

Electroencephalogram and Visual Evoked Potential Changes in Patients with Primary Headaches

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Abstract

This study was a case/control study in which a total of (450) subjects, into (150) with primary headache (male and female) and this was included in this study, 128 with migraine headache, 22 with non-migraine headache and 300 were normal, as control group. The electrophysiological tests were done at the neurophysiology unit of Mirjan Teaching hospital in Babylon City, during the period from 28 / October/ 2014 until 22 / May/2015. This study reveals that involvement of the migraine are more than that of the non-migraine in headache patients when compared with control group and when compared between the migraine and non-migraine headache patients. The most common EEG changes in headaches patients were diffuse slowing and spike wave of back ground activity, in such a way that 54 (42.2%) migraine patients had abnormal EEG changes, in comparison to 4 (18.2%) of non-migraine patients. Visual evoked potential (VEP) was dependable marker for central nervous system affection in primary headache (migraine and non-migraine) and the most common abnormalities were prolonged latency and amplitude is stay in normal ranged (5-10) μ .volt.

Keywords: EEG, VEP, migraine headache, non-migraine headache.

Introduction

Headache is defined as a pain arising from the head or upper neck of the body. The pain originates from the tissues and structures that surround the brain because the brain itself has no nerves that give rise to the sensation of pain (pain fibers) ¹. Headaches can radiate across the head from a central point or have a pinching vise-like quality. They can be sharp, throbbing or dull, appear gradually or suddenly and last for multiple days or less than an hour ².

All headaches are considered primary headaches or secondary headaches. Primary headaches are not associated with other diseases. Examples of primary headaches are migraine headaches Typically the headache affects one half of the head, is pulsating in nature, and lasts from 2 to 72 hours. Associated symptoms may include nausea, vomiting, and sensitivity to light, sound, or smell. The pain is generally made

worse by physical activity ³.

Migraines are believed to be due to a mixture of environmental and genetic factors. About two-thirds of cases run in families. Changing hormone levels may also play a role, as migraines affect slightly more boys than girls before puberty, but about two to three times more women than men ^(4,5). The risk of migraines usually decreases during pregnancy .

Subjects And Methods:

This study was carried out through the period from October 2014 to May 2015 , In the brain alone planning and optic nerve, as well as in consultation nervous in Mirjan Hospital in the city of Babylon. By using the history , as a cross section observational study, we select (150) patients with primary headache ,in addition to (300) healthy control subjects. underwent EEG and VEP study with age and sex matched healthy control group

should be selected for this study . all of them met the criteria of episodic headache (migraine with, without aura & non migraine) according to the international classification of headache disorder.

Subjects:

Two groups of subjects were studied:

The control group:

This group comprised thirty hundred healthy volunteers , (135 males and 165 females) ranging in age from (9 to 67) years , with a mean age of (30.40±7.38) years.

The patients group:

One hundred and fifty headache patients were selected all types of primary headache in Mirjan Teaching Hospital, they were (43 males and 107 females) ranging in age from (7 to 69) years with a mean age of (31.23±12.38) year.

Methods:

All the subjects were approved for:

- Electrophysiological tests:
- Visual evoked potential (VEP) .
- Electroencephalography (EEG) .

Visual Evoked Potentials (VEPs) Study:

Visual Evoked Potential test was carried out in a dark, quite room, with the subjects sitting comfortably on a chair and advised not to move or blink continuously during the test in order to decrease muscle contraction artifacts from eyes and skeletal muscles which blur the evoked potential waves, thus it is of paramount importance to avoid such artifact.

Instrumentation:

Using in this study Evoked Potential machine, serial no.GH 17 H9NW315431B, model 171S, (Italy) was used for electrophysiological analysis of the VEP using the VEP program. The VEP system include four channel preamplifiers which are connected to plasma screen as a photo-stimulator source.

Procedure:

Recording Visual Evoked Potentials:

The subject is call him to sit on the chair while made recording and told him to see in same level of the red point sited in the center of the screen, and the space between his state and the screen is fixed at rate equal to 100 cm.

The patient should asked him to cleaning and lesser his hair then asked him to sleep on the bed then begin applying gel on the surface of electrodes (to made skin impedance slighter) on the subsequent positions : on the right side from the beside of the head and about five centimeters from frontal side placed the three electrodes (F7,T3,T5) ,in the anther side(left side) placed the three other electrodes (F8,T4,T6) ,and then placed the five electrodes from the right side also, in frontal side (Fp1,F3,C3,P3,O1) ,and then placed the five electrodes from left frontal side (Fp2,F4,C4,P4,O2) .At the end all of these electrodes were connected to the referential inputs by electrode cables.

