A Retrospective Descriptive Study of Frequency of Electroencephalographic Abnormalities and Its Correlation with White Matter Subcortical Lesions in Magnetic Resonance Imaging of the Brain in Acute Migraine Attack in a Tertiary Hospital

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ABSTRACT

The neuropathological processes believed to underlie migraine were still widely debated in the literature. This paper reviewed two ancillary procedures, the scalp electroencephalogram (EEG) and magnetic resonance imaging (MRI) of brain for physiological and anatomical characteristics of the syndrome that may be identified which may aid in the demonstration of the pathogenesis and pathophysiology of migraine. In this study, we determined the frequency of abnormal scalp electroencephalographic findings during acute (within 24 hours) migraine attack, and correlate it with MRI white matter lesions.

This was a retrospective descriptive study among migraine patients admitted at a tertiary hospital over a five-year period was done. Patients included were aged 10 to 70 years, both male and female, diagnosed with migraine according to IHS Criteria who underwent both EEG and MRI. Three hundred twelve patients were included in the study. Patients with both EEG and MRI whose results showed abnormalities in both were collected (n=11). Further analyses of correlation of abnormal EEG and white matter lesion on MRI using SPSS software and phi-coefficient statistical tool was used to provide measures of association in terms of lateralization, lobar distribution. Results showed insufficient evidence to make any correlation between laterality or lesions (pvalue 0.292) and lobar distribution (p-value 0.016). There was a positive but weak relationship among patients with abnormal EEG having an abnormal MRI (Odds Ratio= 2.475).

In conclusion, abnormalities in EEG and MRI findings may be seen during the acute attack (<24 hours EEG, < 3days MRI). Abnormal EEG patterns, specifically focal slowing of background activity and epileptiform discharges in multiple lobes in 37% percent of migraineurs was documented. A trend towards a positive correlation between the presence of abnormalities on MRI and abnormalities on EEG, this was not statistically significant. Evidence was insufficient because of the very limited sample size.

Keywords: *Migraine ☐ Electroencephalography (EEG) Magnetic Resonance Imaging (MRI) ☐ Computerised Tomography (CT) scan ☐ T2WI/FLAIR Hyperintensities*

INTRODUCTION

Migraine is a family of chronic disorders with highly variable clinical features, natural histories, and pattern of treatment response. Although, it is internationally recognized under a classification system of the International Headache Society,9 each classification needs to satisfy criteria (see Appendix 1) for every headache disorder. Diagnosis of the syndrome has traditionally been defined by the clinical characteristics of the acute attack, with no pathognomonic findings on laboratory tests and neuroimaging. This is why electroencephalography and neuroimaging are not considered necessary for the diagnosis of migraine headaches, although they can be helpful in further evaluation of patients with atypical headache presentations. With the advent of increasingly sophisticated diagnostic techniques and technologies, particularly in neuroimaging and neurophysiology, physiological and anatomical characteristics of the syndrome may be identified which may aid in the demonstration of the pathogenesis and pathophysiology of migraine.

Nonspecific T2WI/FLAIR subcortical and periventricular white matter hyperintensities have been found in migraine with aura (13 of 161; 8·1%), occurrence was significantly smaller in patients with migraine without aura (three of 134; 2·2%) and controls (one of 140; 0·7%) in the study of Kruit. 10 Cutrer et al proposed that these might be due to focal cerebral hypoperfusion and oligemia and thus be a risk factor for migraine-associated stroke.4 As these white matter lesions are most commonly subclinical, neurologic function of affected individuals is usually preserved. Because MRI is a test of structure rather than function, this study was conceptualized to determine whether electroencephalography, which is a test of function, would demonstrate any dysfunction among migraine patients, particularly in the presence of MRI white matter hyperintensities.

GENERAL OBJECTIVE

To determine the frequency of abnormal scalp electroencephalographic findings during acute (within 24 hours) migraine attacks, and correlate it with MRI abnormalities.

SPECIFIC OBJECTIVES

1. To describe the abnormal patterns of scalp EEG, including their lobar distribution and laterality, among migraine patients during the acute (within 24 hours) migraine attack.
2. To determine the frequency and describe the abnormalities of MRI findings among migraine patients when taken within 3 days of the acute migraine attack.
3. To correlate abnormal EEG findings with abnormal MRI findings.

