Original Article

Multimodal Visual Functions and Cerebrovascular Reactivity in Migraine Patients between Attacks

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ABSTRACT

Background: Migraineurs had significantly interictal altered visual field and contrast sensitivity (CS) and it has long been thought that these field changes are related to vascular cortical origin. **Objective:** To investigate the relationship between visual functions and cerebral flow velocities and vasoreactivity in migraineurs between attacks. **Methods:** The study included 27 migraineurs (23 females and 4 males). Fourteen patients (51.9%) had migraine without aura (MO) and 13 (48.1%) had migraine with aura (MA). Patients are age and sex matched to 14 healthy volunteers. Subjects underwent migraine severity grading by migraine disability assessment score (MIDAS); transcranial Doppler sonography (TCD) with vasoreactivity testing for MCA and PCA; Pattern Reversal Visual Evoked Potential.(PRVEP); CS and visual field study using standard and short wave-length automated perimetry (SAP and SWAP). **Results:** Compared to controls, patients had significantly impaired response of MCA to HV (P=0.018) and a highly significant lower vasoreactivity index (P=0.0014), prolonged mean P100 latency, lower P100 amplitude (P<0.05) and lower CS (P< 0.01). SAP and SWAP detected field deficits in patients (63% and 74% respectively); bilateral minimal criteria of glaucomatous damage (37% and 40.7% respectively) and unilateral changes (26% and 33.3% respectively). There was significant negative correlation between VEP P100 latency, and mean MCA flow velocity, mean MCA flow velocity after HV and percentage of vasoreactivity. **Conclusion:** Migraineurs had significant interictal changes of visual field, contrast function, VEP and cerebrovascular reactivity suggesting that visual function deficits and migraine may share a common vascular etiology. **[Egypt J Neurol Psychiat Neurosurg. 2010; 47(4): 655-664]**

Key Words: migraine – vasoreactivity- VEP- visual field.

INTRODUCTION

Migraine is a complex neurovascular disorder, believed to affect nearly 12% of the world's population.[1] It is well established that the cerebral vasculature plays a key role in the pathophysiology of migraine.[2] Multifactorial etiology of migraine involves an abnormality in the cerebral vascular reactivity. It has been proposed that there is an uncoupling of the neuronal demand to hemodynamic activity in migraine patients, termed as neurovascular coupling disorder.[1]

Migraine has been linked to normal tension glaucoma since the report of Phelps and Corbett in 1985.[3] Theories may relate the pathophysiology of migraine and normal-tension variant of glaucoma on vascular basis. The symptoms of migrainous visual aura are consistent with cortical origin so that visual field deficits associated with migraine would be expected to be cortical.[4] There are also many reports revealing visual field defects that are unilateral and non-homonymous, suggesting that the disease has local pathologic influences as well.[5,6]

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The aim of the current study is to investigate for visual function deficits including visual field using standard and short-wavelength automated perimetry, spatial-contrast function, visual evoked potential as well as cerebral flow velocities and vasoreactivity in headache-free interval in migraineurs with and without aura.

SUBJECTS AND METHODS

Study Population

A cross-sectional case control study was conducted in Neurology and Ophthalmology departments, Ain Shams University hospital in the period from June 2009 to May 2010. The study included 41 subjects of whom 27 fulfilled the International Headache Society criteria for a diagnosis of migraine with or without aura[7], and 14 healthy volunteers matched for age and sex from the medical students, junior neurology and ophthalmology residents and hospital staff free of headache (established by clinical interview with the neurologist). The patients either had never been treated with any prophylactic drugs or had discontinued treatment at least one month prior to the testing. Migraine subjects did not experience any

attack one week prior to the day that they collaborated in the study. All patients had best corrected visual acuity of 6/6 and had a normal ocular health.

Exclusion criteria were; arterial hypertension, other brain diseases, pregnancy, metabolic and pulmonary diseases, migraine preventive treatment with beta-adrenoceptor blocking, calcium antagonists, epilepsy, daily chronic headache (more than 15 days/month) or a strictly unilateral headache. Informed consent was obtained from each participant after the nature of the study was explained.

Study Measurements

All subjects underwent detailed history taking and thorough neurological and ophthalmological examination; migraine severity assessment by the MIDAS (Migraine Disability Assessment Score) questionnaire[8] by the neurologist; Transcranial Doppler Sonography (TCD); pattern reversal visual evoked potential (PR-VEP); intraocular pressure (IOP) assessment; contrast sensitivity (CS); and visual field study using standard automated (SAP) and short wave-length automated perimetry (SWAP).

*Transcranial Doppler Sonography (*TCD) was performed using a pulsed Doppler device operating at 2 MHz (Multi-Dop DWL Electronische Systeme GmbH, Germany) by the same investigator. Insonation was done for the middle cerebral artery (MCA) and posterior cerebral artery (PCA). The MCA and PCA identification followed search techniques described previously by Aaslid[9]. MCA mean flow velocities (MFVs) were recorded at resting and after hyperventilation for 3 minutes. Cerebrovascular reactivity index was calculated as described in previous studies[10-13]. The mean flow velocities for both PCA were recorded at basal condition and after visual stimulation with checkerboard stimulation.

