT can be used as a complementary test for the diagnosis and follow-up of this disease. Researchers consider further studies necessary for confirming the correlation between RNFL and GCL thicknesses and stage of the disease.[5,14] One of the proposed mechanisms of migraine and especially migraine with aura is focal brain hypoperfusion in the posterior circulation, which involves other brain parts and retina.[11] RNFL thickness measurements can be used as an index to assess ganglion cell and retinal nerve fibre damages.

The retinal ganglion cells are populous in the macula and form a stratified multicellular layer within the central six degrees of visual field. Thus, loss of axons and the corresponding soma in this location are likely to induce a thinning of the retinal GCL. The mapping of visual field location to corresponding ganglion cell soma is less complicated than the situation with the RNFL bundles and may show less inter-individual developmental variability.[15]

There are several theories regarding the aetiology of optic nerve damage in patients with migraine, which include vascular abnormalities such as vasospasm or focal

	Control (n = 34)	Migraine (n = 47)	p-value
Age (year)	34 (21–59)	37 (18–53)	0.784
Sex (male/female), n (%)	13 (38.29%) / 21 (61.8%)	13 (27.60%) / 34 (72.34%)	0.342
Migraine duration (months)	–	11–240	–
Number of migraine attacks/month	–	1–12	–
Number of migraine with aura	–	9/47	–
Menstrual cycle related migraines (n)	–	14/47	–
Lesions in cranial MRI (n)	–	7/47	–
Family history of migraine (n)	–	10/47	–
Duration of migraine attack (days)	–	1–4 days	–

Table 1. Characteristics of migraine and control groups

Parameters (right eye)	Control group (n = 34)	Migraine group (n = 47)	p-value
(Normally distributed parameters were presented as mean ± standard deviation and were compared using Student's t-test)			
R-RNFL-NS (μm)	112.17 ± 20.07	115.02 ± 20.03	0.346
R-RNFL-T (μm)	73.29 ± 8.55	75.98 ± 8.96	0.337
(Not normally distributed parameters were presented as median [minimum–maximum] and were compared using Mann-Whitney U test)			
R-RNFL-N (μm)	83 (136–55)	81 (161–45)	0.354
R-RNFL-NI (μm)	123 (214–88)	122 (182–72)	0.563
R-RNFL-TI (μm)	148 (195–116)	151 (190–87)	0.765
R-RNFL-TS (μm)	142 (197–112)	146 (203–114)	0.546
R-RNFL-G (μm)	106 (142–89)	105 (136–80)	0.813
R-CT (μm)	250 (371–218)	316 (460–192)	**<0.001**
R-GCL (μm)	1.20 (1.32–1.14)	1.15 (1.39–1.00)	**0.002**

Bold value indicates statistical significance.
CT: choroidal thickness, GCL: ganglion cell layer, R: right eye, RNFL: retinal nerve fibre layer, RNFL-G: global RNFL thickness, RNFL-N: RNFL of nasal quadrant, RNFL-NI: RNFL of inferionasal quadrant, RNFL-NS: RNFL of superonasal quadrant, RNFL-T: RNFL of temporal quadrant, RNFL-TI: RNFL of inferotemporal quadrant, RNFL-TS: RNFL of superotemporal quadrant.

Table 2. Optic coherence tomography analysis results in migraine and control groups (right eye findings)

ischaemia that may be either intermittent or chronic.[16] In this study, we assessed RNFL, GCL and choroidal thickness with OCT in migraine patients and compared these with the control group.

