Title: Interictal Cortical Electrophysiological Signatures in Migraine with and Without Aura: A Quantitative EEG-Based Investigation of Band Power Al-terations and Symptom Correlations

Authors: Mostafa Jalilifar[1,*], Jafar Fatahi-Asl[1], Siamak Askari[2], Shahram Nasiri[2]

1. *Department of Radiologic Technology, School of Allied Medical Sciences, Ahvaz, Jundishapur University of Medical Sciences, Ahvaz, Iran.*
2. *Department of Pediatric Neurology, Golestan Medical Educational and Research Center, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran.*

***Corresponding Author**: Mostafa Jalilifar, Department of Radiologic Technology, School of Allied Medical Sciences, Ahvaz, Jundishapur University of Medical Sciences, Ahvaz, Iran. Email: jalilifarmostafa@gmail.com

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Abstract

Background: Migraine is a prevalent neurological disorder characterized by recurrent headaches and sensory hypersensitivities such as photophobia and phonophobia.

Objective: This study investigates quantitative electroencephalography (QEEG) patterns in migraine patients with and without aura (MWA and MWO) during a true interictal phase to identify neurophysiological biomarkers associated with clinical symptoms.

Method: Resting-state EEG recordings were obtained from 19 patients with MWO, 17 with MWA, and 16 age- and sex-matched healthy controls. EEG data from 19 scalp electrodes (10–20 system) were recorded under eyes-open and eyes-closed conditions, preprocessed to remove artifacts, and analyzed using Fast Fourier Transform (FFT) to compute absolute power across delta, theta, alpha, beta, and high-beta frequency bands. Z-scored QEEG values were derived using NeuroGuide software. Group comparisons were performed with independent-sample t-tests, and correlations between spectral power and clinical symptoms were assessed using Spearman's rank-order correlation.

Results: QEEG analysis revealed significant increases in delta, theta, and high-beta power in migraine patients, especially MWA, compared to healthy controls. Following False Discovery Rate (FDR) correction, posterior slow-wave alterations remained significant in MWA ($q < 0.05$). Delta and theta power exhibited strong correlations with headache intensity, while reduced alpha power was linked to photophobia.

Conclusion: These findings suggest that cortical hyperexcitability and thalamocortical dysrhythmia play central roles in the pathophysiology of migraine. QEEG biomarkers, while still exploratory, may offer valuable insights for the objective diagnosis and monitoring of migraine, potentially guiding targeted non-invasive therapies. However, further studies are needed to confirm their clinical utility and reliability in larger, diverse cohorts.

Keywords: QEEG, Migraine, cortical spreading depression, Fourier transform, Headache

Introduction

Migraine is a complex neurological disorder that leads to significant disability worldwide and affects approximately 12% to 15% of the general population (Amiri, Kazeminasab et al. 2022). It is 3 times more common in women than men and most common between the ages of 25 to 55 years. Migraine is characterized by recurrent episodes of unilateral pulsatile headache and associated nausea, vomiting, photophobia and phonophobia according to IHS- 2018 classification migraine has different forms that are distinguished by presence or absence of aura (migraine with Aura (MWA) & without aura (MWO)) or by the number of monthly headache days (episodic migraine and chronic migraine) (Alpuente, Tassorelli et al. 2020).

