ORIGINAL ARTICLE

Visual Field Losses in Patients with Migraine without Aura and Tension-Type Headache

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ABSTRACT

The aim of the study was to compare the visual fields during pain attacks in the patients with migraine without aura and tension-type headache using automated perimetry. In this study 25 patients with migraine and 25 patients with tension-type headache were evaluated. The optic disc, macula and retina were assessed and patients with normal values were enrolled into the study. Intraocular pressure of all patients were measured. Furthermore, visual field test was applied to both groups using Humphrey field analyzer (Carl-Zeiss Meditec, model-745 i, Dublin, CA, USA). Both mean deviation and pattern standard deviation values of these two groups were not statistically significant.

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Introduction

Migraine is a common, multifactorial disorder characterised by recurrent attacks of typically unilateral, throbbing headache and autonomic nervous system dysfunction. A genetic vulnerability and exposure to environmental triggers can influence the migraine phenotype. Migraine is a highly prevalent disease with undetermined cause that is estimated to affect 18% of adult women and 6% of adult men of the Caucasian population.¹ The prevalence of women suffering from migraine has been attributed to a role of ovarian hormones.² Symptoms are assessed by symptom-based criteria defined by the International Headache Society (IHS) International Classification of Headache Disorders 3rd edition.³ Attacks usually start with warning symptoms (prodrome). Tiredness, euphoria, depression, irritation, food allergy, constipation, neck stiffness, and yawning may appear during the prodrome period.⁴

There may be some triggering factors for appearance of the migraine. These factors include menstrual cycle, some environmental factors such as weather conditions and heat change, psychological factors including stress in particular, sleep

disorders, frequent travel, contraceptive drugs, some foods, and alcohol.^{5,6}

Once it triggered migraine, spreading depression leads to pain and the other symptoms. Spreading depression is a progressive shutdown of cortical function and is associated with vascular changes. Vasoconstriction and then vasodilation follow the cortical spreading depression (CSD).⁷ CSD is the underlying mechanism of the migraine aura characterised by brief neuronal excitation, which is followed by a prolonged inhibition of neuronal activity. As the spreading oligoemia reaches sensory motor areas of the brain, the patient experiences the local neurological aura symptoms lasting up to an hour or sometimes for several days.⁸

CSD may also trigger the headache phase of migraine attacks by activating and sensitising the trigeminovascular system (TGVS). This system consists of the trigeminal nerve and nerve fibres that innervate the network of extra- and intracranial meningeal blood vessels and brain stem.^{9,10} Activation of the TGVS stimulates the release of vasoactive neurotransmitters from peripheral terminations of the trigeminal nerve that cause the vascular and inflammatory changes associated

with migraine pain. Visual symptoms are common in migraine either in the form aura or photophobia or less specific symptoms such as blur and accommodative dysfunction.¹¹

Tension-type headache (TTH) is the most common headache type. Prevalence of TTH varies between 11% and 93%. Infrequent episodic forms of TTH are more common in men, whereas frequent episodic and chronic forms are more common in women. TTH is an ill-defined heterogeneous syndrome, which is often diagnosed by the absence migrainous features. The reason for this is that we know very little about the pathology of TTH. It is believed that peripheral myofascial and central mechanisms result in dysregulation of the pain pathways.^{12,13} Hyperexcitable peripheral nociceptors are a likely aetiology of the peripheral mechanism of TTH. Physical triggers may include muscle strain during intense mental activity or poor posture.

The goal of this study was to compare the clinical features and visual fields of the patients with migraine and patients with TTH.

Materials and methods

Subjects

Patients who attended our neurology outpatient clinic complaining of headache and diagnosed as migraine or TTH were enrolled in the study. Twenty-five patients (20 female, 5 male) were diagnosed with migraine without aura and 25 patients (20 female, 5 male) diagnosed with TTH. Participants in the migraine group were aged between 20 and 55 years (36.48 ± 9.52), and TTH participants were aged from 18 to 62 years (37.7 ± 13.52). There was no significant difference in mean age between these groups ($p > 0.05$). All participants were required to have best-corrected visual acuity of 18/20 or better. The subjects wore their habitual refractive correction during the test.