In a study that analysed RNFL and ganglion cell complex thickness with spectral OCT in chronic migraine patients, RNFL and GCC thicknesses were statistically different in all quadrants between migraine patients and a control group.[17] Contrary to this, we did not find a significant difference in RNFL thickness between the study and control groups; however, GCL thickness was significantly lower in migraine patients, similar to the previously mentioned study. Finding significant thinning in GCL but no significant difference in RNFL may be due to short mean disease duration or low numbers of migraine attacks. In another study that compared 50 migraine and 50 healthy subjects, RNFL was found thinner in migraine patients but the opposite was found and was statistically significant in our study.[18]

In another study OCT was used to investigate RNFL thickness in migraine patients

Parameters (left eye)	Control group (n = 34)	Migraine group (n = 47)	p-value
(Normally distributed parameters were presented as mean ± standard deviation and were compared using Student's t-test)			
L-RNFL-NI (μm)	124.55 ± 27.29	122.32 ± 22.36	0.597
L-RNFL-TI (μm)	151.00 ± 18.02	152.96 ± 21.95	0.756
L-RNFL-TS (μm)	145.08 ± 14.57	144.62 ± 16.24	0.687
L-RNFL-G (μm)	106.23 ± 11.37	105.97 ± 10.13	0.721
(Not normally distributed parameters were presented as median [minimum-maximum] and were compared using Mann-Whitney U test)			
L-RNFL-NS (μm)	125 (168–88)	120 (205–84)	0.312
L-RNFL-N (μm)	78 (122–56)	76 (141–42)	0.305
L-RNFL-T (μm)	70 (93–55)	71 (93–48)	0.612
L-CT (μm)	244 (404–210)	300 (466–207)	**<0.001**
L-GCL (μm)	1.19 (1.32–1.14)	1.15 (1.36–1.00)	**0.001**

Bold value indicates statistical significance.
CT: choroidal thickness, GCL: ganglion cell layer, L: left eye, RNFL: retinal nerve fibre layer, RNFL-G: global RNFL thickness, RNFL-N: RNFL of nasal quadrant, RNFL-NI: RNFL of inferionasal quadrant, RNFL-NS: RNFL of superonasal quadrant, RNFL-T: RNFL of temporal quadrant, RNFL-TI: RNFL of inferotemporal quadrant, RNFL-TS: RNFL of superotemporal quadrant.

Table 3. Optic coherence tomography analysis results in migraine and control groups (left eye findings)

Parameters	Migraine attack subgroup (n = 14)	Stable migraine subgroup (n = 33)	p-value
R-RNFL-NS (μm)	120 (87–173)	108 (83–151)	0.549
R-RNFL-NI (μm)	122 (72–182)	122 (74–165)	0.748
R-RNFL-N (μm)	78 (48–161)	80 (45–121)	0.711
R-RNFL-TI (μm)	161 (87–190)	151 (114–185)	0.447
R-RNFL-TS (μm)	146 (124–168)	144 (114–203)	0.389
R-RNFL-T (μm)	76 (52–102)	75 (53–89)	0.826
R-RNFL-G (μm)	108 (80–136)	104 (83–124)	0.398
L-RNFL-NS (μm)	123 (96–205)	118 (84–164)	0.304
L-RNFL-NI (μm)	124 (79–160)	121 (71–161)	0.798
L-RNFL-N (μm)	76 (42–141)	77 (46–113)	0.816
L-RNFL-TI (μm)	154 (98–186)	149 (104–190)	0.522
L-RNFL-TS (μm)	147 (114–172)	140 (116–187)	0.113
L-RNFL-T (μm)	72 (48–92)	71 (52–93)	0.742
L-RNFL-G (μm)	106 (86–124)	103 (84–119)	0.296
R-CT (μm)	320 (247–460)	256 (192–428)	**0.003**
L-CT (μm)	319 (223–466)	241 (207–385)	**0.001**
R-GCL (μm)	1.16 (1.00–1.29)	1.14 (1.01–1.39)	0.349
L-GCL (μm)	1.15 (1.08–1.29)	1.15 (1.00–1.36)	0.687

Bold value indicates statistical significance.
CT: choroidal thickness, GCL: ganglion cell layer, L: left eye, R: right eye, RNFL: retinal nerve fibre layer, RNFL-G: global RNFL thichness, RNFL-N: RNFL of nasal quadrant, RNFL-NI: RNFL of inferionasal quadrant, RNFL-NS: RNFL of superonasal quadrant, RNFL-T: RNFL of temporal quadrant, RNFL-TI: RNFL of inferotemporal quadrant, RNFL-TS: RNFL of superotemporal quadrant.