Stimulation:

The full-field checkerboard pattern reversal (black and white checkerboard pattern), displaying at a rate of 3.5 reversals/ second (Hz). Mean luminance of the screen was fixed at 60-cd/m and contrast level of 100%. With visual angle of 16 (degrees) subtended by the stimulus field. The checkerboard size was selected according to the visual acuity of the subject between 60-90 (minutes). Each eye was stimulated separately by covering the other eye with a gauze patch (Monocular testing). VEP tests were filtered and amplified by the VEP computer program, and averaged of 200 runs according to response clearance. The amplifier band width was 0.1 – 100 Hz, with amplifier sensitivity of 2 µV and sweep speed of 500 msec/Div ¹⁸. Two series of examination of each eye were done to ensure reproducibility of the traces and results of VEP. During the test, the fixation point of the eye was kept on a target on the center of the screen (red dot), the subjects eye lids were fully opened to allow maximum amount of face and neck muscles, avoiding swallowing, moving tongue, speaking and frequent blinking, in order to minimize the artifacts during the

recording procedure.

Electroencephalography (EEG) study:

Electroneurodiagnostics is the study and recording of electrical activity in the brain and nervous system. Tests are performed by technologists who record information on paper or computer, and the results are then interpreted by a specially trained physician.

Brain cells continually send messages to each other that can be picked up as small electrical impulses on the scalp. The process of picking up and recording the impulses is known as an EEG.

The billions of nerve cells in your brain produce very small electrical signals that form patterns called brain waves. During an EEG, small electrodes and wires are attached to your head. The electrodes detect your brain waves and the EEG machine amplifies the signals and records them in a wave pattern on graph paper or a computer screen. A normal EEG means that you have a normal pattern of brain D wave activity. An abnormal reading means that abnormal patterns of brain activity are being produced and picked up.

EEG Procedure:

Electrical impulses in the brain are evaluated using an EEG. The test measures this electrical activity through several electrodes placed on your scalp. An electrode is a conductor through which an electric current can pass safely. The electrodes transfer information from your brain through wires to an amplifier and a machine that measures and records the data. The test is administered at a hospital, at your healthcare provider's office, or at a laboratory by a specialized technician. The test usually involves the following steps:

You will be asked to lie down on your back in a reclining chair or on a bed. The technician will measure your head and use a pencil to mark where electrodes will be attached to your scalp. These spots are then scrubbed with a special cream that helps the electrodes get a high-quality reading.

Results

In this study is show the overall mean age of patients with headache and control were (31.23±12.38)

and (30.40±7.38) years old, respectively, the distribution of patients and control by sex, (28.7%) and (47.3%) of patients and control were males, as show in the Figure (4).

In this study is show the differences between patients with headache and control by EEG Changes there was significant difference between patients and control by EEG, Patients with headache were 30 times more likely to have abnormal EEG, in the (Table 1).

In this study is shows the differences of patients with headache types and control during hyperventilation and photic stimulation of EEG. 24%, 14.66%, 1%, 1%, as show in the figure) 5).

Table (2) shows the Mean Differences of VEP amplitude Parameter by Patients with Headache and Control Groups. There were significant mean differences of VEP Amplitude RT and LT by study groups. Table (3) shows the Differences of Patients with Headache and Control Groups by VEP latency and waves morphology. There were significant differences of Patients with Headache and Control Groups by RT Latency Positive1, RT Latency Negative1, LT Latency Positive1, LT Latency Negative1, RT Latency Positive2, and wave Morphology. Case group were 6, 2, 19, 3, 13 and 24 times to have abnormal right latency 1 positive and negative and left latency 1 positive and negative, right latency 2 positive and abnormal wave morphology, respectively.

In this study is shows distribution of patients by types of headache. (71.30%), (13.3%), (10.0%), (2.7%), (2.0%) and (0.7%) of the patients had migraine without aura, migraine with aura, tension headache, cluster headache, chronic headache and TAC headache, respectively, as show in the figure (1).

In this study is shows The distribution of different EEG abnormalities in different types of migraine. Spike 25.96%, poly spike 12.96%, sharp 12.96%, slow 48.14%, as show in the figure (2). In this study show the distribution of different EEG abnormalities in different types of Non-migraine. Spike 0.0%, poly spike 0.0%, sharp 4.54%, slow 13.63%, as show in the figure (3).

Table 1: Differences of Patients with Headache and Control by EEG Findings.