METHODOLOGY

Study Design

This was a retrospective, descriptive study of patients admitted in a tertiary hospital because of migraine attacks from January 2007 to June 2013.

The admitting logbook of the Section of Neurology of a tertiary hospital was reviewed. Patients who were admitted due to an acute (within 24 hours) migraine attack were collected. The diagnosis of migraine was made by a neurologist according to International Headache Society (IHS) criteria.[8] When available, EEG recording done within 24 hours of admission and MRI of the brain (plain or with contrast) done within 3 days of admission were retrieved and reviewed.

Inclusion Criteria

Included in the study were patients both male and female diagnosed with migraine between ages of 10 to 70 years old.

Exclusion Criteria

Patients with history of clinical seizure, brain tumor, stroke, CNS infection, demyelinating disease, traumatic brain injury resulting to structural intracranial lesions or skull fractures and recurrent headaches not compatible with HIS criteria for migraine were excluded. Patients who had received antiepileptic

drugs for migraine prophylaxis during the week prior to the EEG recording were excluded. Patients with history of drug abuse were also excluded.

Data Collection

Age, gender, and performance of EEG recording or brain MRI were recorded using data collection forms. Confidentiality was ensured by not reporting patient identifying information (e.g. name, birthday, hospital number).

EEG Recording

Scalp EEG recordings were done using gold electrodes applied in accordance with the international 10-20 system using a Cadwell, Grass or Nihon-Koden electroencephalography machine with thirty-two (32) channels. Recordings were performed within at least 24 hours after the last headache attack. Each recording session lasted for a minimum of 20 minutes, with 3 minutes hyperventilation, and intermittent photic stimulation with a flash frequency ranging from 1-15Hz.

One independent electroencephalographer, blinded to the patient's identity, clinical profile and diagnosis except age, reevaluated the EEG recordings to eliminate interobserver variability. The following specific EEG findings were specifically identified and reported: (a) slow activity (generalized or focal), (b) attenuation/suppression of amplitude of background activity, (c) focal and generalized increase in EEG activity, (d) interhemispheric asymmetry of alpha rhythm; (e) epileptiform discharges (focal spikes, sharp waves, spike-andwave discharges, polyspike complexes, polyspike-and-slow-wave complexes multiple-sharp-wave complexes, multiple-sharp-and-slow -wave complexes, or generalized epileptiform discharges) and (f) response to photic and hyperventilation. Background activity, lobar distribution and laterality of abnormal EEG findings were also determined.

MRI of the brain

The author reviewed all available brain MRIs of subjects included in the study. In addition, when white matter hyperintensities were found, these were rated as follows.

MRI of the brain was reviewed for the presence of T2-weighted/FLAIR hyperintensities on subcortical regions but not present on T1-weighted image. Periventricular hyperintensities were not analyzed. Subcortical white matter lesions are categorized as small (0-3mm), medium (4-10mm), and large (>10mm). The number of subcortical white matter lesions was rated per size category for the frontal, parietal, occipital, temporal lobes and multilobar distribution. Affected cerebral hemisphere corresponding to its laterality was also listed. Distinction between lobes was according to anatomical landmarks. There were no added features recorded from the MRI images.

STATISTICAL ANALYSIS

Descriptive data were presented as frequency tables, cross-tabulations and summary statistics using Excel and/or SPSS for computation of frequencies, means, standard deviations and odds ratio. The researcher utilized the following statistical tools: frequencies and percentages to summarize the characteristics of flair hyperintensity among patients, and SPSS to determine if there exists a significant correlation between the EEG Results and the characteristics of T2WI/FLAIR hyperintensities. All statistical analyses performed were 2-sided at 95% Confidence Level.

RESULTS

During a five-year period of review, three hundred twelve (312) patients diagnosed to have migraine according to the International Headache Society Criteria were admitted in a tertiary hospital due to an acute migraine attack and were included in the study. EEG and MRI were reviewed, whenever available. Out of the total 312 patients listed in the logbook, 74% did not undergo any of the mentioned ancillary procedures, while the rest completed a combination of procedures.