Table 4. Optic coherence tomography analysis results in migraine subgroups (Mann-Whitney U test, median [min–max])

and this was compared with healthy controls. The results revealed that nasal quadrant RNFL thickness was significantly thinner in migraine subjects compared with those of age-matched healthy subjects. Other parameters showed no difference between the two groups.[19] A study by Martinez et al.[20] examined 70 patients with migraine and 53 normal controls using OCT. The mean retinal layer thickness in patients with migraine was within normal ranges but RNFL thickness in the temporal quadrant was significantly reduced in patients with migraine compared to the normal controls. Except for nasal/temporal quadrant values, RNFL thickness measurements of the studies above were similar to those in our results.

Increased choroidal thickness during acute migraine attack in migraine patients can be explained with the vascular pathogenesis of the disease. A study showed that mean choroidal thickness during migraine attack was significantly higher than the mean choroidal thickness of a control group.[21] In our study, mean right and left choroidal thickness values of patients in the attack period were measured and found to be significantly higher than the patients in the stable migraine phase and the control group.

Another study suggests that migraine leads to a reduction in the peripapillary RNFL thickness and thinning in choroidal structures. These findings can be explained by a chronic ischaemic insult related to migraine pathogenic mechanisms and these findings are considered supportive of the relationship between glaucoma and migraine.[22]

In a study by Ekinci et al.,[23] 90 migraine patients with and without aura and 30 healthy control subjects were included. The migraine with aura group and migraine without aura group were compared with the control group in terms of RNFL, GCL and choroidal thickness. In the migraine patients with aura, RNFL and GCL values were observed to be significantly thin compared to both the migraine patients without aura and the control group. In addition, choroidal thinning was observed in both migraine groups. This study together with our study supports the hypothesis that suggests the pathophysiology of migraine with and without aura to be different.

In another study, mean RNFL and GCL thickness of migraine patients were not correlated with migraine duration, frequency or duration of migraine attacks or presence of aura.[24] Similar to this study, we did not find a significant difference in RNFL and GCL thickness with migraine duration, patients whose migraine attacks are related and not related to menstrual cycle, patients with and without family history of migraine, and patients with and without aura. Theoretically, pathophysiology of the disease suggests that having migraine for a long period of time, higher attack frequency or longer duration of attacks would cause more damage and result in changes in measured thickness values; however, results of various studies show controversial

findings. Further studies are needed to clear the correlation between the progression of the disease and RNFL and GCL thickness values.

In conclusion, we found migraine to be correlated with GCL and choroidal thickness. The small number of patients included in our study was considered to be the reason for not finding a correlation with RNFL thickness.

