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RESEARCH ARTICLE

EVALUATION OF VISUAL EVOKED POTENTIAL IN MIGRAINE PATIENTS

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ARTICLE INFO	ABSTRACT	
<i>Article History:</i> Received 20 th February, 2015 Received in revised form 23 rd March, 2015 Accepted 28 th April, 2015 Published online 31 st May, 2015 <i>Key words:</i> Migraine, Aura, Visual evoked potential.	Aim: The present study was undertaken to evaluate the visual functions in Migraine patients. Materials And Methods: This study was carried out in the Department of Physiology, Thanjavur Medical College, Thanjavur. The subjects were recruited from Outpatient Clinic of Department of Neuromedicine, Thanjavur based on International Headache Society classification for Migraine. Subjects with episodes of headache for atleast 2 yrs, 2 attacks per month in last quarter year were	
	 included in the study and with history suggestive of other types of headache, Tension Type Headache (TTH), cluster headache, sinusitis and Visual field defects were excluded. Forty subjects (16 with Aura and 24 cases – Migraine without aura) and forty age / sex matched controls were selected. Informed written consent was obtained. The results were analysed statistically using student 't' test 	
	Results: There was significant prolongation of P100 and N145 latency (p<0.05) in both Migraine with and without aura compared with controls. P100 Amplitude was increased in study group especially in cases with Aura than the control group but was not statistically significant. Conclusion: Thus, Visual Evoked Potential can be considered as useful, non-invasive, reliable and diagnostic technique for effective management in Migraine.	

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INTRODUCTION

Headache is one of the most frequently encountered Neurological symptom (Nicholas A. Boon et al., 2006). Headache is caused by irritation of pain sensitive Intracranial structures like Dural sinuses, intracranial portions of Trigeminal, Glossopharyngeal, Vagus and upper Cervical nerves. The structures which are insensitive to pain are Brain parenchyma. Ependymal lining of ventricles and the Choroid plexus (Andreoli et al., 2007). Headache disorders can be classified into Primary Headache disorder and Headache secondary to structural brain disease. Primary Headaches are disorders in which headache and associated features occur in the absence of exogenous cause. Migraine, Tension type headache and Cluster headache are most common Primary headache syndromes (Dan L. Longo et al., 2012). Migraine is worldwide common, chronic, incapacitating Neurovascular disorder, characterized by attacks of severe headache, Autonomic nervous system dysfunction and an Aura involving neurologic symptoms. Individuals with Migraine appear to process Auditory and Visual information differently from those without Migraine (Laila El Mosly et al., 2012).

Migraine is the disorder of the brain characterized by complex sensory dysfunction (Till Sprenger and Peter J Goadsby 2009). It is an Episodic headache disorder and second most common type of primary headache (Dan L. Longo *et al.*, 2012) .Migraine occurs at any age either at childhood, adolescent or adulthood, more common in Females than Males in the ratio of 3:1. 60% of patients have positive Family history (Chugh S N, Ashima chugh 2010). Migraine has a great impact on mental, physical, functional and socioeconomic aspects of patient's life (Nofal MKhalil *et al.*, 2000). Migrainous have higher lifetime risk of Depressive disorder, Panic disorder, Generalized Anxiety disorder, phobias and Suicide attempts than the normal subjects (Laila El Mosly *et al.*, 2012).

The Diagnosis of Migraine was based on headache characteristics and associated symptoms which is subjective (Andreoli *et al.*, 2007). Routine Clinical Examination and testing for Visual function also appears to be normal in Migraine patients. So, Electrophysiological and Psychophysical tests have been carried out in Migraine patients (Nofal M Khalil *et al.*, 2000). The Migraineous brain is hyperexcitable not only during the attack but also in between attack i.e., the interictal phase. There is specific involvement of visual system in Migraine patients. Migraineous aura is visual in about 82 to 90% of cases (Nofal M Khalil *et al.*, 2000).

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Due to frequent occurrence of visual symptoms and due to impairment of Visual processing in Migraine many studies are oriented towards evaluation of VEP changes in Migraine patients which is a simple and Non-invasive test (Akbar Hamzei et al., 2013). Visual Evoked Potentials are electrical potential differences recorded from scalp in response to visual stimuli (Misra U K, J Kalita 2014). VEP assess the functional integrity of the central vision at any level of the Visual pathway (Vernon J et al., 2010). Pattern Reversal VEP is the most sensitive and reliable method of recording VEP. The stimulus is change of Black squares to white and white squares to black repeatedly at a specified number of reversals per second. (Walter G.Bradley et al., 2008). PRVEP is less variable than the pattern onset /offset and flash VEPs (Michael J Aminoff 2005). Functional and Electrophysiological alterations in cortical functioning also found, an association between Cognitive impairment and Migraine attack (Laila El Mosly et al., 2012). Hence, Visual Evoked Potential has been carried out in Migraine patients with and without aura to better understand the pathogenesis of Migraine and to utilize this test for Diagnosis and Effective management of Migraine. The aim of this study is to evaluate Electrophysiological parameter in Migraine patients with and without Aura VEP compared with controls and to study the role of VEP in the diagnosis of Migraine.