The distribution of patients belonging into each group is presented in Table 1. Note that for the entire group, female patients admitted due to acute migraine greatly outnumbered male patients. Female -to-male ratio is 3:1. These patients have an average age of 38 year with a standard deviation of 12 years.

Table 1. Summary Characteristics of Patients

Group	Count	Percent %	Age (years) Distribution		Sex Distribution	
			Average	Stdev	Male	Female
EEG Only	28	9.0	33.6	12.2	7	21
MRI Only	6	19	33.0	6.7	2	4
EEG+MRI	34	10.9	37.7	13.3	8	26
None	224	78.2	38.5	11.6	60	184
TOTAL	**312**	**100**	**37.9**	**11.8**	**77**	**235**

From the table above, it can be observed that there were 62 (20%) patients who underwent scalp EEG with or without MRI. Of these 62 patients, 23 (37%) had abnormal EEG findings (Table 2). Patients with abnormal EEG findings had an average age of 38 +/- 14 years, and were mostly female.

Table 2. Characteristics of patients with EEGs

Group	Count	Percent %	Age (years) Distribution		Sex Distribution	
			Average	Stdev	Male	Female
Normal EEG	39	63	34.7	12.4	13	26
Abnormal EEG	23	37	37.9	13.7	2	21
TOTAL	**62**	**100**	**35.9**	**12.9**	**15**	**47**

Of the 62 patients who underwent EEG, 34 also underwent MRI (Table 3). Of the 34 subjects, 21 (61.8%) had abnormal MRI findings, among whom 11 subjects also had abnormal EEG findings, whereas 10 subjects had normal EEG findings. Of the 34 subjects, 9 had normal MRI and normal EEG findings while 4 had normal MRI but abnormal EEG findings.

Table 3. Normal and Abnormal EEG and MRI results

		MRI		
		Normal MRI	Abnormal MRI	Total
EEG	Normal EEG	9	10	19
	Abnormal EEG	4	11	15
	TOTAL	**13**	**21**	**34**

To test the relationship between MRI and EEG results (see Table 4), measure of association was done to determine the strength and significance should a relationship exist between the findings of these two tests. The following table provides Phi Coefficient, which was a chi-square-based measure of association that involves dividing the chi-square statistic by the sample size and taking the square root of the result. It measured the level of association between two nominal or categorical data.

The resulting Phi coefficient of 0.212 suggested a positive but weak relationship between MRI and EEG findings. This implied that there was a small chance that findings of normality or abnormality in both tests would coincide with each other. However, this finding is not statistically significant (p-value of 0.217) because of the small sample size.

Table 4. Relationship between MRI and EEG results

Symmetric Measures

		Value	Approx. Sig.
Nominal by Nominal	Phi	.212	.217
	Cramer's V	.212	.217
N of Valid Cases		34	

a. Not assuming the null hypothesis.
b. Using the asymptotic standard error assuming the null

Using the same table, odds ratio was computed to compare the relative odds of the MRI findings, given knowledge of EEG findings (results in Table 5). Given the data, odds ratio was computed to be 2.475. Thus, patients with abnormal EEG findings have a 2.475 times higher probability of having abnormal MRI findings than those with normal EEG findings. This supports the positive Phi coefficient obtained earlier. However, since 1 is included in the 95% confidence interval (0.577, 10.617), the observed relationship was deemed to be not statistically significant.

Table 5. Risk estimate between EEG and MRI findings

Risk Estimate

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for EEG ABNORMAL/NORMAL)	2.475	.577	10.617
For cohort MRI = ABNORMAL	1.393	.825	2.354
For cohort MRI = NORMAL	.563	.215	1.476
N of Valid Cases	34		

From the 34 patients who had undergone both MRI and EEG, the sample was further narrowed down to those patients with abnormal EEG who underwent MRI of the brain regardless of MRI result (N=23). Of these 23 patients, only 15 patients with abnormal EEG had undergone MRI. This brought down the sample size of our succeeding analyses to only 15 patients as shown in table 6, consisting of 1 male and 14 female patients. Patients were older with an average age of 43 + 13 years.