The outdated vascular theory of migraine pathophysiology has been largely replaced by the prevailing view that migraine aura originates from a primary neuronal disturbance known as cortical spreading depression (CSD), which serves as the electrophysiological basis of this phenomenon (Close, Eftekhari et al. 2019). This propagating cortical wave also results in activation of trigeminovascular sensory system and subsequent release of vasoactive neuropeptide such as substance P, CGRP and neurokinin, that induces neurogenic inflammation (Ashina, Hansen et al. 2019) and trigger the headache phase of migraine (Bigal, Ferrari et al. 2009, Noseda and Burstein 2013, Charles 2018). Beyond CSD, contemporary models of migraine increasingly emphasize large-scale network dysfunction involving the brainstem, hypothalamus, and salience network. Functional neuroimaging studies suggest that altered connectivity within these networks may contribute to abnormal sensory processing, pain modulation, and susceptibility to attacks, supporting the view of migraine as a systems-level brain disorder rather than a purely focal cortical phenomenon. Recent advancements in brain mapping techniques, particularly quantitative electroencephalography (QEEG), have emerged as powerful tools for elucidating the underlying pathophysiological mechanisms of migraine (Minnerly, Shokry et al. 2021). Previous electrophysiological investigations in migraine have employed heterogeneous methodological approaches, including conventional resting-state EEG, event-related potentials (ERP), and quantitative EEG (QEEG) analyses. Resting-state EEG primarily evaluates baseline cortical oscillatory dynamics, whereas ERP paradigms assess stimulus-locked cortical reactivity and sensory processing (Zhang, Pan et al. 2023, Zebhauser, Heitmann et al. 2024). In contrast, QEEG absolute power analysis provides a quantitative spectral decomposition of spontaneous neural activity across predefined frequency bands, offering a frequency-specific index of cortical excitability and large-scale network dynamics. Differentiating these methodological approaches is essential, as they reflect

distinct neurophysiological dimensions of migraine pathophysiology. By delivering a detailed and precise representation of cortical electrical activity, this technique has facilitated the identification of migraine-related biomarkers and has opened avenues for novel, non-invasive therapeutic approaches, most notably neurofeedback (Lia, Carenini et al. 1995). In this regard, different studies have also revealed that patients with migraines show certain electrophysiological changes in some frequency bands of brain waves, which can be used as diagnostic indicators or criteria for evaluating and monitoring treatment efficacy (BS 1962, Whitehouse, Pappas et al. 1967, Rowan 1974, Schoenen 1997). Nevertheless, the QEEG findings in migraine research remain heterogenous and, at times, contradictory, (Sand 1991, Schoenen 1992, Gronseth and Greenberg 1995, Sand 2003) which might be due to the conditions wherein QEEG was acquired, such as some patients were in a pre-attack or attack phases (Farlas, Benninger et al. 1987, Neufeld, Treves et al. 1991, Genco, Tommaso et al. 1994, Lia, Carenini et al. 1995, Rainero, Amanzio et al. 2001, Bramanti, Grugno et al. 2005). Notably, QEEG holds potential for capturing neurophysiological alterations preceding migraine attacks, offering insight into the pre-ictal cortical state (Bjørk and Sand 2008). Therefore, it is essential to reevaluate EEG recordings obtained during a true interictal window, minimally influenced by either preceding or imminent migraine episodes, to ensure the reliability and accuracy of electrophysiological data. Although contemporary models emphasize shared pathophysiological mechanisms between MWA and MWO, emerging neurobiological evidence suggests partially distinct cortical dynamics. CSD, considered the electrophysiological substrate of aura, predominantly involves posterior cortical regions and has been linked to altered thalamocortical interactions and structural modifications within visual processing networks. These findings provide a hypothesis-driven rationale for separately examining MWA and MWO during the interictal phase to determine whether aura-related mechanisms are associated with distinct oscillatory signatures. In this regard, multiple studies have reported the correlation between possible ischemic, white matter lesions (Kruit, van Buchem et al. 2004, Kruit, Launer et al. 2005, Kruit, Launer et al. 2005, Kruit, Launer et al. 2006, Rozen 2007) and grey matter changes (Rocca, Ceccarelli et al. 2006, Valfrè, Rainero et al. 2008) with attack frequency (Kruit, van Buchem et al. 2004, Valfrè, Rainero et al. 2008) and disease duration in migraineurs (Rocca, Ceccarelli et al. 2006). These structural alterations may reflect or coexist with functional oscillatory disturbances detectable by QEEG, although a direct causal relationship cannot be assumed. For example, it was reported that cerebral ischemia leads to the increase of theta and delta while decrease of alpha activity (Cohen, Bravo-Fernandez et al. 1976, Nuwer, Jordan et al. 1987, Finnigan, Walsh et al. 2007). In this

connection, it can be intriguing area of study to explore the association of interictal cortical function with migraine symptoms and severity.