MATERIALS AND METHODS

This study, a case control study was conducted in the Research laboratory, Department of Physiology, Thanjavur Medical College & Hospital, Thanjavur. The study period extended from August 2013 to June 2014. The subjects were recruited from the Out-patient clinic of Department of Neuromedicine. The study group comprises of 40 Migraine patients who are subdivided into 16 patients – Migraine with Aura and 24 patients – Migraine without Aura , 4 males and 36 females of age group 19 to 52 yrs were selected according to International Headache Society Diagnostic Criteria for Migraine. Out of 40 controls, 6 males and 34 females of age group 19 to 55yrs with no history of headache, healthy controls were selected for the study.

Inclusion Criteria: Patients in the age group of 19 to 52 yrs diagnosed as Migraine with episodes of headache for atleast 2 yrs and atleast 2 attacks per month in the last quarter year were included in the study.

Exclusion Criteria: Subjects with Neurological diseases, Ophthalmic diseases, ENT & Systemic diseases, Visual and Auditory deficits were excluded.

Ethical Committee approval was obtained from the institution before commencing the study. The nature of the study was explained to the subjects, an informed written consent was obtained from the subjects prior to the study. A detailed history of Headache duration, frequency and history suggestive of aura and history to rule out other types of headache were noted. Ophthalmologic examination was done to determine visual acuity, Field of Vision, extraocular movements and pupillary diameter. All patients had Visual acuity 6/6 or corrected with optical lenses and none had any visual disorder. **Methodology:** VEPs were performed by checkerboard reversal pattern displayed on Zebronics CRT monitor showing pattern reversal stimuli with reversal rate 2/sec, contrast 50-80 %, check size 28-32 of arc with 100 average number of trials. The subject is instructed to sit at a distance of 1m from the VEP screen. Standard disc EEG electrodes were placed according to 10-20 international system. Active Electrode - Placed at Oz position- 10% from the inion. Reference electrode is placed at FPz position. Ground electrode is placed at vertex Cz. A waveform is obtained. VEP latencies (N75, P100 and N145 in ms) and P100 amplitude were measured.

RESULTS

Statistical analysis was done by using Statistical package SPSS version 20. The statistical analysis was done using unpaired student 't ' test. Values were expressed as mean with standard deviation. P value less than 0.05 was considered as statistically significant.

Table 1. Electrophysiological findings in cases (Migraine with aura) and Control group

Parameters	Migraine with Aura	Control	P value
	Mean \pm SD	Mean \pm SD	
VEP Latency			
N75	71.5438±3.41818	70.5750±4.20058	0.416
P100	102.0625±5.70782	95.1625±3.56512	0.000*
N145	136.218±10.45302	129.9875±8.61	0.025*
P100	6.2344±3.28791	5.7975±3.27955	0.654
Amplitude			

*P Value < 0.05

 Table 2. Electrophysiological findings in cases

 (Migraine without aura) and Control group

_	Migraine without Aura	Control	
Parameters	Mean \pm SD	Mean \pm SD	P value
VEP Latency			
N75	71.4896 ± 4.62653	70.5750 ± 4.20058	0.420
P100	99.9792 ± 5.06261	95.1625 ± 3.55126	0.000*
N145	134.5833 ± 5.45369	129.9875 ± 8.61	0.022*
P100	5.5371 ± 2.23703	5.7975 ± 3.27955	0.732
Amplitude			
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*P Value < 0.05

Our VEP study results showed that P100 and N145 latencies were significantly prolonged with P value < 0.05 in both Migraine with and without Aura when compared with controls whereas P100 Amplitude was increased in study group especially in cases with Aura than the control group but it was not statistically significant.

DISCUSSION

In the present study, the Visual Evoked Potential parameters were evaluated in Migraine patients with and without Aura and in 40 control group - healthy volunteers. Migraine, the most common Neurological disorder is associated with substantial functional impairment, involving both physical and emotional ramifications. Migraine can best be explained as a 'Brain state 'in which the cellular and vascular functional changes occur at the same time due to dysfunction of subcortical structures,

brainstem and diencephalic nuclei that modulate sensory inputs. These nuclei act as a 'Migraine Mediator' whose dysfunction will lead to abnormal perception and activation of Trigeminal Vascular System (TVS) which then activate the central structures. Thus, Migraine is mainly due to TVS activation generated within the brain without a peripheral sensory input. Migraine is the central sensory processing disorder, there is dysfunction of descending brainstem pain modulatory system. The hyperexcitability of the nociceptive circuitry downstream is responsible for the central sensitization in Migraine patients (Eric A.Moulton *et al.*, 2008).