Variable	Study Groups		χ^2	P Values	Odds Ratio (95% C.I.)
	Patients with headache (%)	Control (%)			
EEG Normal Abnormal	92 (61.3) 58 (38.7)	294 (98.0) 6 (2.0)	110.20	<0.001*	30.89 (12.91-73.92)

*p value ≤ 0.05 is significant

EEG = Electroencephalography

χ^2 = Chi-Square , C.I. = Confidence Interval

Table 2: Mean Differences of VEP amplitude by Patients with Headache and Control Groups.

Variable	Study groups	Mean	S.D	t-test	P value
VEP Amplitude RT (μ volt)	Case	7.47	2.25	8.622	<0.001*
	Control	6.22	0.80		
VEP Amplitude LT (μ volt)	Case	6.75	1.60	2.876	0.004*
	Control	6.37	1.15		

*p value ≤ 0.05 is significant

VEP = Visual Evoked Potential

RT = Right, LT = left

S.D = Standard deviation

Table 3: Differences of Patients with Headache and Control Groups by VEP latency and Waves Morphology.

Variable	Study Groups		χ^2	P values	Odds Ratio (C.I. 95%)
	Case (%)	Control (%)			
RT Latency Positive1 Normal < 75 (m.sec) Abnormal \geq 75 (m.sec)	103 (68.7) 47 (31.3)	281 (93.7) 19 (6.3)	49.938	<0.001*	6.749 (3.783-12.037)
RT Latency Negative1 Normal < 110 (m.sec) Abnormal \geq 110 (m.sec)	107 (71.3) 43 (28.7)	262 (87.3) 38 (12.7)	17.344	<0.001*	2.771 (1.696-4.527)
LT Latency Positive1 Normal < 75 (m.sec) Abnormal \geq 75 (m.sec)	79 (52.7) 71 (47.3)	287 (95.7) 13 (4.2)	121.79	<0.001*	19.841 (10.45-37.69)
LT Latency Negative1 Normal < 110 (m.sec) Abnormal \geq 110 (m.sec)	102 (68.0) 48 (32.0)	261 (87.0) 39 (13.0)	23.148	<0.001*	3.149 (1.948-5.092)
RT Latency Positive2 Normal < 145 (m.sec) Abnormal \geq 145 (m.sec)	130 (86.7) 20 (13.3)	99 (33.0) 201 (67.0)	115.24	<0.001*	13.19 (7.78-22.39)
LT Latency Positive2 Normal < 145 (m.sec) Abnormal \geq 145 (m.sec)	127 (84.7) 23 (15.3)	265 (88.3) 35 (11.7)	1.197	0.274	0.729 (0.414-1.286)
Wave Morphology Normal Abnormal	120 (80.0) 30 (20.0)	297 (99.0) 3 (1.0)	53.123	<0.001*	24.75 (7.413-82.635)

*p value \leq 0.05 is significant,

**reference group

RT = Right, LT = Left

χ^2 = Chi-Square

C.I. = Confidence Interval

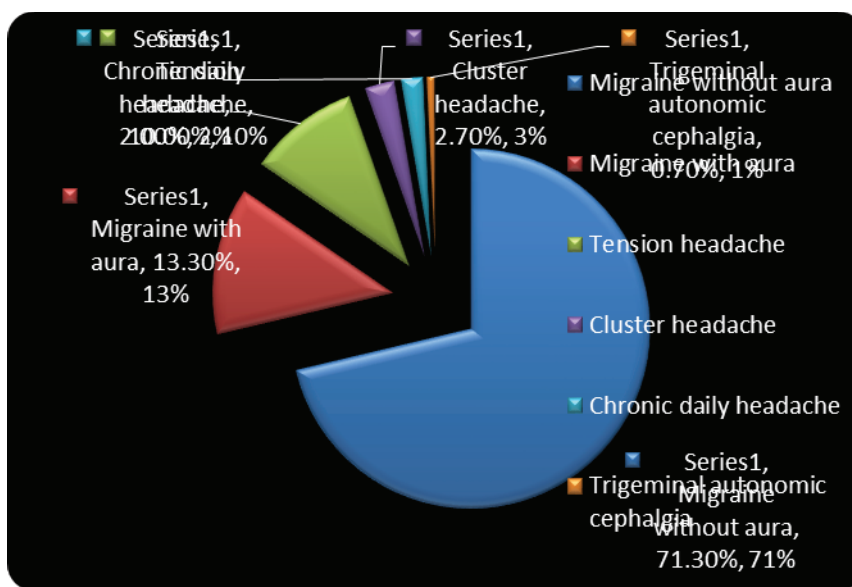


Fig. 1: Distribution of Patients by types of Headache.