Visual Evoked Potentials (VEPs) were performed by checker board pattern reversal displayed on a TV monitor subtending $15^{o} \times 12^{o}$ at a viewing distance of 100 cm. The subjects were monitored while stimulation was done monocular. Two hundred individual trials were averaged and a repeated trial to verify reproducibility of the results was performed. The P100 latency and amplitude were recorded.

Assessment of visual field by standard (SAP) and short wave-length (SWAP) automated perimetry were performed using Oculus twinfield (Oculus Optikgerate GmbH, Wetzlar-Dutenhofen). For SAP, Goldmann size III white target projected on a 31.8 apostilb (asb) (10candelas per square meter [cd/m2]) white background. For SWAP, Goldmann size III blue target projected on a 31.8 (asb) yellow background. A visual field defect was defined according to previous studies.[14]

Contrast Sensitivity Function was assessed for far under both mesopic and photopic conditions using CVS-1000 E chart test (Vector vision Co., Greenville, Ohio, USA) at recommended test distance of 2.5 meters. The corresponding spatial frequencies are 3, 6, 12 and 18 cycles per degree (cpd).

Statistical Analysis

All results were collected with averaging the data from the right and left eyes for each participant. Data were analyzed statistically using SPSS for windows version13.0. Student's t-test was used to test the probability, one way ANOVA (Analysis of Variance) test to compare more than two groups as regards quantitative variables, and Pearson's correlation coefficient to test correlation between migraine characteristics and the results of regional cerebral blood flow, VEP, perimetry and contrast sensitivity. P-value $\leq$ 0.05 was considered as statistically significant.

RESULTS

The study included 27 patients diagnosed as Migraineurs. Twenty three were females (85.2%) and 4 (14.8%) were males. The control group included 14 subjects; 9 (64.3%) females and 5 (35.7%) males. Mean age of the patient group was 24.4 years (±5.67 SD) compared to 26.0 (±3.50 SD) in the control group. There was no statistically significant difference between the patients and controls as regards the mean age (P= 0.44) and sex distribution. (P= 0.73). Migraine patients are further subdivided into migraine without aura (MO) (no=14, 51.9%) and migraine with aura (MA) (no. =13, 48.1%) (Table 1).

The mean duration of headache was 6.94 years (±3.16 SD) in all migraine patients. MIDAS grading showed grade IV(indicating severe disability) in 15/27 patients (55.6%), grade III (moderate disability) in 7/27 patients (26%), grade II (mild disability) in 3/27 patients (11%) and grade I (infrequent or minimal disability) in 2/27 patients (7.4%).

Transcranial Doppler Results

Comparisons between the study and control groups as regards the basal MCA blood flow velocity (BFV), MCA BFV after Hyperventilation (HV), PCA BFV and PCA BFV after visual stimulation are shown in Table (2). There is significant impaired response of MCA to HV (P=0.018) and a highly significant lower percentage of vasoreactivity of MCA flow after HV in patients compared to control group (P=0.0014).

Comparison between the three groups using one-way ANOVA test show that there was a highly significant difference as regards the mean MCA BFV after HV in MA patients compared to MO and control group (P=0.014), and percentage of vasoreactivity after HV (the lowest being in MA patients) as compared to MO and the control group respectively (P=0.0004) (Table 3 and Figure 1).

As regards correlation of mean flow velocities and vasoreactivity to severity of headache, patients with moderate and severe headache (MIDAS III and IV) showed significantly impaired vasoreactivity compared to control subjects; P <0.05 (Figure 2).

Visual evoked potential results

Mean P100 amplitude of both eyes was significantly lower in patients compared to the controls (P=0.03). There was significantly prolonged mean P100 latency of both eyes in MO compared to the MA and control groups (P=0.04, Figure 3). Severity of headache did not affect VEP results (latencies and amplitudes, P > 0.05).

Ophthalmological Evaluation

No significant difference between right and left eye in the control and study groups regarding the refractive errors, the mean spherical equivalents as well as intraocular pressure (IOP) (P>0.05).

Spatial Contrast Sensitivity

Patients had significantly lower contrast at all spatial frequencies under both photopic and mesopic conditions compared to the controls (p< 0.01; Figure 4).

CSV was significantly lower at low special frequency 3.0 cpd under mesopic condition in MA compared to MO patients (p=0.04).

There was a significant negative correlation between CSV and severity of headache under both photopic and mesopic conditions at all spatial frequencies with more significance at low spatial frequencies (3.0 and 6.0 cpd) especially under mesopic condition (p< 0.01).

Visual field results

There was no significant difference between right and left eye in each group and between the control and study groups as regards the SAP global indices, which were also not affected by the severity of headache (according to MIDAS grading) (p>0.05; Figure 5). However, normal visual field was encountered in only 10/27 (37%) patients, while 10/27 (37%) patients had bilateral minimal criteria of glaucomatous damage and 7/27 (26%) patients had unilateral changes. So a total of 17/27 (63%) patients (9 MA, 8 MO) had visual field deficits.

There was no significant difference between right and left eye in each group and between the control and study groups as regards the SWAP global indices except mean short-term fluctuation (SF) which was higher in the right eye of the patients compared to the controls (p=0.007; Figure 5).

Also SWAP short –term fluctuation (SF) eyes of patients with MIDAS grade IV (severe headache) was 3.78± 1.51 which was significantly higher than the control group (2.48±0.9) with p=0.017 (Figure 5).