The study results showed that P100 and N145 latencies were significantly prolonged with P value < 0.05 in both Migraine with and without Aura when compared with controls. EL Shater et al. (2006) Studied, PRVEP in 30 Migraine patients (11 patients with aura & 19 patients without aura) P100 latency was significantly prolonged in Migraine with aura cases not in without aura patients when compared with controls. There is no significant difference in P100 amplitude between patients and controls. They demonstrated that there is subtle neuronal damage within the visual system of migraine patients especially in patients with aura. The changes were due to recurrent cerebral hypoperfusion and cortical hyperexcitability between attacks. These results were consistent with our present study. Kennard et al., (1987) Studied VEP in Migraine patients and reported longer P100 latency in Migraine patients with aura. They suggested that prolonged latency have a structural basis due to ischemic damage from repeated attacks and due to hyperexcitability of the brain in Migraine. Similar results were found in our study. Laila EL Mosly et al. (2012) Evaluated the effect of Migraine on quality of life in females and associated changes in evoked potentials. They recorded VEP in 30 Migrainous females and reported that P100 latency was prolonged in Migraine patients but there was no significant difference in P100 amplitude. The prolongation was due to occipital cortex dysfunction that plays a role in the pathogenesis of Migraine and found to have a structural basis. These results are in accordance with our present study.

Drake et al., (1990) Recorded VEP in 50 patients with common Migraine. They found significant prolongation of P100 & N145 latencies in Migraine without aura and VEP amplitudes were minimally greater due to dysfunction of brainstem centers probably related to endorphin or serotonin neurotransmission. Mariani et al., (1990) Obtained VEP - PR in 20 Migraine patients with visual aura and without aura and reported an increase in P100 latency in Migraine with & without aura whereas Amplitudes were quite dispersed among patients and controls. Suggested the alterations in the monoamine neuromediators. The present study is congruent with the literature cited. Bockowski L et al., (2003) found significant prolongation of P100 latency whereas amplitudes N1-P100 & P100-N2 were prolonged in Migraine children than the children with tension type headache. They also reported that these amplitudes were lower in Migraine with aura when compared with Migraine without aura patients. These changes were due to visual dysfunction that might be secondary to a loss of inhibitory GABAergic interneurons in the visual cortex from repeated parenchymal insults. P100 latency showed similar findings in our study.

N. Ashjazadeh, B. Varavipour (2003) measured VEP in 53 Migraine cases (27 with aura, 26 are with common Migraine)and reported significant prolongation of P100 latency in classic Migraine patients with no change in the Amplitude due to subtle neuronal damage in the visual system of Migraine patients from repeated transient ischemia due to constitutional change. These results were consistent with our present study. Nofal M Khalil, Nigel J Legg, Duncan J Anderson (2000) Studied PRVEP in 92 Migraine patients. The mean latency of P100 was increased significantly in both Migraine with & without aura patients and explained it to be due to synaptic delay. They also showed positive correlation between disease duration and changes in P100 amplitude. The results of P100 latency were consistent with the present study.

Pedro. F Moreira Filho, Adalmir M. Dantas (1994) Studied PRVEP in 27 Migraine patients without aura. The study revealed that there is a significant increase in P100 latency when compared with controls and explained it to be due to alterations in Monoamine neuromediators. Similar results were found in our study.

Thus, Migraine patients with and without aura show significant prolongation of latency in Electrophysiological studies probably due to hyperexcitability of the cortex in Migraine patients even between attacks, due to synaptic delay and there is subtle neuronal damage within the visual system especially in patients with aura due to recurrent cerebral hypoperfusion. These findings suggest dysfunction of neuronal excitability due to defective neurotransmitter signaling and cerebral bioelectrical dysrhythmia.

Conclusion

Migraine patients with aura and without aura showed significant prolongation of P100, N145 Latency with no change in P100 amplitude. These findings suggest that there is defect in the central processing of visual function in Migraine. Thus, Visual Evoked Potential study can be considered as useful, non-invasive, reliable & diagnostic techniques for understanding the Neurophysiological processes involved in Migraine patients which aid in the selection of adequate, effective treatment in Migraine subjects. However, further studies are needed to compare the duration of the disease with the changes in the Electrophysiological study and to explain the Neuromodulatory centers in the brainstem in role of pathophysiology of Migraine. Then, the role of pattern reversal check size on VEP parameters in Migraine patients and lack of habituation during prolonged stimulation should be evaluated.

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