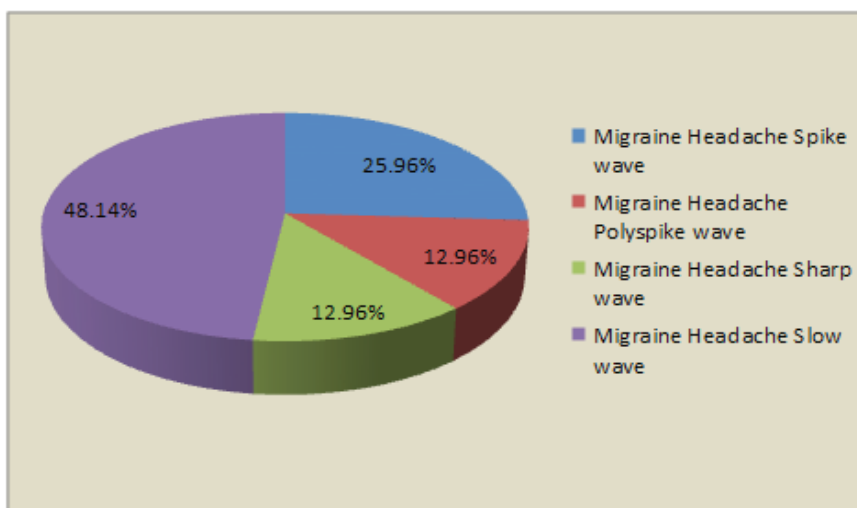


Fig. 2: The distribution of different EEG abnormalities in different types of migraine.

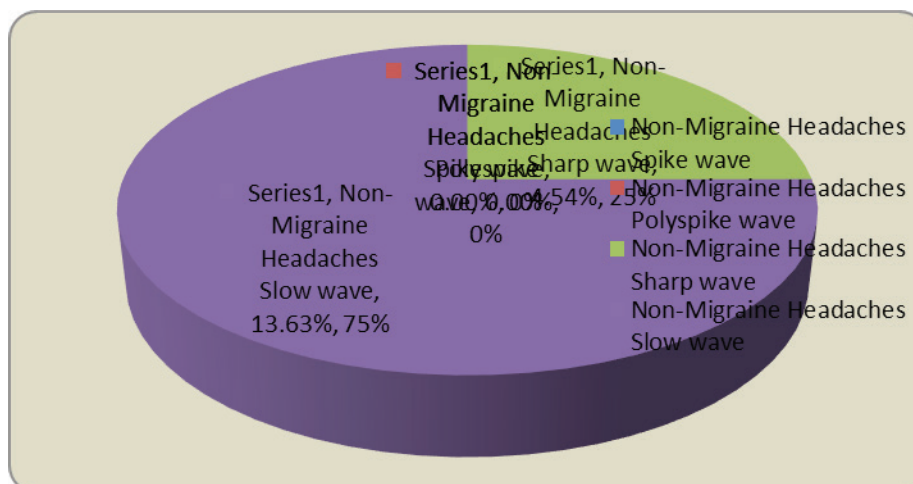


Fig. 3: The distribution of different EEG abnormalities in different types of non-migraine.

Discussion

Concerning the gender distribution, we have found that migraine is more common in females than males in different age groups at ratio of about 2.3:1. We found in figure (4) that female (47.30%) was susceptible to infected headache more than male (28.70%) in compared to control group. These findings are agreed with ²⁰ who found females 2:1 for headache, as well as with other studies of ²¹ and also agreed with ²² who found that female to male ratio is about 3:1, but ²³ found female to male ratio 3:2, so that there is a significant preponderance in females over males, this may be attributed to hormonal changes that occur in females mainly estrogen ²⁴. we found higher incidence of migraine without aura, then followed by migraine with aura, tension headache, cluster headache, chronic daily headache, trigeminal autonomic cephalgia (TAC). (66.0%), (13.3%), (10.0%), (2.7%), (2.0%) and (0.7%). This agreement with (Stephen L. Hauser, MD, 2006) who found Migraine without aura is more common than migraine with aura in our study group with ratio of 2:1.

Conclusion

This study stated that EEG, the significant abnormal findings in patients with migraine headaches are more than patients with non-migraine headaches. The abnormal EEG findings in migraine patients were found mainly during hyperventilation and photic stimulation. Epileptic discharge were found in about of the whole migraine patients represented by spike and wave, polyspike and sharp waves respectively, 25.69%, 12.69%, 12.69%. In VEP, regarding the latency of P75, N100 and P 145 there were significantly higher in patients with headache in compared to the control group, while there were significant changes related to the amplitude, but stay in normal range.