Despite increasing interest in the electrophysiological underpinnings of migraine, the neural signatures that differentiate MWA from MWO remain incompletely understood, particularly during the interictal phase, where the brain is free from the immediate influence of an attack. Although numerous studies have employed QEEG to explore cortical activity in migraine (Bramanti, Grugno et al. 2005, Bjørk and Sand 2008, Ashina, Hansen et al. 2019, Barbanti, Brighina et al. 2020), variations in recording conditions and analytical approaches have hindered the comparability and generalizability of their results. Identifying consistent electrophysiological markers, particularly spectral power alterations across specific EEG frequency bands, may provide new insight into the neurophysiological mechanisms that differentiate migraine subtypes.

To address these gaps, the present study performed a comprehensive multiband QEEG absolute power analysis across five cortical regions during a rigorously defined interictal window in patients with migraine with and without aura. We further examined associations between regional spectral power alterations and key clinical features, including headache intensity, photophobia, and phonophobia. By integrating frequency-specific and region-specific metrics within a strictly interictal framework, this study aimed to determine whether MWA and MWO exhibit distinct oscillatory signatures that may reflect differences in thalamocortical and large-scale network dynamics. This approach extends previous work, which often focused on a single frequency band or limited cortical regions, by providing a multiband, multiregional analysis linked to clinically relevant symptoms, offering a more refined and clinically meaningful electrophysiological characterization of migraine subtypes.

Materials and methods

Subjects

Participants aged between 20 and 50 years were enrolled and categorized into three groups: migraine without aura (MWO; n = 19), migraine with aura (MWA; n = 17), and a healthy control group (n = 16) with no prior history of headache or psychiatric disorders, including anxiety or depression. All participants underwent structured clinical interviews conducted by a board-certified neurologist. The diagnosis of migraine was established based according to the

International Classification of Headache Disorders, 2nd Edition (ICHD-II), using diagnostic criteria 1.1 for MWO and 1.2.1 for MWA.

To minimize confounding influences on QEEG measures, participants were excluded if they had taken medications known to affect cortical excitability (e.g., antiepileptics, neuroleptics, benzodiazepines) within 48 hours of EEG recording. Individuals with comorbid primary headache disorders (particularly tension-type headaches) or other neurological conditions affecting cortical function were also excluded. These steps ensured that spectral alterations primarily reflected interictal cortical activity related to migraine.

Patients completed a structured questionnaire assessing various headache characteristics, including headache duration (>72 hours, 4–72 hours, <4 hours), number of headache days (0–8 days, 8–14 days, >15 days), attack frequency, presence of nausea and vomiting, headache exacerbation due to stress or missed meals, and their capacity to work during headache episodes. Based on these responses, headache intensity was rated on a scale from 1 to 3 reflecting severity, while associated photophobia and phonophobia were independently scored from 0 to 3.

Clinical evaluations and EEG recording

EEG recordings were obtained during a strictly defined interictal window, operationally defined as at least 48 hours after the last migraine attack and at least 48 hours before any subsequent reported attack, based on patient self-report and follow-up confirmation. The EEG cap (Electro-Cap International Inc., OH, USA) was placed on the patient head according to the international 10-20 system (Figure 1). EEG recordings were obtained from 19 scalp electrodes positioned according to the international 10–20 system (Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T3, T4, T5, T6, Fz, Cz, and Pz). Linked earlobes (A1 and A2) were used as the reference, and the ground electrode was placed at AFz. Recordings included 10 minutes with eyes closed and 10 minutes with eyes open, using a Mitsar-201 amplifier and WinEEG software. The signals were sampled at 250 Hz with a bandpass filter set between 0.16 and 70 Hz. Additionally, notch filters at 45–55 Hz and 95-105 Hz were employed to eliminate power line and harmonic noise artifacts**.**