This study was conducted in accordance with the tenets of the Declaration of Helsinki, and written informed consents were obtained from all of the subjects. The study protocol was reviewed and approved by the local institutional review board and ethics committee. Patients were diagnosed migraine or TTH according to the criteria of the IHS. Each patient

was interviewed by a neurologist who reviewed the patients' demographic and clinical information. Clinical data included type of migraine and TTH, triggering factors, accompanying symptoms, disease duration, attack frequency, attack duration, and headache severity. Migraine and TTH severities were assessed by visual analogue scale (VAS). All participants were in attack period at the time of testing. None of the patients have had medication overuse headache. Some of them used medication when they had headache since before or the medication has just been started. None of these medications were associated with the field losses. The exclusion criteria were pregnancy, previous ocular trauma, intraocular surgery, cataract, glaucoma, strabismus, laser treatment, neurological disorders, retinal or optic nerve disease, and diabetic or hypertensive retinopathy.

On attending the clinic all participants were asked to complete a short questionnaire detailing their symptoms and history, including questions on headache. Baseline questionnaire included these questions: migraine attack frequency (daily, weekly, monthly, every other month, less than 6 times a year), inhibition of daily activity (yes/no), nausea/vomiting (yes/no), sensitivity to light (yes/no), sensitivity to sound (yes/no), attack duration, and aggravation by routine physical activity. Age at migraine onset, severity, location (unilateral, behind one eye, front of head, back of head, whole head), character (pounding, pulsating, crushing, sharp, aching, burning), relation to menstruation, and migraine history were asked the patients in migraine-specific questionnaire.¹⁴

Ophthalmic examination

All subjects underwent a detailed ocular examination, including slit-lamp biomicroscopy, fundus examination with 90 D lens, and intraocular pressure (IOP) measuring with Goldman applanation tonometer. A normal optic disc was defined as having a cup/disc ratio less than or equal 0.4 and a neuroretinal rim with no glaucomatous changes.

Visual field

Swedish Interactive Threshold Algorithm (SITA) 24.2 Humphrey visual field test was performed on both eyes of the patients with Humphrey field

analyser. (Humphrey Instruments Inc. model 745 i; Carl-Zeiss Meditec, Dublin, CA, USA). Right and left eyes were analysed separately to determine whether unilateral or bilateral involvement was present. We excluded any pathology with bad reliability indices or patients who could not perform the test. SITA 24.2 gives an assessment of central 24 degrees of visual field. Mean deviation (MD) and pattern standard deviation (PSD) were evaluated for the statistical analysis. MD is an indication of any general depression across the visual field compared with the internal normative database of the perimeter. The index is based on data and attempts to remove any influence of highly abnormal values from its calculation. Negative values indicate worse than expected performance. PSD is an indication of any local abnormalities in an individual's visual field calculated by the instrument. Reliability indices are also reported so that any unreliable visual fields could be rejected. For SITA, an unreliable field was classified as one that had >20% false-positive errors or >20% false-negative errors.¹⁵

The global indices, MD, and PSD were recorded. MD and PSD in 50 eyes of 25 patients with migraine and 50 eyes of 25 patients with TTH were compared. Significantly lower general retinal sensitivity across the visual field and a high-incidence localised field defects were recorded. The deficits in the visual field were not correlated with the age or gender of the patients, nor with the severity, frequency, and duration.¹⁶

Statistical analysis

The MD and PSD values obtained from the visual fields of 50 eyes with migraine and 50 eyes with TTH were compared using Mann-Whitney *U* test. The data were analysed by SPSS version 20 (IBM, Armonk, NY, USA). Finally, the association of headache severity of the cases with the visual field losses was assessed.

Results

Mean age of the patients with migraine and TTH were similar ($p > 0.05$), and the majority of the

Table 1. Age and gender of the patients with migraine and tension-type headache.

Type	Age range	Mean age	Gender (F/M)
Migraine	20–55	36.48 ± 9.52	20/5
TTH	18–62	37.70 ± 13.52	20/5

Note. F = female; M = male; TTH = tension-type headache.

Table 2. Visual field defects in migraineurs and TTH patients.

	Migraine	TTH
VF defect	<i>n</i> (%)	<i>n</i> (%)
Normal	28 (56%)	32 (64%)
Constricted field	7 (14%)	5 (10%)
Peripheral island	4 (8 %)	7 (14%)
Total field defect	3 (6%)	—
Nasal step	3 (6%)	—
Superior altitudinal defect	1 (2 %)	1 (2%)
Inferior altitudinal defect	—	1 (2%)
Hemiscotosis	—	1 (2%)
Unilateral field defect	4 (8%)	3 (6%)

Note. VF = visual field; TTH = tension-type headache.

patients were women (Table 1). Visual field defects varied from a small peripheral scotoma to a complete field loss. These losses were detected in the fields of 22 eyes with migraine and 18 eyes with TTH. Scotomas were non-specific (Table 2). Visual field losses of a patient with migraine and a patient with TTH are shown in Figures 1 and 2, respectively.