Table 6. Patients with Abnormal EEG who underwent MRI of the brain according to age and sex distribution

Group	Count	Percent %	Age (years) Distribution		Sex Distribution	
			Average	Stdev	Male	Female
With MRI	15	65	42.9	12.9	1	14
Without MRI	8	35	28.5	9.9	1	7
TOTAL	**23**	**100**	**37.9**	**13.7**	**2**	**21**

Test Results of Abnormal EEG Findings

EEG abnormalities found in the 15 patients with abnormal EEGs who had undergone MRI were studied. Summary of abnormal EEG findings corresponding to lateralization, lobar distribution and number of lobes involved were summarized in Table 7. Out of 15, 60% of abnormal findings were unilateral, with 40% on the left cerebral hemisphere and 20% on the right cerebral hemisphere. In 40% of the patients, abnormal findings were observed over both cerebral hemispheres. Moreover, there was an obvious trend towards higher incidence of abnormality involving multiple lobes in 86%.

Table 7. Characteristics of abnormal EEG findings of patients with abnormal EEG Result

EEG with ABNORMAL FINDINGS				
Lateralization	**Left**	**Right**	**Bilateral**	**Total**
Count	6	3	6	15
%	40	20	40	100
Number of Lobes		**Single Lobe**	**Multilobar**	**Total**
Count		2	13	15
%		13	87	100
Lobar Distribution	**Occipital**	**Temporal**	**Multilobar**	**Total**
Count	1	1	13	15
%	7	7	86	100

Specific EEG abnormalities were enumerated in table 8. The most frequently observed abnormal EEG pattern were focal slowing (46.7%) and epileptiform discharges (46.7%), followed by interhemispheric asymmetry of alpha rhythm (5%). Photic stimulation elicited asymmetric photic driving in 4% and hyperventilation further elicited abnormal response in 5%.

Table 7. Characteristics of abnormal EEG findings

EEG with ABNORMAL FINDINGS			
	Present %	**Absent**	**Total**
Generalized Slow Activity	2 (13.3)	13	15
Focal Slow Activity	7 (46.7)	8	15
Attenuation of Background Activity	1 (6.7)	14	15
Fast Activity	2 (13.3)	13	15
Interhemispheric Asymmetry of Alpha Rhythm	5 (33.3)	10	15
Epileptiform Discharges	7 (46.7)	8	15
Photic Response	4* (26.7)	11	15
Hyperventilation Response	5** (33.3)	10	15
*Asymmetrical photic driving **Abnormal response			

MRI Results

We reviewed the MRIs of patients done within three days of the acute migraine attack. No acute infarctions, hemorrhages or mass lesions were seen. From the 15 patients with abnormal EEG findings who underwent MRI, only 11 had abnormal MRIs. All MRI abnormalities consisted of the subcortical white matter lesions, which appeared isointense on T1-weighted image (T1WI) and hyperintense on T2-weighted image (T2WI) and Fluid Attenuated Inversion Recovery (FLAIR). Table 9 presents characteristic data of subcortical white matter lesions. In terms of number of white matter lesions, results ranged from as low as 1 to a maximum of 16 recorded flair hyperintensities. Right hemisphere was found to have frequent T2WI/FLAIR hyperintensities in 55% and it was the frontal lobe (55%) that was frequently involved. Small hyperintensities (1-3mm) were the most common in 73%.

Table 7. Summary Characteristics of Subcortical White Matter Lesions (T2WI/FLAIR Hyperintensities)

MRI Results: Number of Flair Hyperintensities					
Valid	Mean	Median	Stdev	Maximum	Minimum
11	4.3	2.0	4.9	16	1
MRI Results					
Lateralization		**Left**	**Right**	**Bilateral**	**Total**
Count		2	6	3	11
%		18	55	27	100
Lobar Distribution	**Frontal**	**Temporal**	**Parietal**	**Multilobar**	**Total**
Count	6	1	1	3	11
%	55	9	9	27	100
Size of WML*			**Small**	**Combination**	**Total**
Count			8	3	11
%			73	27	100

**WML - White Matter Lesions*

Correlation / Measures of Association between EEG and MRI findings

To further analyze if there was a relation between the abnormal results of EEG and MRI in the 11 patients who had both abnormal EEG and abnormal MRI results, statistical tests were used to determine their level of association both in terms of lateralization and lobar distribution.

For this kind of analysis, SPSS software was utilized to generate the necessary statistics and significance levels. The following test provides Phi Coefficient, which was a chi-square-based measure of association that involves dividing the chi-square statistic by the sample size and taking the square root of the result. It measured the level of association between to nominal or categorical data.