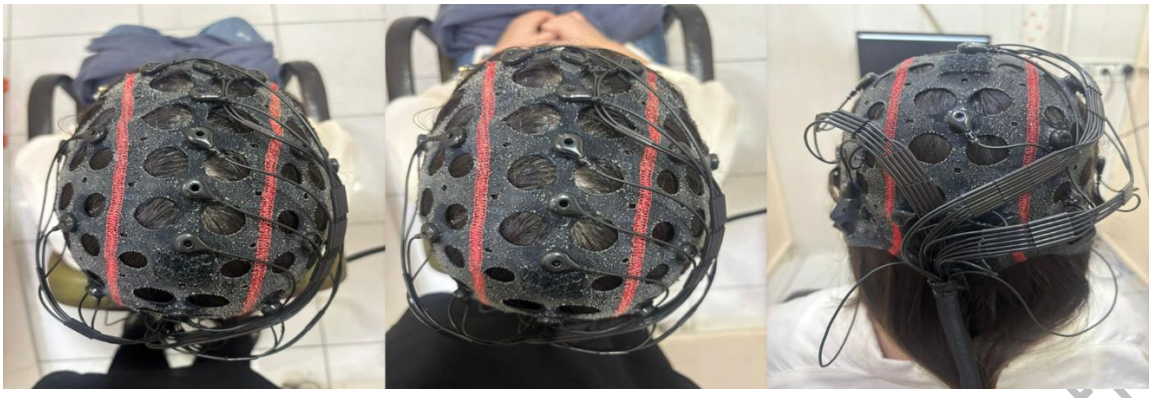

Figure 1. representation of the placement of EEG-cap during EEG recording.

QEEG extraction and data processing

Quantitative EEG (qEEG), as an advanced analytical method, aims to extract meaningful features from EEG data to provide a deeper understanding of brain dynamics. In this study, qEEG analysis was conducted using NeuroGuide software (version 3.2.1; Applied Neuroscience, Inc.). The preprocessing pipeline began with a visual inspection of the raw EEG data to remove artifacts. This process was conducted in three stages to minimize residual contamination. First, automated artifact detection algorithms were applied to identify common physiological artifacts (e.g., ocular, cardiac, and muscle activity) as well as environmental noise. Second, all recordings were manually reviewed on a patient-by-patient basis by an experienced neurologist. Finally, a secondary expert review was performed by another neurologist in collaboration with a medical physicist to ensure consistency and reduce observer bias. This multi-step procedure was designed to maximize signal integrity and minimize residual artifact influence on spectral analyses. Recordings containing electrodes with poor signal quality (defined as >10% unusable epochs or persistent high-impedance noise) were excluded from further analysis rather than interpolated, to preserve spectral integrity. This conservative approach was adopted to prioritize spectral validity over data retention and to minimize the risk of spurious frequency-specific findings. Artifact-free EEG segments were transformed from the time domain to the frequency domain using the Fast Fourier Transform (FFT) algorithm. This transformation enabled detailed spectral analysis by decomposing the EEG signals into distinct frequency bands: Delta (1.0–4.0 Hz), Theta (4.0–8.0 Hz), Alpha (8.0–12.0 Hz), Beta (12.0–25.0 Hz), and High Beta (HB) (25.0–30.0 Hz). For each participant, absolute power values derived from fast Fourier transform (FFT) analysis were converted into age-adjusted z-scores. These z-scores were then averaged within anatomically defined cortical

regions including frontal, central, temporal, parietal, and occipital to generate regional mean spectral indices for each frequency band. To identify abnormal neural activity, electrodes showing z-scores exceeding ±1.96 (i.e., beyond the 95% confidence interval based on NeuroGuide normative data) were considered statistically significant outliers.

Statistical analysis

The normality of data distribution was first assessed using the Shapiro–Wilk test. Independent sample *t*-tests were applied to compare the mean power values between the MWO and control groups, as well as between the MWA and control groups, for each frequency band (delta, theta, alpha, beta, and high-beta) and cortical region separately. For variables that did not meet the normality assumption, non-parametric alternatives were considered.

Given the number of comparisons across multiple cortical regions and frequency bands, False Discovery Rate (FDR) correction (Benjamini–Hochberg procedure) was applied to control for Type I error inflation. Both uncorrected and FDR-adjusted p-values were examined. Results with p-values less than 0.05 were initially considered statistically significant, and those that survived the FDR correction (i.e., q-values < 0.05) were deemed statistically robust. The correlation between QEEG measures (delta, theta, and alpha absolute power) and clinical variables including headache intensity, phonophobia, and photophobia were explored using the Spearman rank-order correlation test.