Clinically, MD and PSD data were analysed by comparison with normative databases. We compared the MD and PSD in the migraine group with equivalent variables in the TTH group. Figures 3 and 4 show the frequency distributions for the MD and PSD for the SITA results of migraineurs and TTH patients. The distributions of both SITA variables appeared skewed, and these data were not normally distributed.

Figure 3 shows the frequency distributions for MD of migraine and TTH patients, whereas Figure 4 shows the frequency distributions for PSD of migraine and TTH patients. In the migraine group, MD was between −0.11 and −21.85 dB, whereas MD was between −0.05 and −18.07 dB in the TTH group. PSD was 1.04 to 16.64 dB and 1.04 to 11.43 dB in the migraine and TTH groups, respectively. When the eyes of the patients in both groups for MD and PSD were compared by Mann-Whitney *U* test, despite a slight elevation of MD and PSD levels in the migraine group, no significant difference was

Figure 1. Bilateral visual field of a patient with a migraine.

Figure 2. Bilateral visual field of a patient with tension-type headache.

found between the groups. In the migraine group, mean MD was -7.28 ± 6.20 dB; in the TTH group, mean MD was -5.53 ± 4.91 dB. Comparison of both groups revealed $p > 0.05$. In the migraine group, mean PSD was 4.65 ± 3.64 dB; in the

TTH group, mean PSD was 4.22 ± 2.74 dB. When both groups were compared, $p > 0.05$ was found. Consequently, there was not any significant difference between visual field losses of the migraine and TTH groups ($p > 0.05$) (Table 3).

Figure 3. Histogram of the median deviation (MD) for the Humphrey Swedish Interactive Threshold Algorithm (SITA) visual field test.

Figure 4. Histogram of the pattern standard deviation (PSD) for the Humphrey Swedish Interactive Threshold Algorithm (SITA) visual field test.

Table 3. Comparison of visual field losses of the patients with migraine and tension-type headache.

Type	n (eye)	Mean	Median	Min	Max	SD	Mann-Whitney <i>U</i> test
							<i>p</i>
MD migraine	50	-7.28	-5.53	-0.11	-21.85	6.20	0.162
MD tension-type	50	-5.53	-3.93	-0.05	-18.07	4.91	0.162
PSD migraine	50	4.65	3.29	1.04	16.64	3.64	0.997
PSD tension-type	50	4.22	3.55	1.04	11.43	2.74	0.997

Note. *n* = number; min = minimum; max = maximum; SD = standard deviation; MD = mean deviation; PSD = pattern standard deviation. $p > 0.05$ (Mann-Whitney *U* test).

Figure 5 is the scattergram of MD versus PSD with full threshold test, illustrating the distribution of visual field loss in the eyes of 25 migraineurs. Figure 6 illustrates the distribution of visual field loss in the eyes of TTH patients. There is a positive correlation between MD and PSD values in migraineurs. Additionally, there is a positive correlation between two variables of TTH patients.

Minimum and maximum IOPs were 9 and 21 mm Hg, respectively, and within normal limits. The mean IOP was 14.7 mm Hg for the migraine sufferers and 15.3 mm Hg for the TTH patients.

These values did not differ significantly in the two groups and neither did they differ between the eyes ($p > 0.05$).

There were not any patients between a severity of 0 and 2 for both migraine and TTH groups in accordance with the VAS determined by IHS. There were two patients with severity of 3 and 4. All remaining patients had a pain severity between 5 and 10 (Table 4). There was not any significant difference between the two patient groups according to the association of the attack severity with visual field defect ($p > 0.05$). Additionally, field

Figure 5. Scattergram of mean deviation (MD) versus pattern standard deviation (PSD) with the full threshold test, illustrating the distribution of visual field loss in 50 migrainous eyes.

Figure 6. Scattergram of mean deviation (MD) versus pattern standard deviation (PSD) with the full threshold test, illustrating the distribution of visual field loss in 50 tension-type headache eyes.

losses of the migraineurs and TTH patients were compared in terms of attack frequency and attack duration. Attack frequencies were assessed as everyday, several times a week, several times a month, and several times a year. There was no significant difference between the two patient groups based on the attack frequency and field deficit ($p > 0.05$). We also couldn't detect any significant difference according to the visual field loss and attack duration varying from a few minutes to 1 week ($p > 0.05$).