In each of the tests, p-values were given which aided in determining the significance of each relationship. At 5% level of significance, a p-value of less than 0.05 suggests strong and statistically significant relationship between the variables of interest.

For lateralization (see Tables 10.1 and 10.2), the test resulted in a p-value of 0.292, showing that there was insufficient evidence to make any correlations between laterality of lesions on MRI and laterality of findings on EEG.

Table 10.1 Correlation of abnormal EEG findings vs Laterality of Subcortical White Matter Lesions (T2WI/FLAIR Hyperintensities) on MRI

Contingency Table: Lateralization					
			MRI		
		Bilateral	**Left**	**Right**	**Total**
EEG	**Bilateral**	2	0	2	4
	Left	0	2	3	5
	Right	1	0	1	2
	TOTAL	3	2	6	11

Table 10.2 Correlation of abnormal EEG findings vs Laterality of Subcortical White Matter Lesions (T2WI/FLAIR Hyperintensities) on MRI Symmetric Measures

		Value	**Approx. Sig**
Nominal by Nominal	Phi	.671	.292
	Cramer's V	.474	.292
N of Valid Cases		11	

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis

For lobar distribution (see Tables 11.1 and 11.2), the resulting p-value is 0.016, suggesting that there was insufficient evidence to conclude that lobar distribution of MRI abnormalities are positively correlated with lobar distribution of abnormal EEG findings.

Table 11.1 Correlation of abnormal EEG findings vs Lobar Distribution of Subcortical White Matter Lesions (T2WI/FLAIR Hyperintensities) on MRI

Contingency Table: Lobar Distribution							
					MRI		
		Multilobar	Frontal	Pariental	Temporal	Occipital	Total
EEG	Multilobar	3	4	1	1	-	9
	Frontal	-	-	-	-	-	-
	Pariental	-	-	-	-	-	-
	Temporal	-	1	-	-	-	1
	Occipital	-	1	-	-	-	1
	Total	3	6	1	1	-	11

Table 11.2 Correlation of abnormal EEG findings vs Lobar Distribution of Subcortical White Matter Lesions (T2WI/FLAIR Hyperintensities) on MRI Symmetric Measures

		Value	Approx. Sig
Nominal by Nominal	Phi	.430	.916
	Cramer's V	.304	.916
N of Valid Cases		11	

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis

Correlation/Measures of Association between Age and EEG and MRI findings

In the following analysis, age was associated with the EEG and MRI findings. In Table 12.1, the study tests the hypothesis that as age increases, the higher the chance of abnormal findings on EEG. The second hypothesis tests whether the number of T2WI/FLAIR hyperintensities on MRI increases with age.

In the following test, Kendall's tau-b was use to detect a relationship between the variables and measure its strength if it exists. Kendall's tau-b was a nonparametric measure of correlation for ordinal or ranked variables that take ties into account. The sign of the coefficient indicates the direction of the relationship, and its absolute value indicates the strength, with larger absolute values indicating stronger relationships. Possible values range from -1 to 1, but a value of -1 or +1 can be obtained only from square tables.

Table 12.2 demonstrated a negative value of Kendall's tau-b (-0.124) suggesting that as age increases, abnormal EEG findings were more frequently observed. However, this relationship was not statistically significant (p=0.283).

Table 12.1. Age Distribution of EEG results both normal and abnormal

Contingency Table: Age vs EEG Findings				
	ABNORMAL	NORMAL	N.I	TOTAL
11-25	4	11	3	18
26-40	10	17	13	40
41-55	7	8	3	18
56-70	2	3		5
TOTAL	**23**	**39**	**19**	**81**

Table 12.2 Age Distribution of EEG results both normal and abnormal

Symmetric Measures

		Value	Asymp. Std. Error[a]	Apporx. T[b]	Approx. Sig
Ordinal by Ordinal	Kendall's Tau-b	-.124	.115	-1.074	.283
N of Valid Cases		62			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis

In the second hypothesis, the number of T2WI/FLAIR hyperintensities was correlated against the age of patients with acute migraine attack. Since both of the variables were numeric in nature, Pearson's correlation was used instead to detect linear relationship. Note however that only 21 MRI results indicated the number of T2WI/FLAIR hyperintensities, hence the rest did not demonstrate such findings on MRI and were excluded from the test. The following SPSS result indicated a positive value of the coefficient (0.695) consistent with a strong statistically significant positive relationship between patient's age and number of T2WI/FLAIR hyperintensities (p,0.01). This suggests that as age increases, the number of T2WI/FLAIR hyperintensities on MRI also increases. (see Table 13)

Table 13 Correlation of AGE with abnormal EEG finding and number of T2WI/FLAIR hyperintensities,

		AGE	**Number of T2WI/FLAIR Hyperintensities**
Age	Pearson Correlation	**1**	**.695****
	Sig. (2-Tailed)		**.000**
	N	**40**	**21**
Flair_number	Pearson Correlation	**.695****	**1**
	Sig. (2-tailed)	**.000**	
	N	**21**	**21**

***. Correlation is significant at the 0.01 level (2-tailed)*

DISCUSSION

Most of the published studies using routine EEG were done in children with all types of recurrent headaches including tension-type headache, migraine, sinus headache and so on. Studies of Grosneth[7], Kruit[10], and Bockowski et al[3] reported EEG findings to occur more frequently in headache patients. They described the following patterns: generalized or focal slowing, rhythmic highamplitude slowing during hyperventilation, excessive fast activities, epileptiform activities and prominent photic driving, and differences in the asymmetry, modulation or peak frequencies of the alpha rhythm. Similar EEG findings were also observed in our subjects. However, the percentage of the patients with abnormal EEG varies for different studies from 20.6% to 40.8% that was consistent with our result.[6] Despite the low frequency of abnormal EEG findings, migraineurs are more likely to have an abnormal EEG. These data correlate cardinally with our results. EEG was interpreted as abnormal in 28% with the most frequent abnormalities described as focal slowing and presence of epileptiform discharges in the form of sharps, spikes, and spike-and-slow waves. Our patients with epileptiform discharges did no show any periodic lateralized epileptiform discharges (PLEDS) unlike in study of Brinciotti.[8]

The differentiating factor in our study was the timing of the EEG recording, which was done during the acute migraine attack (within 24 hours) – whereas timing of EEG recording was not mentioned in most of the previous EEG and headache studies. Some studies were done during the interictal period. This study showed a trend towards higher frequency of abnormal EEG patterns among older patients, mean age of 37.9+13.7 years as opposed to subjects with normal EEG tracings with mean age of 34.7+12.4 years. However, this finding was not statistically significant.

Consistent findings in our migraine patients of prominent asymmetrical photic driving at high flash rates during the acute attacks3 are compatible with the findings reported by Grosneth6 in a review of 40 studies and in studies of Golia and Winter.[6] However, asymmetry of photic driving may be considered a normal variant.

Presence of EEG abnormalities, might lead to the request for neuroimaging to rule out a structural pathology particularly when such findings were demonstrated in a focal distribution. However, findings on MRI showed T2WI/FLAIR hyperintensities that were isointense on T1WI was common and majority had small sizes between 1-3mm. Linking the association of abnormal EEG pattern and white matter lesions on MRI based on the laterality and lobar distribution of both findings did not show any correlation statistically. Hence, an abnormal EEG finding may not always necessitate request for MRI.

White matter lesions on MRI were independent findings among our patients with migraine, but most migraineurs will not have them. Increasing age was not correlated with increased frequency of abnormal EEG findings. However, this study showed that increasing age is statistically significantly correlated with increase in the number of white matter lesions on MRI. Hence, MRI may be more useful when done among migraine patients in the older age groups.

Limitations of the Study

This study was only limited to patients with the diagnosis of migraine who were admitted because of an acute attack of migraine. The fact that collection of subjects was a retrospective approach limited the amount of data collected. In particular, our patients were not classified according to the type of migraine because not all admitting diagnoses indicated the type of migraine. Frequency of attacks, duration of migraine, presence of family history and previous history of benign febrile convulsions were not assessed.