Regarding SWAP, it was found that; normal visual field was found in only 7/27 (26%) patients. Twenty patients (74%) had minimal criteria of glaucomatous damage; 11 (40.7%) bilateral and 9 (33.3%) unilateral changes; (ten MA and ten MO).

Comparison was done between patients with minimal criteria for glaucomatous damage and the control group as regards mean visual field global indices (Table 4). There was a highly statistically significant difference between them regarding SWAP (mean MD, PSD and SF) and SAP (mean PSD).

There was a highly significant negative correlation between SWAP (PSD, and CPSD) and contrast sensitivity under both mesopic and photopic condition at all spatial frequencies (p<0.05).

There was significant negative correlation between VEP P100 latency, and mean MCA flow velocity, mean MCA flow velocity after HV and percentage of vasoreactivity (r =-0.39, p=0.04; r =-0.51, p=0.006; r=-0.28, p=0.01) respectively. However, this was not the case regarding the correlation between them and VEP- P100 amplitude. There was positive correlation between cerebral BFV and reactivity index and CS; however this was not statistically significant.

Table 1. Age and sex distribution in the study subgroups of migraine patients.

Variable	Migraine without aura n=14/27 (51.9%)	Migraine with aura n =13/27 (48.1%)	p-value
Mean age±SD	25.0 ± 6.5	23.6 ± 4.8	
Range (years)	17.0 - 43.0	18.0 - 32.0	0.82
Number of Females (%)	12/27 (44.5%)	11/27 (40.7%)	
Number of Males (%)	2/27(7.4%)	2/27 (7.4%)	0.81

No significant difference between MA and MO patients as regards mean age and sex (p >0.05)

Table 2. Transcranial Doppler data in migraine patients and control groups.

	Migraine patients (N=27)		Control group (N=14)		Student t- test P value
	Mean±SD	Range	Mean±SD	Range	
Mean MCA flow velocity (cm/sec)	75.66±11.89	50.0-106.0	75.64±8.85	67.0- 100.0	0.99
Mean MCA flow after hyperventilation (HV) (cm/sec)	56.74±12.66	39.0-96.0	49.21±6.9	67.0-100.0	0.018
% of vasoreactivity	25.9±10.89	2.0-45.2	34.90±5.78	23.75-42.35	0.0014
Mean PCA flow (cm/sec)	36.26±6.14	24.0-49.0	34.90±5.78	20.0-45.0	0.347
Mean PCA flow after visual stimulation (cm/sec)	41.07±7.66	26.0-57.0	42.57±7.79	23.0-52.0	0.56

Significantly impaired MCA flow response to hyperventilation (P=0.018) and highly significant reduction (P=0.0014) in vasoreactivity percentage of MCA flow velocity in migraine group compared to control group.

Table 3. Comparison between migraine with and without aura and control groups as regards the TCD data.

TCD data	Migraine without aura group (MO)		Migraine with aura group (MA)		Control group		P-value
	Mean±SD	Range	Mean±SD	Range	Mean±SD	Range	
Mean MCA flow velocity (cm/sec)	75.07±11.45	61.0-93.0	76.27±13.34	50.0-106.0	75.64±8.85	67.0-100.0	0.97
Mean MCA flow after hyperventilation (HV) (cm/sec)	52.07±10.63	39.0-69.0	59.9± 8.15	49.0 -71.0	49.21±6.9	41.0-62.0	0.014
% of vasoreactivity	30.68±8.74	16.92-45.2	20.25±10.55	2.0-35.36	34.90±5.78	23.75-42.35	0.00043
Mean PCA flow (cm/sec)	43.21±6.73	24.0-49.0	38.18±5.07	29.0-48.0	43.21±6.65	20.0-45.0	0.22
Mean PCA flow after visual stimulation (cm/sec)	38.79±8.55	26.0 - 57.0	43.63±6.45	30.0 -53.0	42.57±7.79	23.0-52.0	0.26

One-way ANOVA test show a significantly lower MCA flow velocity after HV (P=0.014) and a highly significant reduction in MCA reactivity (P=0.00043) in MA compared to MO and control group.

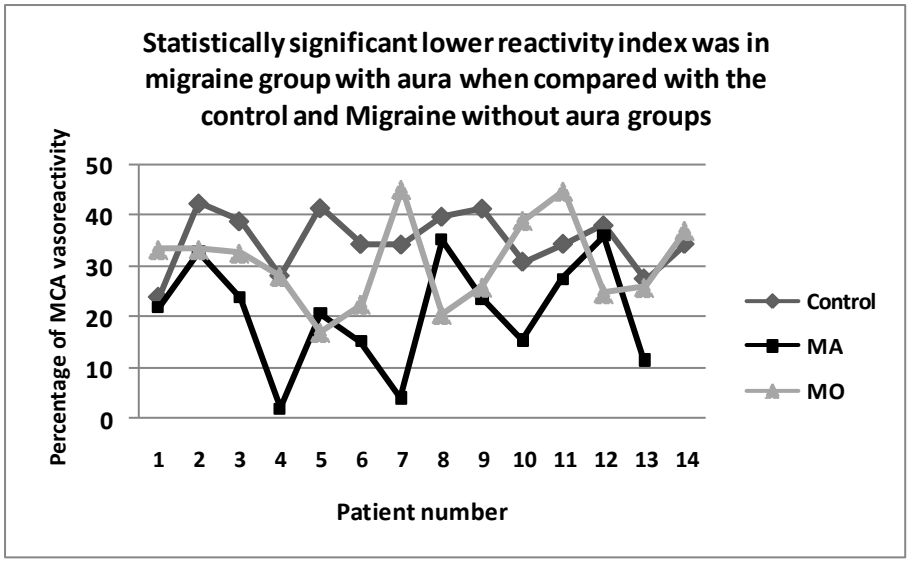

Figure 1. Comparison between the control group, migraine with and without aura groups as regards percentage of vasoreactivity of MCA flow after hyperventilation.

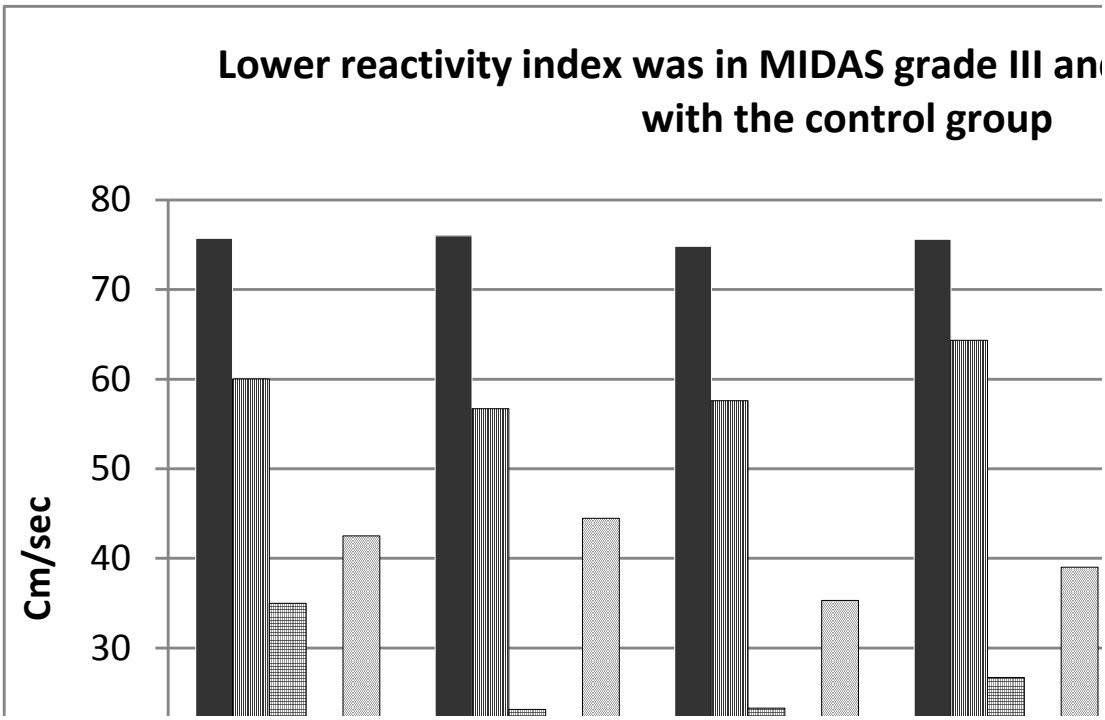

Figure 2. Correlation between TCD findings and severity of headache among migraine patients.

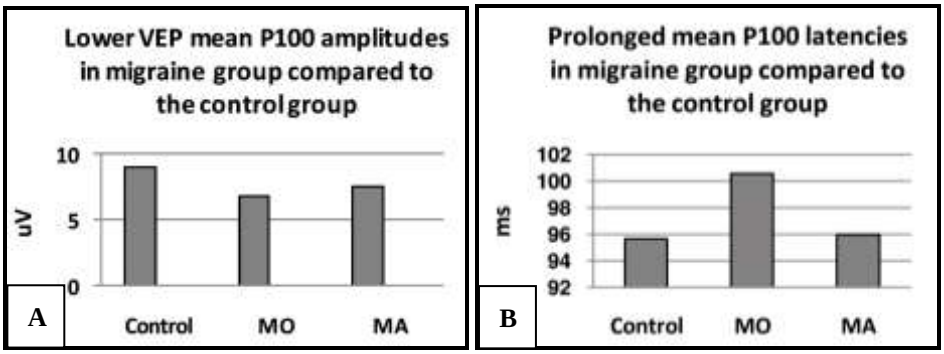

Figure 3. Comparison between migraine group and control group regarding VEP mean P100 amplitude (a) and latency (b).

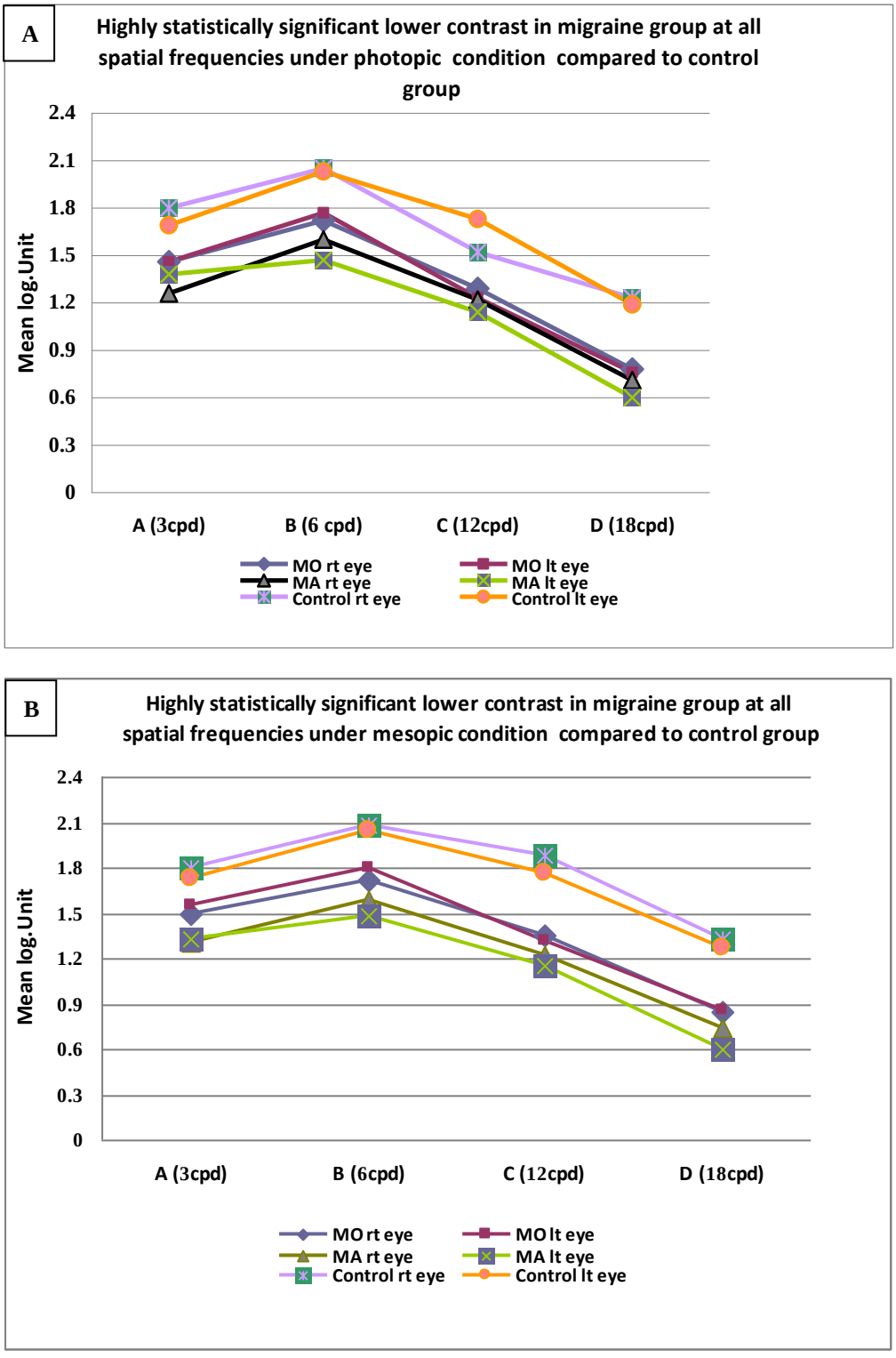

Figure 4. Comparison between the migraine patinets' groups and control groups as regards mean CSV (contrast sensitivity values) under photopic conditions (A) and mesopic conditions (B).

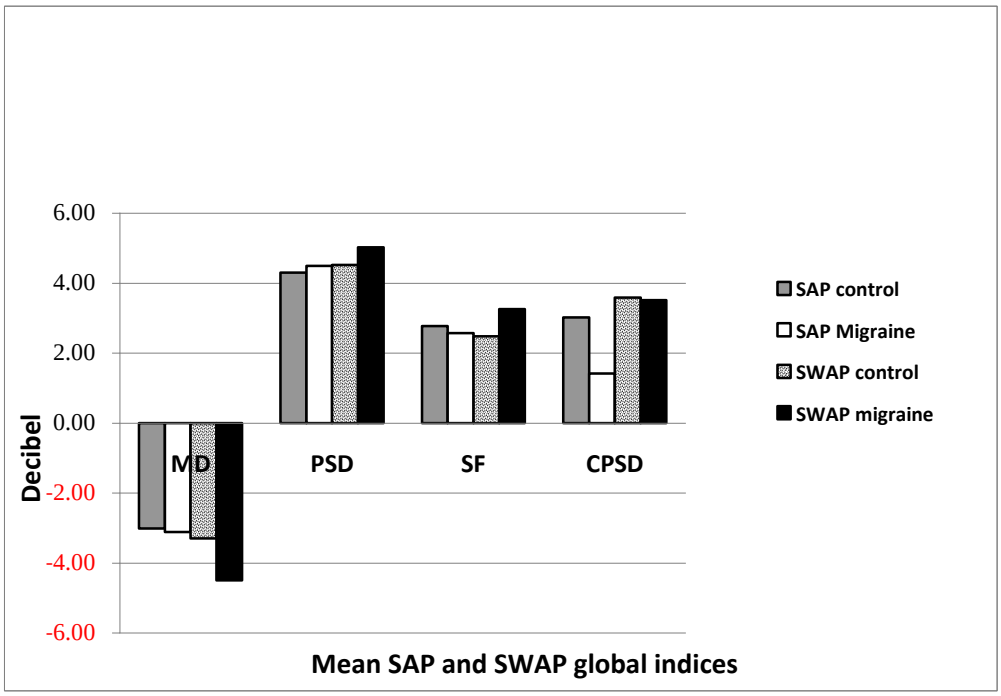

Figure 5. Comparison between the migraine and control group as regards the SAP and SWAP global indices.

Table 4. Mean SAP and SWAP global indices in patients with minimal criteria for glaucomatous damage as compared to control group.

SAP	Mean ± SD			
	MD	PSD	SF	CPSD
Rt eye	-4.69±3.3 (-1.37 to-13.24)	6.7±1.02 (4.17-8.05)	3.2±0.89 (1.94-4.51)	4.47±1.45 (2.1-7.8)
Lt eye	-4.38±3.9 (-0.04to-11.6)	5.48±1.26 (4.17-7.4)	3.21±1.5 (1.32±6.38)	4.09±1.6 (0.09-6.43)
RT+LT	-6.87±4.0 (-0.04 to-13.24)	5.6 ±1.1 (4.17-8.05)	3.2±1.15 (1.32-6.38)	4.32±1.5 (1.09-7.8)
p-value	0.24	0.047	0.25	0.059
SWAP				
Rt eye	-6.67±4.12 (-0.49 to -14.73)	6.3±1.53 (3.52-8.81)	4.34±1.89 (1.34-7.0)	3.98±2.35 (0.0-8.1)
Lt eye	-5.82±4.23 (-0.41to-14.19)	4.26±1.53 (2.42-8.5)	3.73±1.7 (2.02-8.0)	4.49±2.08 (0.0-7.3)
RT +LT	-6.87±4.0 (-0.49 to-14.73)	6.4±1.5 (3.52-8.81)	4.2±1.83 (1.34-8.0)	3.43±1.58 (0.0-8.1)
p-value	0.0032	0.0029	0.0023	0.28

There was a highly significant difference regarding SWAP mean (MD, P=0.0032, PSD, P=0.0029 and SF,P=0.0023) and SAP mean (PSD, P=0.047) compared to control group.

DISCUSSION

Migraine is a common neurological condition affecting 12% of adults.[1,15] As the visual pathways are involved in the pathophysiology of migraine, it is important to investigate the visual functions in involved patients.

Migraineurs had significantly interictal altered visual field and contrast function at all spatial frequencies to the normal population and it has long been thought that these field changes are of cortical origin. However, these defects share some features with early stages of glaucoma and may release a possibility for a common vascular disease pathogenesis in these two conditions.[16-18]

In this study, we investigated interictal cerebral blood flow and vasoreactivity, abnormalities in visual pathway using PR-VEP together with contrast sensitivity and visual field testing using SAP and SWAP in migraineurs with and without aura. Our data revealed interictal mean cerebral blood flow velocities comparable to control subjects, a finding previously described in another Egyptian study by Talaat et al.[19]. However, the more important is the significant reduced cerebral vasoreactivity in MA patients, which is suggested to reflect inter-attack persisting vasomotor changes which are of pathophysiological interest and may be used as a monitoring tool under prophylactic medication.[20] The positive correlation between severity of headache and the impaired vasoreactivity may raise the point that the severity of migraine may reflect diffuse vascular affection which may influence the cerebral vasomotor changes. In our study, neither basal PCA mean flow nor flow after visual stimulation showed intergroup difference, a point that may raise the question: is visual stimulation alone is sufficient for testing the vascular reserve or should we shift to other tests. However, absence of PCA abnormalities may explain the visual abnormalities on basis other than being cortical.

PR-VEP findings revealed statistically significant lower P100 amplitude in patient compared to controls and prolonged mean P100 latency in MO compared to MA and controls. The basis for interictal VEP changes is unclear. Oelkers and his colleagues[21] suggested that VEP abnormalities reflect dysfunction of magnocellular pathway. Also, Kennard et al.[22] suggested that they might have a structural basis, due to ischemic damage during repeated attacks. Correlation between VEP latency and MCA velocity and vasoreactivity, is evident in this study and augments the underlying vascular role of retinocortical pathways deficits detected by PR-VEP. The degree of ischemia could certainly account for transient neurological deficits and possibly persistent deficits if the damage was repeated. However, if relative cerebral ischemia during a migraine aura was considered to be the only cause, the abnormalities should be confined to MA, whereas our findings were in both MA and MO patients. Another possible cause of ischemia would be recurrent ergotamine intake, which produce results in both MA and MO, thus, both the aura and the vasoconstrictive action of the drug is postulated. However, in this study we did not investigate the effect of chronic ergotamine intake on the variables of the study.

This study demonstrated that contrast processing is not normal in subjects with migraine when tested at times between episodes. These lower contrast defects neither correlate to duration of migraine nor presence or absence of visual aura. However, it significantly correlated with severity of the attacks. CSVs were significantly lower in migraine patients with and without aura, more significantly reduced under mesopic conditions and at low spatial frequencies in migraine with aura. This is suggestive of both selective parvocellular (P) and magnocellular (M) pathways dysfunctions with more significant affection of M pathways. This pathway is similarly affected in glaucoma. Other studies confirmed dysfunction of the M and P pathways by using various test stimuli.[23-25] In our study there was strong correlation between headache characteristics and contrast sensitivity deficits. The possibility for some precortical sites of pathophysiological actions in migraine was detected by interocular difference as described by Benedek and his colleagues.[26]

Although, previous reports suggested similar structural basis (magnocellular pathways), and proposed underlying vascular basis for interictal PR-VEP[23] and visual filed deficits using SWAP[25], this study demonstrated discrepancy between the two visual functions. VEP significantly correlated to headache duration; and SWAP correlated to severity of headache. This may explain absence of correlation between PR-VEP and SWAP.

Interestingly, although we all expect the visual field changes in migraine cases to be cortical, SAP and SWAP demonstrated bilateral minimal criteria of glaucomatous damage and unilateral deficits, with no homonymous defects which indicates a cortical origin. So detected field deficits are of non-cortical origin. Comparable to Yenice and his colleagues[25], we detected early glaucomatous like field defects in 74% of cases. Bilateral field affection may reflect rather global or systemic ischemia involving both sides rather than a strict unilateral process that affect unilateral structures only. A considerable proportion of unilateral field losses (26%) suggest a precortical locus in at least some individuals, which appears contradictory to the cortical locus often considered to mediate the appearance of an aura.

The present study highlights the interictal visual functions deficits and demonstrates the correlation between VEP deficits and migraine durations, interictal MCA mean velocity and vasoreactivity changes, deficits are apparent in both MO and MA, significant correlation between vasoreactivity and VEP changes suggesting the vascular mechanism, detected visual field defects, and apparent glaucomatous field defects.

A challenge remains in determining a relationship between glaucoma and migraine.[27,28] If these deficits in visual-related tests are shown to be progressive and persistent and relate with glaucomatous neuropathy, a different issue will be discussed in the near future, like the use of antiglaucomatous drugs in migraine patients or migraine medications in normal tension glaucoma patients. Whether migraine populations have a high risk for glaucoma is still unknown and needs further analyses with prospective studies. Then headache patients with a definitive diagnosis of migraine might be in need for visual field testing to detect early glaucomatous defects.

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REFERENCES

1. Akin A, Bilensoy D, Emir UE, Gulsoy M, Candansayar S, Bolay H. Cerebrovascular dynamics in patients with migraine: near-infrared spectroscopy study. Neuroscience letters. 2006 May 29; 400(1-2): 86-91.
2. Rosengarten B, Sperner J, Gorgen-Pauly U, Kaps M. Cerebrovascular reactivity in adolescents with migraine and tension-type headache during headache-free interval and attack. Headache. 2003 May; 43(5): 458-63.
3. Phelps CD, Corbett JJ. Migraine and low-tension glaucoma. A case-control study. Investigative ophthalmology & visual science. 1985 Aug; 26(8): 1105-8.
4. Yenice O, Onal S, Incili B, Temel A, Afsar N, Tanridag T. Assessment of spatial-contrast function and short-wavelength sensitivity deficits in patients with migraine. Eye (London, England). 2007 Feb; 21(2): 218-23.
5. Drummond PD, Anderson M. Visual field loss after attacks of migraine with aura. Cephalalgia. 1992 Dec; 12(6): 349-52.
6. McKendrick AM, Badcock DR. Decreased visual field sensitivity measured 1 day, then 1 week, after migraine. Investigative ophthalmology & visual science. 2004 Apr;45(4):1061-70.
7. Society IC. The International Classification of Headache Disorders: 2nd edition. Cephalalgia 2004. pp. 9-160.
8. Stewart WF, Lipton RB, Dowson AJ, Sawyer J. Development and testing of the Migraine Disability Assessment (MIDAS) Questionnaire to assess headache-related disability. Neurology. 2001; 56(6 Suppl 1): S20-8.
9. Aaslid R, Markwalder TM, Nornes H. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. Journal of neurosurgery. 1982 Dec; 57(6): 769-74.
10. Taha AM, Abdulghani MO, Gadallah H, Sobhi S. cerebrovascular hemodynamics in chronic liver disease: a Transcranial Doppler study. Egypt j neurol psychiat Neurosurg. 1995 (32): 99-107.
11. Abdulghani MO, Allosh TK, Raafat M, Zakaria M. transcranial Doppler in clinical practice; applications and prospects. Egypt J Neurol Psychiat Neurosurg 1994; 31: 19-26.
12. Abdulghani MO, Raafat M, Emam A, Allosh TK, Abdelrahman A. The impact of cervical spondylosis on vertebrobasilar blood flow: a Transcranial Doppler study. Egypt J Neurol Psychiat Neurosurg. 1995; 32: 75-81.
13. Pfefferkorn T, von Stuckrad-Barre S, Herzog J, Gasser T, Hamann GF, Dichgans M. Reduced cerebrovascular CO(2) reactivity in CADASIL: A transcranial Doppler sonography study. Stroke; a journal of cerebral circulation. 2001 Jan; 32(1): 17-21.
14. Budenz DL. Atlas of visual fields. Philadelphia, New York.: Lippincott Raven Publishers; 1997. pp. 143-94.
15. Stewart WF, Shechter A, Rasmussen BK. Migraine prevalence. A review of population-based studies. Neurology. 1994 Jun; 44(6 Suppl 4): S17-23.
16. McKendrick AM, Vingrys AJ, Badcock DR, Heywood JT. Visual field losses in subjects with migraine headaches. Investigative ophthalmology & visual science. 2000 Apr; 41(5): 1239-47.
17. McKendrick AM, Vingrys AJ, Badcock DR, Heywood JT. Visual dysfunction between migraine events. Investigative ophthalmology & visual science. 2001 Mar; 42(3): 626-33.
18. McKendrick AM, Cioffi GA, Johnson CA. Short-wavelength sensitivity deficits in patients with migraine. Archives of ophthalmology. 2002 Feb; 120(2): 154-61.
19. Talaat FM, Alboraey MF, Hamdy MM, Sakr MA-A. Transcranial Doppler study in patients with migraine. Egypt J Neurol Psychiat Neurosurg. 2004; 41(2): 479-87

20. Wolf ME, Jager T, Bazner H, Hennerici M. Changes in functional vasomotor reactivity in migraine with aura. Cephalalgia. 2009 Nov; 29(11): 1156-64.
21. Oelkers R, Grosser K, Lang E, Geisslinger G, Kobal G, Brune K, et al. Visual evoked potentials in migraine patients: alterations depend on pattern spatial frequency. Brain. 1999 Jun; 122 (Pt 6): 1147-55.
22. Kennard C, Gawel M, Rudolph Nde M, Rose FC. Visual evoked potentials in migraine subjects. Research and clinical studies in headache. 1978; 6: 73-80.
23. McKendrick AM, Badcock DR. Contrast-processing dysfunction in both magnocellular and parvocellular pathways in migraineurs with or without aura. Investigative ophthalmology & visual science. 2003 Jan; 44(1): 442-8.
24. McKendrick AM, Sampson GP. Low spatial frequency contrast sensitivity deficits in migraine are not visual pathway selective. Cephalalgia. 2009 May; 29(5): 539-49.
25. Yenice O, Temel A, Incili B, Tuncer N. Short-wavelength automated perimetry in patients with migraine. Graefe's archive for clinical and experimental ophthalmology = Albrecht von Graefes Archiv fur klinische und experimentelle Ophthalmologie. 2006 May; 244(5): 589-95.
26. Benedek K, Tajti J, Janaky M, Vecsei L, Benedek G. Spatial contrast sensitivity of migraine patients without aura. Cephalalgia. 2002 Mar; 22(2): 142-5.
27. Cole KL, Wang EE, Aronwald RM. Bilateral Acute Angle-closure Glaucoma in a Migraine Patient Receiving Topiramate: A Case Report. The Journal of emergency medicine. 2009 Dec 10.
28. Harle DE, Evans BJ. Perimetry and migraine--deficits may not implicate glaucoma. Graefe's archive for clinical and experimental ophthalmology = Albrecht von Graefes Archiv fur klinische und experimentelle Ophthalmologie. 2006 Oct; 244(10): 1377-8; author reply 9-80.

الملخص العربي

الوظائف البصرية المختلفة و تفاعلية الأوعية الدماغية لدى مرضى الشقيقة فى الفترة ما بين نوبات الصداع

يعتبر مرض الشقيقة اعتلالا عصبيا وعائيا, حيث يشمل اضطراب تفاعلية الأوعية الدماغية, كما يختص بوجود تغيرات فى الوظائف البصرية المختلفة فى الفترة ما بين نوبات الصداع.

لذلك كان الهدف من الدراسة هو دراسة الوظائف البصرية المختلفة لدى مرضى الصداع النصفى عن طريق فحص مجال الإبصار باستخدام أهداف ضوئية ذات ضوء موجى مختلف والموجات المستفزة للعصب البصرى و قياس حساسية التباين تحت اضاءات مختلفة وربط ذلك بقياس سرعة تدفق الدم ورد فعل الأوعية الدموية الدماغية لهؤلاء المرضى ما بين النوبات.

شملت الدراسة على ٢٧ مريضا يعانون من الشقيقة و١٤ شخصا لا يعانون من أمراض بالعين ولا أمراض تؤدى إلى قصور بالدورة الدموية ويمثلون العينة الضابطة. تم دراستهم باستخدام الموجات المستفزة والدوبلر عبر الدماغ و مجال الإبصار و مقياس حساسية التباين.

وقد أثبتت الدراسة وجود قصور فى معدل تدفق الدم فى الأوعية الدموية الدماغية و تفاعلية الأوعية الدماغية لعديد من التغيرات. وأيضا وجود تغييرات بالموجات المستفزة للعصب البصرى ونقص حساسية التباين وتغييرات فى مجال الإبصار فى بعض المرضى لا ترجع إلى اصل دماغى مركزى. وهذه التغييرات فى قياس حساسية التباين ومجال الإبصار يشابه إلى حد كبير التغييرات التى توجد فى مرضى المياه الزرقاء ذات الضغط الغير مرتفع. ويبقى التساؤل عن وجود علاقة بين المرضين وإمكانية ثبات أو تدهور هذا التغييرات فى الوظائف البصرية المتعددة وإمكانية متابعة مرضى الشقيقة بأحدث الوسائل والاختبارات للكشف المبكر عن هذه العلاقة.