Discussion

We tried in this study to demonstrate that the TTH patients might have had visual field deficits like migraineurs. Thus, the main goal of the present

Table 4. Comparison of visual field losses of the patients with migraine and tension-type headache basing on the attack severity.

VF	Migraine		Tension-type headache	
	<i>n</i> (patient)	Severity of pain	<i>n</i> (patient)	Severity of pain
Normal VF	14	8.33	16	7.8
VF defect	11	6.77	9	7.4

Note. VF = visual field; *n* = number. $p > 0.05$.

study was to show a similarity between these two primary headache disorders. Additionally, the associations of visual field losses with the headache frequency, duration, and severity were analysed. Primary headache disorders of migraine and TTH are distinct diagnostic entities according to the classification systems. For each of the nosological classification to date, the differential diagnosis between migraine and TTH is entirely a function of differing self-reported symptom typologies. They presuppose that fundamentally different pathophysiologies underlie each of the primary disorder. In support of a continuum perspective, a growing number of studies have highlighted similarities between migraine and TTH in symptomatology, response to treatment, and pathophysiology. This study showed a new similarity between these headaches.

Modifications in the blood vessels and some cerebrovascular diseases causing microembolization as well as some hypoperfusion states may play a role in the pathophysiology of aura in migraine.¹⁷ The same mechanism plays a role in central retinal or cilio-retinal arterial obstructions that are observed in some migraine patients. Decrease in thickness is observed in peripapillary nerve fibre layer and choroid in migraine. This is explained by chronic ischaemic attacks caused by migraine. This is considered to be related with normal tension glaucoma (NTG) and ischaemic optic neuropathy (ION) in the patients with migraine.¹⁸ Migraine was shown to be significantly frequent in the patients with NTG and ION.^{19–21} Drance et al. reported that NTG patients with migraine had faster course of visual field deterioration than those without a history of migraine.²²

Any link between glaucoma and migraine needs to be carefully considered. In particular, migraine has been considered a risk factor for glaucomatous visual field progression. Glaucomatous visual field

defects have been found in patients with migraine in the absence of raised IOP, and this has led to the suggestion that there might be a relationship between the pathophysiology of normal tension glaucoma and migraine. In our study, IOPs of all patients were found to be within normal limits. Glaucomatous visual field defects were not observed in TTH patients, whereas a few migraineurs had some glaucomatous field losses such as nasal step.

Due to the connection between migraine and glaucoma, primary open-angle glaucoma should be excluded in migraine patients by measuring IOP. In our study, we measured IOPs of both eyes of a total of 50 patients. IOPs were found to be within normal limits. On the other hand, we observed optic disc carefully by using +90 D fundus lens. Hereby we tried to understand whether the patients had NTG or ION. None of the patients had optic disc abnormality. Thus, we eliminated the possibility that the visual field deficits were associated with glaucoma.

As the headache progress, it may be accompanied by a variety of autonomic symptoms (nausea, vomiting), affective symptoms (depression and irritability), cognitive symptoms (attention deficit, difficulty finding words, amnesia, and reduced ability to navigate in familiar environment), and sensory symptoms (photophobia, phonophobia, osmophobia, muscle tenderness). Recently, much attention has been given to the hypothalamus because it plays a key role in many aspects of human circadian rhythms (wake-sleep cycle, body temperature, food intake, and hormonal fluctuation).²³

The visual field loss was shown in migraine patients through the studies. Lewis and colleagues observed such a loss in 35% of the patients during attack period and prevalence increased by the patient age and period of the disease.²⁴ The ratios in our study were 44% for the patients with migraine and 36% for TTH patients. Majority of the defects were in shape of constricted field (14% in migraineurs, 10% in TTH patients) and peripheral island (8% in migraineurs, 14% in TTH patients). Total field deficit (3 patients, 6%) and nasal step (3 patients, 6%) were observed only in migraine patients. Lewis et al. found unilateral deficits in 14 of 21 affected individuals. However,

both our data and those of others would suggest that homonymous losses form a minority of cases. Unilateral field defects were obtained in 4 patients (8%) with migraine and in 3 TTH patients (6%) in the study. These defects were nearly same when the MD and PSD values were compared with each other.

McKendrick and colleagues showed increase in visual field losses when aura existed and attacks became frequent.²⁵ The visual field defects were shown to decrease and continue up to 75 days in the patients without aura.²⁶ If migraine field loss results from cumulative damage after repetitive attacks, it would be expected that older subjects should display more field loss. The exact nature of the field loss due to migraine warrants some consideration because it might help identify the locus of the defects. None of our subjects demonstrated visual dysfunction consistent with a cortical locus (bilateral homonymous deficits). Hence, our findings indicate that precortical visual dysfunction may be common in migraine sufferers.

The pathophysiology of chronic TTH is not well understood. Abnormal and sustained contraction of pericranial muscle was assumed to be the primary cause of pain; however, TTH sufferers do not reliably exhibit either abnormal resting levels of pericranial electromyography (EMG) activity or abnormal levels of EMG activity in response to stress. The most common trigger factors experienced by the patients were weather (82%), stress (66%), and menstruation (51%).²⁷ TTH, especially the chronic form, has a significant comorbidity and an indirect impact, which adds to the burden of disease. It is typically a bilateral headache with a pressing or band-like quality of low to moderate intensity.

We chose to evaluate the validity of our data using the intra-test reliability data. The reliability indices were good and showed that the results were of similar reliability in each group. Our results suggest that migraine sufferers are no more likely to have abnormal visual fields than TTH patients. We found no more inter-eye differences or inter-hemifield differences in migraine sufferers than in TTH group. As these non-significant results were found for SITA, our data do not support a magnocellular specific dysfunction in migraine.

In this study, we compared the mean deviations and pattern standard deviations of the patients with migraine and TTH. First, we excluded the fields had >20% false-positive and >20% false-negative errors and then evaluated MD and PSD of the two groups. MD of the migraineurs and TTH patients are mostly seen from -6 to 0 dB (Figure 2). PSD of 99 eyes with migraine and TTH are observed between 0 and 12 dB (Figure 3). We couldn't detect any significant difference between the two groups in terms of MD and PSD. We also couldn't find any relationship between severity of attacks, attack frequency and attack duration.

Ocular symptoms are a common, transient, initial component of migraine. Although permanent visual loss has been reported in a limited number of patients, detailed evaluations of the visual fields have not been conducted. In our study, all patients were in attack period and had at least 2-year history of migraine or TTH and no ocular problem by history or as determined by a vision screening examination consisting of acuity, IOP, and evaluation of the disc. Lewis et al.'s results for 60 migraine patients showed that 21 had some form of visual field abnormality. The prevalence of field loss was greater with increasing age and duration of disease. These results suggest that visual field losses from migraine may be more common than previously considered. This information also may be useful in elucidating the relationship between migraine and certain vascular conditions of the eye.

Multiple classification systems have been created for the diagnosis of headache disorders. According to these classification systems, primary headache disorders of migraine and TTH are distinct diagnostic entities. On the other hand, a number of studies have reported similarities between migraine and TTH in symptomatology and pathophysiology. Turner et al. showed in their taxometric analysis study that diagnostic/symptomatic distinctions between headache types would be less prominent among individuals with frequent headaches and those of younger age.²⁸ Migraine and TTH are differentiated by their characteristic symptoms and as such imply differing pathophysiologies and required treatments. Despite this categorical nosology, findings continue to emerge suggesting that migraine and TTH instead reflect dimensions of severity within the same headache construct.

In general, Turner et al. refute the notion that migraine and TTH represent discrete entities that differ by type rather than degree. As migraine chronification occurs, prototypical migraine features can overlap with TTH symptoms as the latter become more prominent. Many individuals meet criteria for both migraine and TTH. Among children or adolescents, 20–25% transition from migraine to TTH or from TTH to migraine within a matter of years, thus supporting the continuum theory of headaches in this subgroup of individuals. Individuals with migraine often mention TTH symptoms such as muscle tension and neck pain, and those with TTH often experience photophobia, phonophobia, and aggravation by activity. Migraine and TTH patients endorse similar attack triggers. Diagnostic distinction between these two common headache types is not readily apparent in patients with chronic headaches and of young age.²⁸

To our knowledge, this is the first study demonstrating that patients with TTH show some visual field deficits like migraineurs. In our study, field loss was 44% and 36% in migraineurs and TTH patients, respectively. Many studies were conducted about effect of the migraine on vision. A visual field loss in the migraine patients and increase of such a loss in proportion with the patient age, disease period and frequency of the attacks were shown. The present study indicates that field loss may also appear in the patients with TTH and such a loss is not less than migraine. We think that we could obtain more helpful information in larger series especially in terms of the relationship between attack frequency, attack duration, severity of attack, and visual fields in migraineurs and TTH patients.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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