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REFERENCES

1. Bolay H, Moskowitz MA. The neurobiology of migraine and transformation of headache therapy. In: Stephen Waxman, ed. Neuroscience, Moleculer Medicine and the Theraputic Transformation of Neurology. Elsevier, 2004; pp: 107–123.
2. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders. *Cephalalgia* 2004; 24 (Suppl.1): 9–160.
3. Mwanza JC, Oakley JD, Budenz DL, Chang RT, Knight OJ, Feuer WJ. Macular ganglion cell inner plexiform layer: automated detection and thickness reproducibility with spectral domainoptical coherence tomography in glaucoma. *Invest Ophthalmol Vis Sci* 2011; 52: 8323–8329.
4. Kim KE, Yoo BW, Jeoung JW, Park KH. Long-term reproducibility of macular ganglion cell analysis in clinically stable glaucoma patients. *Invest Ophthalmol Vis Sci* 2015; 56: 4857–4864.
5. Marziani E, Pomati S, Ramolfo P et al. Ganglion layer cells thickness as biologic marker of Alzheimer's disease. Milan: University of Milan, 2009.
6. Ferrari MD. Migraine. *Lancet.* 1998; 351: 1043–1051.
7. Olesen J, Friberg L. Xenon-133 SPECT studies in migraine without aura. In: Olesen J, ed. Migraine and other headaches: The vascular mechanisms. Philadelphia, PA: Lippincott Williams & Wilkins, 1991; pp 237–243.
8. Killer HE, Forrer A, Flammer J. Retinal vasospasm during an attack of migraine. *Retina* 2003; 23: 253–254.
9. Abdul-Rahman AM, Gilhotra JS, Selva D. Dynamic focal retinal arteriolar vasospasm in migraine. *Indian J Ophthalmol* 2011; 59: 51–53.
10. Beversdorf D, Stommel E, Allen C, Stevens R, Lessell S. Recurrent branch retinal infarcts in association with migraine. *Headache* 1997; 37: 396–399.
11. Martinez A, Proupim N, Sanchez M. Retinal nerve fibre layer thickness measurements using optical coherence tomography in migraine patients. *Br J Ophthalmol* 2008; 92: 1069–1075.
12. Frisen L, Hoyt WF. Insidious atrophy of retinal nerve fibers in multiple sclerosis. Funduscopic identification in patients with and without visual complaints. *Arch Ophthalmol* 1974; 92: 91–97.
13. Elbol P, Work K. Retinal nerve fiber layer in multiple sclerosis. *Acta Ophthalmol (Copenh)* 1990; 68: 481–486.
14. Valenti DA. Neuroimaging of retinal nerve fiber layer in AD using optical coherence tomography. *Neurology* 2007; 69: 106.
15. Kardon RH. Role of the macular optical coherence tomography scan in neuroophthalmology. *J Neuroophthalmol* 2011; 31: 353–361.
16. Tan FU, Akarsu C, Gullu R. Retinal nerve fiber layer thickness is unaffected in migraine patients. *Acta Neurol Scand* 2005; 112: 19–23.
17. Uludag G, Ozer D, Tanyildiz B, Ceylan E, Cinici E. Evaluation of retinal nerve fiber layer and ganglion cell complex thicknesses by optical coherence tomography in chronic migrain patients. *J Glaucoma-Cataract* 2014; 9: 263–266.
18. Yülek F, Dirik EB, Eren Y et al. Macula and retinal nerve fiber layer in migraine patients: analysis by spectral domain optic coherence tomography. *Semin Ophthalmol* 2015; 30: 124–128.
19. Karalezli A, Simsek C, Celik G, Eroglu FC. Evaluation of choroidal thickness using spectral-domain optical coherence tomography in migraine patients during acute migraine attacks: a comparative study. *Eye (Lond)* 2014; 28: 1477–1481.
20. Martinez A, Proupim N, Sanchez M. Retinal nerve fibre layer thickness measurements using optical coherence tomography in migraine patients. *Br J Ophthalmol* 2008; 92: 1069–1075.
21. Sorkhabi R, Mostafaei S, Ahoor M, Talebi M. Evaluation of retinal nerve fiber layer thickness in migraine. *Iran J Neurol* 2013; 2: 51–55.
22. Demircan S, Ataş M, Arık Yüksel S et al. The ımpact of migraine on posterior ocular structures. *J Ophthalmol* 2015: 868967.
23. Ekinci M, Ceylan E, Cağatay HH et al. Retinal nerve fibre layer, ganglion cell layer and choroid thinning in migraine with aura. *BMC Ophthalmol* 2014; 14: 75.
24. Can GD, Sarac O, Can ME. Evaluation of retinal nerve fiber layer, and ganglion cell complex with optical coherence tomography in migraine patients. *Medical Network Ophthalmol* 2013; 20: 127–131.