Sample Size and Power Calculation:

An a priori power analysis was conducted based on effect sizes reported in prior quantitative EEG studies in migraine. Previous studies has demonstrated pronounced interictal spectral differences in slow-frequency power (delta and theta bands) between migraineurs and healthy controls (Lia, Carenini et al. 1995, Bjørk, Stovner et al. 2009). Based on these findings, assuming a large effect size (Cohen's $d \geq 1.0$), a significance level of $\alpha = 0.05$, and 80% power, the estimated sample size required for between-group comparisons was approximately 14–16 participants per group. The final sample size in the present study met or exceeded this threshold, providing sufficient power to detect large interictal spectral differences across groups. These calculations were focused on primary regional spectral power comparisons. Differences of smaller magnitude may not be detectable with the current sample and warrant investigation in larger cohorts. All statistical analyses were performed using IBM SPSS Statistics version 23.0 (IBM Corp., Armonk, NY, USA), and a *p*-value < 0.05 was considered statistically significant.

Results

According to the results presented in Table 1, delta, theta, and HB amplitudes were elevated in both MWO and MWA patients compared to healthy control subjects. In the MWO group, significant increases were observed in the frontal delta, theta, HB, central HB, parietal delta, and theta bands (Table 2). However, after controlling for multiple comparisons using the False Discovery Rate (FDR) procedure, none of these differences remained statistically significant (all $q > 0.05$). Despite the loss of statistical significance after correction, the direction of change was consistent across frontal and parietal slow-wave bands, and several confidence intervals did not cross zero, indicating moderate effect magnitudes and a coherent spatial pattern of increased cortical activity in MWO patients. As shown in Figure 2, the Z-scored absolute power spectrum of QEEG illustrates the distribution of neural activity across frequency bands in an MWO patient, visually demonstrating enhanced slow-wave and high frequency components. Moreover, MWA patients demonstrated a more extensive and statistically robust pattern of spectral enhancement relative to controls. Nominally significant increases were observed in the frontal delta, theta, HB; central delta and HB; parietal delta and theta; as well as occipital delta, theta, beta, and HB bands ($p<0.05$). Importantly, after FDR correction, several posterior slow-wave alterations, particularly parietal theta and occipital delta and theta bands, remained statistically significant ($q < 0.05$), indicating stable electrophysiological differences in patients with aura.(Table 3).

Figure 3 shed light on the mean absolute power values for each frequency band across different cortical regions in the MWA, MWO, and control groups. Both migraine subtypes exhibited higher delta and theta power compared to controls, particularly in the frontal, parietal regions at the nominal level ($p<0.05$); however, occipital slow-wave increases reached statistical robustness after FDR correction only in the MWA group ($q<0.05$), highlighting a stronger posterior involvement in migraine with aura. HB power was also elevated across multiple regions in MWA patients, suggesting enhanced cortical excitability; nevertheless, these findings only in this subgroup remained significant after correction. In contrast, alpha power was reduced in the temporal region in both migraine groups, reaching nominal significance only in the MWA group ($p < 0.05$). After FDR correction, this reduction did not remain statistically significant ($q > 0.05$). Nevertheless, the consistent direction of change across groups and the QEEG topographical patterns can suggest a potential alteration in alpha activity that may be related to the subtle disturbances in cortical inhibitory mechanisms, warranting further investigation in larger cohorts. Overall, these findings reflect a pattern of increased

slow-wave and beta activity in migraine, with more spatially extensive and statistically robust alterations in individuals with aura. In Figure 4, the Z-scored absolute power spectrum of QEEG is depicted for a representative patient with MWA, illustrating the characteristic alterations in frequency band distribution.

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Table 1. Summary of the average of different frequency band amplitudes in different brain regions. The values are reported as mean ± Std. Error.