CONCLUSION

In conclusion, although EEG and MRI are not recommended in the diagnosis of migraine, abnormalities in EEG and MRI findings may be seen during the acute attack (<24 hours EEG, < 3days MRI). This study showed that abnormal EEG patterns, specifically focal slowing of background activity and epileptiform discharges, may be seen in multiple lobes during acute migraine attacks in 37% percent of migraineurs. It also showed that MRI abnormalities, specifically

RECOMMENDATIONS

Our results were preliminary findings relating to a small number of subjects. A larger population-based blinded study would open up new avenues likely to our further understanding of the complexity of migraine syndrome. By combining electrophysiological data obtained through electroencephalogram with hemodynamic data probably through functional magnetic resonance imaging (fMRI), may be possible to arrive at a more detailed and comprehensive picture of the altered processes present in migraine taking into consideration the epoch of headache (both ictal and interictal), and the type of migraine. A blinded prospective controlled study was recommended before firm conclusions were drawn.

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APPENDIX I

INTERNATIONAL HEADACHE SOCIETY DIAGNOSTIC CRITERIA FOR MIGRAINE (2004)

Diagnostic criteria for migraine without aura:

A. At least five attacks fulfilling criteria B-D
B. Headache attacks lasting 4-72 hours [when untreated in adults]
C. Headache has at least two of the following characteristics:
   1. unilateral location
   2. pulsating quality
   3. moderate or severe pain intensity
   4. aggravation by or causing avoidance of routine physical activity
D. During the headache, at least one of the following [is present]:
   1. Nausea and/or vomiting
   2. Photophobia and phonophobia
E. Not attributable to another disorder

— International Classification of Headache Disorders[6]

Diagnostic criteria for Pure menstrual migraine without aura (A1.1.1)

A. Attacks, in a menstruating woman, fulfilling the criteria for migraine without aura
B. Attacks that occur exclusively from days -2 to +3 of menstruation in at least 2 out of 3 menstrual cycles and at no other times of the cycle.

Note: The first day of menstruation is day +1, and the preceding day is day -1; there is no day 0.

— International Classification of Headache Disorders[6]

Diagnostic Criteria for Migraine with Aura

Description: Recurrent disorder manifesting in attacks of reversible focal neurological symptoms that usually develop gradually over 5–20 minutes and last for less than 60 minutes. Headache with the features of "migraine without aura" usually follows the aura symptoms. Less commonly, headache lacks migrainous feature or is completely absent [i.e., the aura may occur without any subsequent headache].

Diagnostic criteria for Migraine with Aura

A. At least two attacks fulfilling criterion B
B. Migraine aura fulfilling criteria [described below]
C. Not attributed to another disorder.

...[Criteria for "Typical aura":] Aura consisting of at least one of the following, but no motor weakness:

1. Fully reversible visual symptoms including positive features (e.g. flickering lights, spots or lines) and/or negative features (i.e., loss of vision)
2. Fully reversible sensory symptoms including positive features (i.e., pins and needles) and/or negative features (i.e., numbness)
3. Fully reversible dysphasic speech disturbance [Aura also has] at least two of the following:
    1. Homonymous visual symptoms [i.e., affecting just one side of the field of vision] and/or unilateral sensory symptoms [i.e., affecting just one side of the body]
    2. At least one aura symptom develops gradually over [at least] 5 minutes and/or different aura symptoms occur [one after the other] over [at least] 5 minutes 3. Each symptom lasts [from] 5 [to] 60 minutes

...[Other potential aura criteria:]

5. Fully reversible motor weakness...
6. Each aura symptom lasts [from] 5 minutes [to] 24 hours...
   [In the case of a "Basilar-type" migraine], Dysarthria [difficulty speaking], vertigo [dizziness], tinnitus [ringing in the ears], [and other symptoms].

- International Classification of Headache Disorders[6]

Diagnostic Criteria for Abdominal Migraine

Diagnostic criteria:

A. At least 5 attacks fulfilling criteria B-D.
B. Attacks of abdominal pain lasting 1-72 hours (untreated or unsuccessfully treated)
C. Abdominal pain has all of the following characteristics:
    1. midline location, periumbilical or poorly localized
    2. dull or "just sore" quality
    3. moderate or severe intensity
D. During abdominal pain at least 2 of the following:
    1. anorexia
    2. nausea
    3. vomiting
    4. pallor
E. Not attributed to another disorder

- International Classification of Headache Disorders[6]