Financial Disclosure: There is no financial disclosure.

Conflict of Interest: None to declare.

Ethical Clearance: All experimental protocols were approved under the Hilla Teaching Hospital Neurosurgical department, Iraq and all experiments were carried out in accordance with approved guidelines.

References

1. Kunkel Robert S . “Headache”. Disease Management Project: Publications. Cleveland Clinic. Retrieved. 2010.
2. Krymchantowski AV. Headaches due to external compression. *Current Pain and Headache Reports*, 2010;14: 321.
3. Piane M , P Lulli ,I Farinelli ,S Simeoni , S De Filippis , FR Patacchioli ,P Martelletti. “Genetics of migraine and pharmacogenomics: some considerations”. *The journal of headache and pain*, 2007;8(6): 334-9. doi:10.1007/s10194-007-0427-2. PMC 2779399. PMID 18058067.
4. Stovner LJ , JA Zwart, K Hagen, GM Terwindt, J Pascual. “Epidemiology of headache in Europe”. *European Journal of Neurology*, 2006;13(4): 333-45.doi:10.1111/j.1468-1331.2006.01184.x. PMID 16643310.
5. Lay CL , SW Broner. “Migraine in women”. *Neurologic Clinics*, 2009;27(2): 503-11. doi:10.1016/j.ncl.2009.01.002. PMID 19289228.
6. Bartleson JD , FM Cutrer. “Migraine update. Diagnosis and treatment”. *Minn Med*, 2010;93(5): 36-41. PMID 20572569.
7. Dodick DW , JJ Gargus. “Why migraines strike”. *Sci. Am.* 2008;299(2): 56-63. Bibcode: SciAm.299b..56D. doi:10.1038/scientificamerican0808-56. PMID 18666680.
8. Cullom, Douglas R , Benjamin Chang, A Mark Friedberg , Christopher Rapuano. *The Wills Eye Manual, Office and Emergency Room Diagnosis and Treatment of Eye Disease*. J.B. Lippincott Company. 2004.
9. Hay KM , MJ Mortimer, DC Barker, LM Debney , PA Good. 1044 women with migraine: theeffect of environmental stimuli. *Headache*, 1994;34: 166-168.
10. Wilkins AJ, MI Nimmo-Smith, A Tait, C McManus, S Della Sala, A Tilley, A Arnold, M Barrie, S Scott. A neurological basisfor visual discomfort. *Brain*, 1984;107: 989-1017.
11. Marcus DA , MJ Soso. Migraine and stripe-induced visual discomfort. *Arch Neurol*, 1989;46: 1129-1132.
12. Wray SH . Mijovic-Prelec, D. and Kosslyn, S. M. Visual processing in migraineurs. *Brain*, 1995;118: 25-35.

13. Chaeles A . Advances in the basic and Clinical science of migrainr. *Ann Neurol*, 2009;65: 491.
14. Vos T , AD Flaxman, M Naghavi ,et al. Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990–2010: a systematic analysis for the Global Burden of Disease Study. *Lancet*. 2010;380: 2163-96
15. Pinessi L , I Rainero, C Rivoiro, E Rubino, S Gallone. “Genetics of cluster headache: an update”. *J Headache Pain (Review)* 2005;6(4): 234-6. doi:10.1007/s10194-005-0194-x. PMC 3452030. PMID 16362673.
16. Olesen J , MG Bousser, H C Diener, D Dodick, M First, P J Goadsby, et al. Headache Classification Committee. New appendix criteria open for a broader concept of chronic migraine. *Cephalalgia*, 2006;26: 742-6. [PubMed].
17. Bussone G. Clinical considerations on chronic migraine, pharmacoresistance and refractoriness. *Neurol Sci.*, 2010;31(Suppl 1): S83-5.
18. Goadsby PJ , RB Lipton. A review of paroxysmal hemicranias, SUNCT syndrome and other short-lasting headaches with autonomic feature, including new cases. *Brain*, 1997;120: 193-209.
19. Odom JV , M Bach, M Brigell, GE Holder, D L McCulloch, A P Tormene, Vaegan. ISCEV standard for clinical visual evoked potentials (2009 update) *Doc Ophthalmol.*, 2010;120: 111-119.
20. Russell MB , BK Rasmussen, P Thorvaldsen, J Olesen. Prevalence and sex-ratio of the subtypes of migraine. *Int J Epidemiol.*, 1995;24: 612–618. doi: 10.1093/ije/24.3.612.
21. Solomon S , N Mathew. Classification of daily and near daily headaches: proposed revisions to the IHS classification. *Headache.*, 1994;34(1): 1-7.