Area	Groups	delta	theta	Alpha	Beta	HB
Frontal	MWO	4.25±0.13	3.71±0.098	2.12±0.05	1.61±0.01	1.37±0.04
	MWA	4.61±0.01	4.07±0.02	1.95±1.88	2.13±0.01	1.54±0.07
	Control	3.47±0.07	2.74±0.05	2.44±0.03	1.58±0.01	0.82±0.01
Central	MWO	4.18±0.02	3.86±0.005	2.56±0.04	2.15±0.03	1.34±0.03
	MWA	4.70±0.01	3.99±0.01	2.47±0.07	2.38±0.05	1.54±0.004
	Control	4.13±0.02	3.55±0.01	2.93±0.03	1.93±0.02	0.95±0.01
Temporal	MWO	3.91±0.04	3.63±0.03	2.45±0.03	2.08±0.02	1.32±0.01
	MWA	3.96±0.06	4.04±0.01	1.81±0.09	2.03±0.07	1.4±0.07
	Control	3.80±0.03	3.33±0.02	3.27±0.02	1.74±0.01	1.13±0.01
Parietal	MWO	2.78 ±0.03	2.63±0.02	2.35±0.01	2.00±0.01	1.58±0.01
	MWA	3.31±0.04	3.11±0.02	1.22±0.03	2.19±0.04	1.77±0.044
	Control	2.50±0.2	2.33±0.01	2.03±0.01	1.76±0.01	0.98±0.01
Occipital	MWO	2.46±0.02	2.36±0.01	1.05±0.01	0.85±0.01	0.88±0.01
	MWA	2.67±0.045	2.60±0.03	0.89±0.03	1.02±0.02	0.96±0.02
	Control	2.02±0.02	1.71±0.01	1.52±0.01	0.70±0.01	0.52±0.03

Table 2. Independent sample t-test results comparing mean EEG power values between the MWO and control groups across cortical regions. Significant differences ($p < 0.05$) were mainly observed in the frontal and parietal delta, theta, and high-beta bands.

MWO	F	P-value	q-value	t	df	Mean difference	Std. Error Difference	Confidence Interval 95 %	
								Lower	Upper
Frontal Delta	6.35	0.017	0.142	2.50	33	0.42	0.17	0.08	0.76
Frontal theta	5.29	0.028	0.145	2.30	33	0.38	0.16	0.05	0.71
Frontal alpha	1.62	0.212	0.379	1.27	33	-0.11	0.09	-0.29	0.07
Frontal beta	0.65	0.425	0.506	0.81	33	0.03	0.04	-0.05	0.11
Frontal HB	17.89	0.011	0.142	2.70	33	0.25	0.09	0.07	0.43
Central Delta	0.08	0.768	0.800	0.30	33	0.01	0.03	-0.05	0.07
Central theta	2.13	0.154	0.350	1.45	33	0.10	0.07	-0.04	0.24
Central alpha	0.85	0.363	0.454	0.92	33	-0.07	0.07	-0.21	0.07
Central beta	1.18	0.284	0.426	1.10	33	0.08	0.07	-0.06	0.22
Central HB	7.75	0.029	0.145	2.25	33	0.19	0.09	0.02	0.36
Temporal Delta	1.64	0.209	0.379	1.29	33	0.07	0.06	-0.05	0.19
Temporal theta	2.44	0.127	0.318	1.55	33	0.09	0.06	-0.03	0.21
Temporal alpha	0.50	0.484	0.550	0.71	33	-0.05	0.07	-0.19	0.09

Temporal beta	3.78	0.060	0.167	2.00	3 3	0.15	0.07	0.00	0.30
Temporal HB	4.17	0.051	0.167	2.08	3 3	0.14	0.07	0.00	0.28
Parietal Delta	6.41	0.016	0.142	2.50	3 3	0.21	0.08	0.05	0.37
Parietal theta	4.30	0.046	0.167	2.10	3 3	0.18	0.09	0.00	0.36
Parietal alpha	1.15	0.290	0.426	1.09	3 3	0.06	0.06	-0.06	0.18
Parietal beta	3.08	0.212	0.379	1.27	3 3	0.08	0.06	-0.05	0.21
Parietal HB	3.95	0.055	0.167	2.03	3 3	0.30	0.15	0.00	0.60
Occipital Delta	0.43	0.515	0.560	0.66	3 3	0.15	0.22	-0.30	0.60
Occipital theta	0.87	0.357	0.454	0.93	3 3	0.22	0.24	-0.27	0.71
Occipital alpha	0.00	0.961	0.961	0.05	3 3	-0.01	0.23	-0.49	0.47
Occipital beta	1.48	0.232	0.387	1.22	3 3	0.10	0.08	-0.06	0.26
Occipital HB	0.95	0.335	0.454	0.97	3 3	0.12	0.12	-0.13	0.37

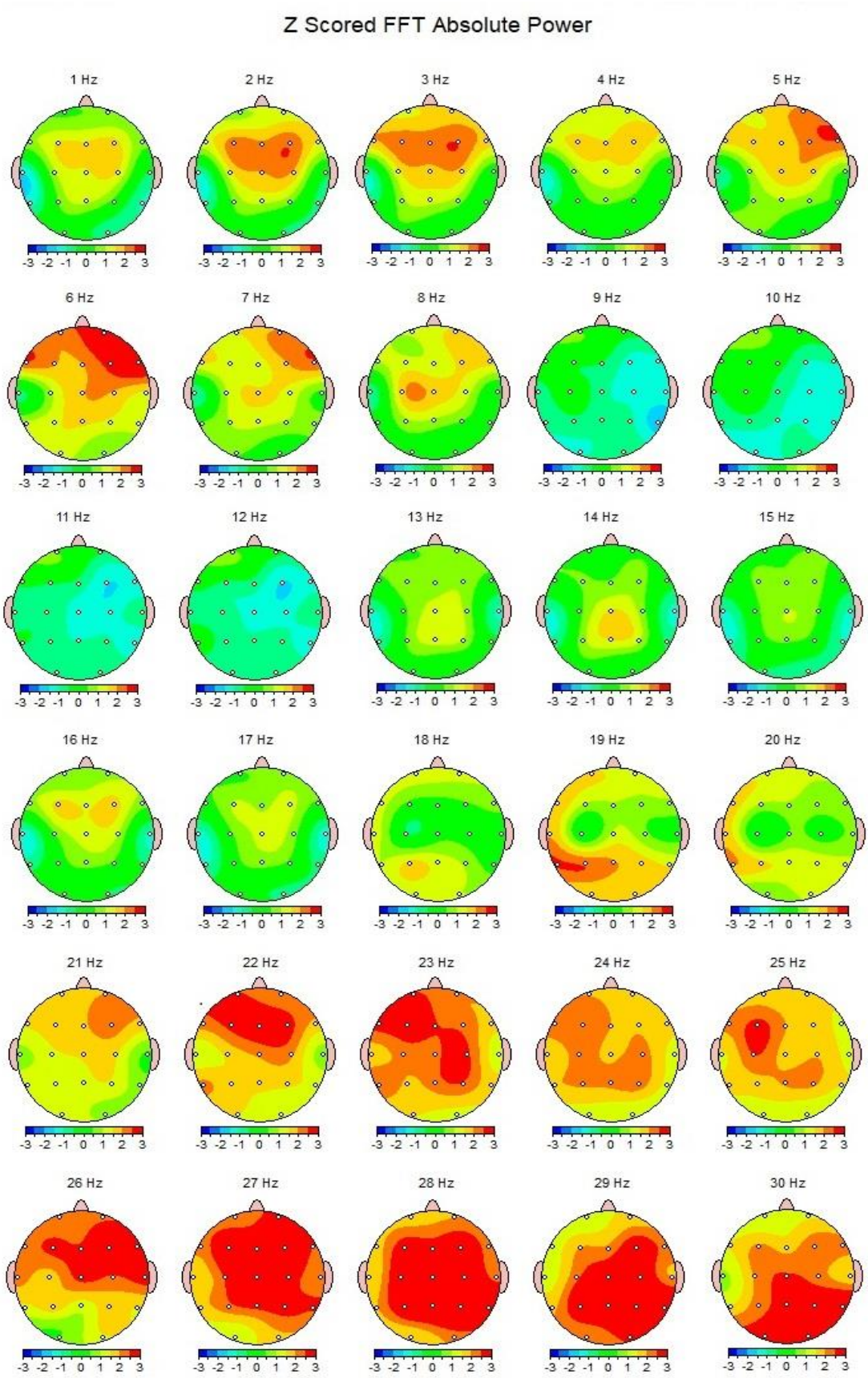

Figure 2. An example of the Z-scored absolute power spectrum of EEG across different frequency bands in an MWO patient.

Table 3. Independent sample t-test results comparing mean EEG power values between the MWA and control groups across cortical regions. