Special Focus, Glaucoma, Original Article

Glaucoma and optical coherence tomography changes in migraine: A comparative cross-sectional study

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Purpose: To study the prevalence of glaucoma among adults with migraine and the effect of migraine on peripapillary retinal nerve fiber layer (pRNFL) and central macular thickness (CMT) using optical coherence tomography (OCT) compared to those without migraine headache, i.e. in tension-type headache (TTH) and normal group. **Methods:** One hundred and eleven patients (222 eyes) were recruited in three groups. migraine, TTH, and normal subjects visiting hospital outpatient services. After noting demographic details and pertinent history, ophthalmological evaluation including optic disc for glaucomatous changes along with computerized visual field testing and OCT for pRNFL thickness and CMT was performed in all eyes. Continuous variables were compared using ANOVA or Kruskal–Wallis test, while categorical variables including the association of glaucoma with migraine were analyzed using Chi-square or Fisher's exact test. **Results:** Prevalence of glaucoma in migraine group (12.2%) was more than in comparison groups (6.8% in TTH, 4.1% in normal) which was however not significant (Fisher's exact $P = 0.207$). Average pRNFL thickness (103.59 ± 12.82 µm) and thickness in nasal (90.49 ± 19.19 µm) and temporal quadrants (70.58 ± 16.13 µm) and CMT (213.78 ± 19.81 µm) were significantly reduced (ANOVA $P < 0.05$) in migraine patients when compared to the other groups and this was independent of the presence of glaucoma. **Conclusion:** Prevalence of glaucoma is not significantly higher in migraine patients. However, migraine causes thinning of retinal layers on OCT that is statistically significant.

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Migraine is a recurrent typically unilateral pulsating headache disorder manifesting in attacks lasting 4–72 hours affecting 10% to 11% of people worldwide.[1] Though considered a disorder of the central nervous system, both neural and vascular systems are implicated in migraine pathogenesis.

Glaucoma involves the progressive loss of retinal ganglion cells resulting in characteristic changes of the optic nerve head along with visual field defects with or without elevated intraocular pressure and is the leading cause of irreversible blindness worldwide.[2]

The role of the trigeminovascular system in migraine pathogenesis is well understood. It mediates vasodilatation of meningeal vessels and dural extravasation of plasma protein and inflammatory mediators which in turn results in sterile inflammation and vasospasm producing the typical pulsatile headache.[3,4] Among the glaucoma types, normal tension glaucoma (NTG), a subtype of primary open angle glaucoma (POAG), is associated with retinal vascular dysregulation and poor blood flow to the optic nerve head and is seen more frequently in migraine patients suggesting a similar underlying vasospastic mechanism in both.[5]

It is proposed that attacks of migraine lead to vasospasm and decreased blood flow in the retina and optic nerve. Thus, ischemia of the ocular neural tissues results in retinal ganglion cells (RGC) loss and thinning of retinal nerve fiber layer and macular layers which can be measured using optical coherence tomography (OCT). Ocular blood flow changes are involved both in the pathogenesis of glaucoma and the progression of glaucomatous damage.[6]

Based on the above, we hypothesized that glaucoma and retinal thinning on OCT are more common among adult patients with migraine when compared to nonmigraineurs. Previous studies have separately found no conclusive evidence of increased association of any type of glaucoma with migraine[7-9] and some extent of significant thinning of retinal layers on OCT in migraine patients[6,10,11] but as yet, there is a lack of consensus.

Our main purpose for this study was to investigate whether a common condition like migraine is a predictor of glaucoma along with studying the benefit of using tools like OCT to pick up thinning of retinal layers in migraine patients even in those eyes which do not show obvious changes of the optic disc or field defects and may clinically be judged as normal.

Methods

Study population and design

This was a cross-sectional comparative study conducted in a tertiary care hospital in South India on patients attending our

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outpatient services. Migraine patients were included in the study if they were between 18 and 80 years of age and were diagnosed by the neurologist with clinical features of migraine with or without aura which continued or had started in the last 5 years fulfilling the criteria laid down by ICHD 3 guidelines. Exclusion criteria were the presence of past history of migraine which remitted more than 5 years before, glaucoma diagnosed before onset of migraine, any secondary headache disorders, and poor vision states and any ocular media opacities which interfere with diagnostic tests like examination of the optic nerve head, OCT, and field analysis.

For the selection of patients into comparison groups, we enrolled age (plus or minus 2 years) and gender matched subjects visiting ophthalmology OPD for tension-type headache (TTH) referred by the neurologist selected as comparison group 1, while age and gender matched headache-free normal subjects were selected as comparison group 2. The same exclusion criteria were applied to these groups.

We followed the convenience sampling technique to recruit patients in three groups from June 2018 till the sample size was achieved. The sample size was calculated as 30 in each group estimated with 95% confidence interval, 90% power, and ratio between the groups to be 1:1:1. Considering nonresponsiveness of 20%, final sample size arrived at was 37 in each group, i.e. 111 overall.

Study procedure

The study protocol was in accordance with the tenets of the Declaration of Helsinki and was approved by the institutional ethics committee before commencement.

All 37 patients in each of the groups underwent thorough ophthalmological evaluation after giving informed consent for study participation. Apart from demographic details, the type and duration of migraine, presence of comorbidities like diabetes, hypertension, coronary artery disease (CAD), and family history of glaucoma, and history of spectacle use and myopia were noted.

Best-corrected visual acuity was recorded using Snellen's chart, intraocular pressure (IOP) was measured by Goldmann applanation tonometry, and gonioscopy was performed using Goldmann 2 mirror gonioprism for grading anterior chamber angles. Slit-lamp biomicroscopy was performed for optic disc examination using + 90 Diopter lens for glaucomatous changes. Central corneal thickness (CCT) was measured using an optical biometer (Lenstar LS 900, Haag-Streit, USA). All patients underwent computerized visual field testing using Humphrey's field analyzer (HFA II 750, Carl Zeiss Meditec. Inc, USA). Optical coherence tomography (Cirrus HD Model OCT500, Carl Zeiss Meditec. Inc, USA) was used to measure peripapillary retinal nerve fiber layer (pRNFL) and central macular thickness (CMT).

Definite glaucoma was diagnosed when cup disc ratio was more than 0.5 with localized rim loss, IOP greater than 21 mmHg with glaucomatous field changes. Only rise in IOP above 21 mmHg with no cupping or only a suspicious disc with normal pressures were considered as a glaucoma suspect. A subject has no glaucoma if the cup disc ratio is less than 0.5 with normal intraocular pressures. Patients with definite glaucoma and suspicious glaucoma were kept on close follow up and treatment initiated if the need arose accordingly.

Statistical analysis

Data was successfully collected from 111 patients, i.e. 37 patients (74 eyes) in each of the three groups and analyzed using SPSS 20.0.

Continuous variables like age, duration of headache, IOP, CCT, and OCT parameters like RNFL parameters (such as superior, inferior, nasal, temporal quadrants and average pRNFL) and CMT were expressed as mean, standard deviation with 95% CI, or median and interquartile range based on normality of the data distribution. They were compared between groups using ANOVA or Kruskal–Wallis test based on normality. A probability value (*P* value) of less than 0.05 was considered significant.

Categorical variables like gender, presence of comorbidities, family history of glaucoma, refractive errors, type of headache, gonioscopy findings, presence of visual field, and glaucomatous changes were expressed as percentages or proportions with 95% CI. These were compared between groups using the Chi-square test/Fisher's exact test. A *P* value of less than 0.05 was considered significant. Association of migraine with the presence of glaucoma was assessed using Fisher's exact test.

Results

Data was collected from a total of 111 patients (222 eyes), i.e. 37 patients (74 eyes) in each group, i.e. migraine, TTH, and normal subjects.

All 3 groups were found to be comparable with respect to age and sex distribution, the presence of comorbidities, family history of glaucoma, and type of refractive error [Table 1].

Out of 74 eyes in each group, 9 eyes (12.16%) in migraine group, 5 eyes (6.76%) in TTH group, and 3 eyes (4.05%) in normal group were found to have either glaucoma or suspicious glaucoma. All 17 eyes had normal IOP with disc and visual field changes and were categorized as normal tension glaucoma. Above patients were reassessed 6 monthly for evidence of progression and none have been started on any antiglaucoma medications so far.

Even though glaucomatous changes were found to be more common among patients in the migraine group, this was not statistically significant (Fisher's exact *P* value = 0.207) when compared to TTH or normal subjects. This was further corroborated with odds ratio value of 1 within 95% confidence interval suggesting no association between migraine headache and presence of glaucoma.

Outcome measures compared between the groups were CCT, IOP, gonioscopic status of angle, visual field changes, and OCT parameters. Both CCT and IOP were found to be similarly distributed in all three groups and there was no statistically significant difference between their distribution in eyes with and without glaucoma [Table 2].

Open angles were the more common gonioscopic finding in all three groups and there was no statistically significant difference between the groups (Fisher's exact *P* = 0.737). In more than one-third eyes in all groups, the visual field was found to be within normal limits [Fig. 1]. Though there was a clear trend for visual field defects in migraine patients, it failed to reach statistical significance (Fisher's exact *P* value = 0.073).

All OCT parameters were normally distributed in each group. ANOVA test was used to compare each OCT parameter among the 3 comparison groups. Average pRNFL, RNFL thickness in nasal and temporal quadrants, and CMT were significantly lower among migraine patients in all eyes, irrespective of the presence of glaucoma [Table 3] when compared to both control groups after a Bonferroni correction. However, there was no significant difference in OCT thickness among the 3 groups in eyes with glaucoma [Table 3] or among migraine patients with and without glaucoma [Fig. 2]

Table 1: Distribution of baseline characteristics between the groups

Baseline characteristics		Migraine	Tension-type headache (TTH)	Normal	Statistical test*
Age Distribution	Mean±SD (years) or Median (IQR) (years)	32.22±8.67	30.76±8.84	25 (13)	Kruskal-Wallis *P*=0.119
Sex distribution	Female *n* (%)	29 (78.4%)	30 (81.1%)	31 (83.8%)	Chi-square test *P*=0.703
	Male *n* (%)	8 (21.6%)	7 (18.9%)	6 (16.2%)	
Presence of Comorbidities (HTN)	Yes (*n*=2)	1 (2.7%)	1 (2.7%)	0	Fisher's exact test *P*=0.549
	No (*n*=109)	36 (97.3%)	36 (97.3%)	37 (100%)	
Family history of glaucoma	Yes (*n*=2)	1 (2.7%)	1 (2.7%)	0	Fisher's exact test *P*=0.549
	No (*n*=109)	36 (97.3%)	36 (97.3%)	37 (100%)	
Refractive error distribution	Emmetropia *n* (%)	50 (67.6%)	52 (70.3%)	62 (83.8%)	Fisher's exact test *P*=0.175
	Myopia *n* (%)	12 (16.2%)	8 (10.8%)	4 (5.4%)	
	Hypermetropia *n* (%)	6 (8.1%)	4 (5.4%)	2 (2.7%)	
	Astigmatism *n* (%)	6 (8.1%)	10 (13.5%)	6 (8.1%)	

*Statistical test was used to compare the distribution of baseline characteristics among the three study groups, i.e., migraine, TTH, and normal population.
Table 1 shows no difference in distribution of baseline characteristics among the three study groups

Table 2: Comparison of IOP and CCT

Study Group	Migraine	TTH	Normal	Statistical test ANOVA
IOP Mean±SD (mmHg)	13.58±2.96	13.90±2.98	13.69±2.77	*P*=0.763
IOP in eyes without glaucoma Mean±SD (mmHg)	13.18±2.42	13.70±2.70	13.63±2.19	*P*=0.403
IOP in eyes with glaucoma Mean±SD (mmHg)	16.44±4.77	16.80±5.21	14.33±2.08	*P*=0.747
CCT Mean±SD (μm)	514.47±26.87	520.40±23.85	521.94±18.59	*P*=0.124
CCT in eyes without glaucoma Mean±SD (μm)	516.49±27.20	519.06±22.27	522.53±18.73	*P*=0.303
CCT in eyes with glaucoma Mean±SD (μm)	499.89±19.88	539±38.64	508±6.24	*P*=0.048

Table 2 shows no difference in IOP and CCT among the three study groups, i.e., migraine, TTH, and normal population

Discussion

In this hospital-based comparative cross-sectional study, we evaluated the prevalence rates of glaucoma among migraine patients and those without migraine, i.e. among TTH and normal group.

Migraine and other non-IOP dependent risk factors for glaucoma

While IOP is one of the most important risk factors for primary glaucoma, several pressure independent risk factors have been described. Studies have found that a significant increase in the risk of glaucoma is seen with old age, male gender, African race, myopia, hypertension, definite or borderline diabetes mellitus, Raynaud's phenomenon, migraine, cigarette smoking, and family history of glaucoma.[12,13] We also found that IOP was not associated with the presence of glaucoma, highlighting the presence of other etiopathogenic factors in the causation of glaucomatous changes in our study population.

Studies of the relationship between migraine and primary glaucoma have yielded conflicting results, which might be attributed to the differing study designs and ethnicity of study populations. Most previous studies included glaucoma patients and looked for a history of migraine either based on self-reports or previous medical records. These designs were prone to selection bias and recall bias, especially when self-reporting was used to elicit the presence of migraine.[5,8,9] A recent meta-analysis of similar studies reported that those studies using a nested case-control or cohort designs, which are epidemiologically stronger to provide evidence, failed to show any significant association between migraine and glaucoma.[14]

In our study, we compared the prevalence rates of glaucoma (POAG/ primary angle closure glaucoma (PACG)/ NTG, including glaucoma suspects) among migraine patients and nonmigraineurs, using two matched comparative groups, first being patients with another primary headache disorder, i.e. TTH and second including entirely normal subjects. We found no significant increased risk of glaucoma among migraine patients when compared to both comparison groups. Similarly, a large cohort study conducted in Taiwan using insurance claims data found that migraine did not affect the risk of POAG or PACG in their ethnic population. However, they found that when considering the joint effect of migraine along with comorbidities such as hypertension and hyperlipidemia, the risk of POAG was significantly greater in migraineurs.[7]

Migraine, glaucoma, and vasospasm: A probable link

In migraine, activation of trigeminovascular system and associated release of neuropeptides leads to vasospasm and dramatic changes in vessel caliber with consequent pain.[15] Therefore, during or prior to an acute attack of migraine, there is transient cerebral hypoperfusion, which though often limited to the posterior region of one hemisphere, may develop at other areas of the brain or even outside it like in the retina or choroid.[16]

The underlying pathophysiological mechanism of migraine acting as a risk factor for glaucoma is based on this recurrent vasospasm of cerebral, retrobulbar, and retinal vessels which induces ischemia of the optic nerve, retina, and choroid during acute attacks of migraine.[17] Though the vasospasm and hypoperfusion are transient, repetitive attacks have been shown to produce some permanent cerebral and retinal damage.[18]

This is highlighted by studies on the effect of migraine on ocular blood flow and posterior ocular structures. Retinal

Table 3: OCT parameters summary statistics: All eyes and eyes with glaucoma

All eyes OCT parameters	Migraine (*n*=74) Mean±SD (µm)	Tension-type headache (TTH) (*n*=74) Mean±SD (µm)	Normal (*n*=74) Mean±SD (µm)	Statistical test ANOVA (*P*)
RNFL inferior average	134.53±18.33	134.73±20.96	135.11±14.69	0.981
RNFL superior average	128.04±18.16	131.72±19.19	134.81±15.25	0.067
RNFL nasal average	90.49±19.19	97.78±16.93	103.39±15.59	<0.001
RNFL temporal average	70.58±16.13	79.19±15.37	81.18±11.75	<0.001
Average pRNFL thickness	103.59±12.82	108.90±12.44	109.11±8.99	0.006
Central macular thickness	214.73±20.14	224.5±17.57	227.08±14.19	<0.001
Eyes with glaucoma OCT parameters	**Migraine (*n*=9) Mean±SD (µm)**	**Tension-type headache (TTH) (*n*=5) Mean±SD (µm)**	**Normal (*n*=3) Mean±SD (µm)**	**Statistical test ANOVA (*P*)**
RNFL inferior average	135.78±25.58	125.00±13.32	132.00±10.15	0.425
RNFL superior average	121.44±22.83	132.00±11.00	120.67±11.59	0.582
RNFL nasal average	81.89±12.71	91.00±5.24	82.00±22.27	0.851
RNFL temporal average	71.22±8.15	72.80±9.44	74.00±13.86	0.111
Average pRNFL thickness	101.22±13.49	97.60±4.56	109.62±8.72	0.267
Central macular thickness	221.56±22.44	229.60±9.63	223.00±17.32	0.298

Table 3 shows comparison of OCT parameters among the three study groups, i.e., migraine, TTH, and normal population. When comparing all the eyes in each group, there is significant OCT thinning in migraine patients for nasal, temporal RNFL, average peripapillary RNFL, and central macular thickness. However, on comparing only eyes with glaucoma, there was no significant difference in OCT thickness.

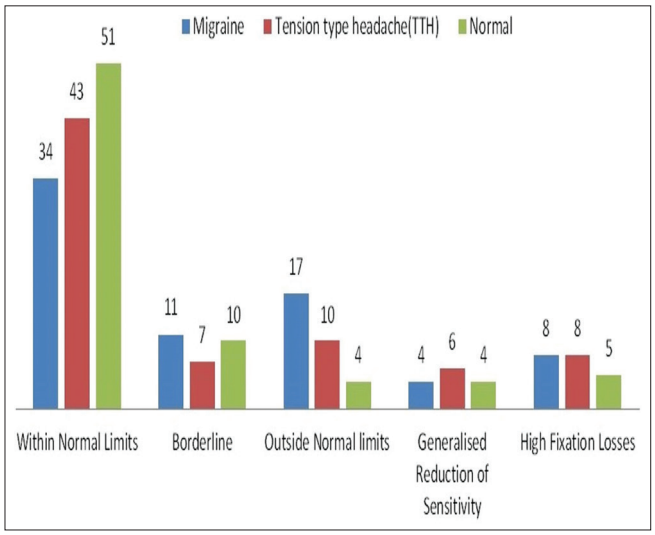

Figure 1: Bar diagram showing distribution of visual fields (no. of eyes) in the three groups— migraine, TTH, and normal. Number of patients with the type of field change in each group depicted along the corresponding bars

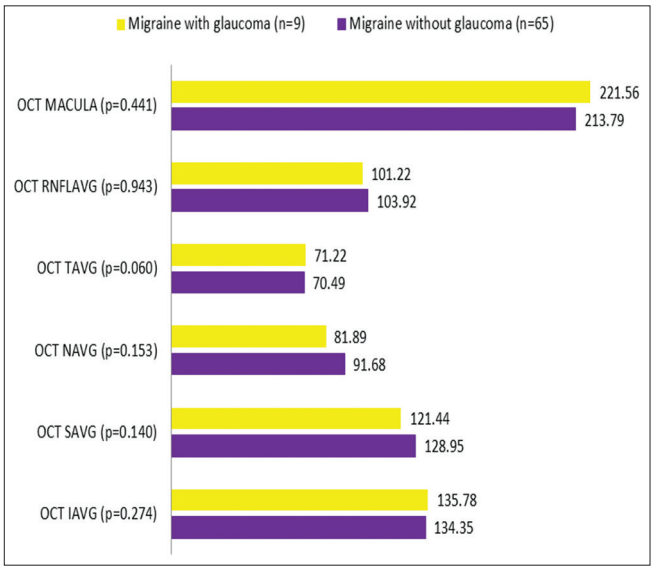

Figure 2: Bar diagram comparing OCT parameters (thickness in micrometers) within migraine group. In parenthesis, *P* values of independent *t*-test comparing the OCT parameters between migraine with and without glaucoma show no significant difference. Average values for each depicted along the corresponding bars

ischemia secondary to vascular occlusions has been reported in migraine patients.[19,20] Meanwhile other studies have used tools such as Doppler to show changes in ocular blood flow and vessel caliber during migraine attacks.[21] The functional outcome of compromised ocular blood flow in migraine patients has been elicited by visual field analysis showing glaucoma like field defects,[22] while scanning laser polarimetry[23] and OCT have demonstrated structural thinning of retina and choroid.[11,24]

OCT parameters in migraine

Using spectral-domain (SD) OCT, we found reduced RNFL thickness in both nasal and temporal quadrants, average RNFL thickness, and CMT among migraine patients irrespective of the presence of glaucoma [Fig. 3]. This corroborated well with past studies showing either reduction in sectoral RNFL[11,25-27] or average peripapillary RNFL[28,29] thickness among migraine patients. The possible reason for selective RNFL involvement can be explained by the understanding that migraine causes focal vasospasm in the retina just like in the brain with adjacent areas being well perfused[30] and also the difference in the vulnerability of RGC axons to ischemic insult.[17] This is also supported by finding localized perimetric changes in migraine.[22]

However, while our study found a significant reduction in CMT in migraine patients, previous studies have shown conflicting results.[6,24,26,31] Migraine causes alteration in choroidal blood flow and the resulting ischemia to outer retinal layers can lead to retinal pigment epithelial dysfunction and RGC drop out from the macula where they are most densely

concentrated, with macular thinning over time,[32,33] which can be picked up by SD-OCT as we have found.

It is now common clinical practice to use OCT in early glaucoma and to detect progression along with automated perimetry. In our study, we found that migraine has a similar thinning effect that is independent of the presence of glaucoma. Thus, a history of migraine headache is important for the ophthalmologist evaluating OCT of a glaucoma suspect patient.

Strengths

One of the strengths of our study was that we recruited patients diagnosed by the neurologist (as per IHS diagnostic criteria) with migraine or TTH and evaluated them for the presence of glaucoma. This resulted in the reduction of confounders due to misdiagnosis and also less recall bias than studies using a questionnaire or self-report to assess history of migraine. Another important strength is the study design using two comparison groups for migraine patients to reduce the Berksonian selection bias that may occur while selecting migraine patients using the convenience sampling technique from a hospital setting. Previous studies have separately looked for OCT thickness of retinal layers in migraine patients, but they had not correlated the same with the presence or absence of glaucoma like in ours, which makes our study unique.

Limitations

Though we took adequate measures to lessen confounders and bias, our study did have some limitations. First, a cohort study design to follow up migraine patients for development or progression of glaucoma would have been stronger to find any association, but was not undertaken due to limited study duration. Second, when measuring macular thickness using SD-OCT, we included all macular layers instead of

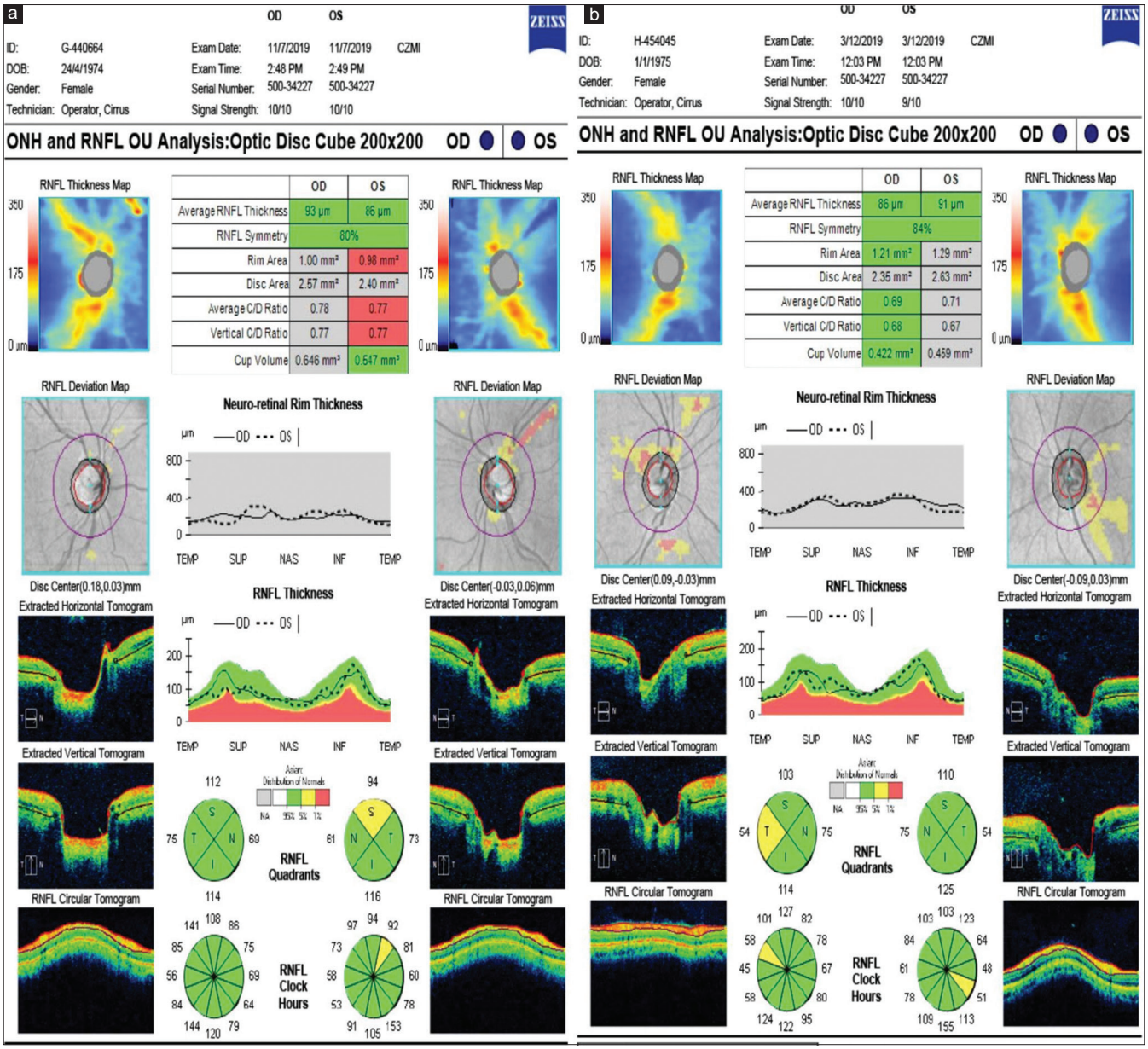

Figure 3: OCT RNFL images of two migraine patients both showing sectoral and generalized thinning. Patient A had glaucomatous cupping while patient B had healthy discs clinically

concentrating on ganglion cell layer analysis which would give a more accurate assessment of the effect of migraine on RGC dropout. Further studies on this using segmentation with SD-OCT may be carried out.

Directions for future

Newer technologies such as transcranial Doppler and OCT angiography can be used to study changes in cerebral and ocular blood flow and resultant structural changes in thickness and vasculature of select retinal layers and choroid in migraine patients thereby providing additional clues to confirm presence of a common vascular etiology in both migraine and glaucoma. Further OCT studies of migraine patients for monitoring treatment response, progression, etc., may be undertaken.

Conclusion

Migraine causes significant thinning of retinal layers (pRNFL and CMT) on OCT, which may be attributed to a common underlying vascular dysregulation mechanism similar to that postulated in the pathogenesis of NTG/POAG. Though this suggests a possibility of increased risk of developing glaucoma in migraine patients, it was not translated to an increased prevalence of glaucoma among migraine patients in this and several previous studies. From our study, we have seen that migraine causes thinning on OCT even in eyes without glaucoma. In clinical scenarios, our findings suggest the importance of a history of migraine while evaluating OCT of early glaucoma/suspect patients.

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Conflicts of interest

There are no conflicts of interest.

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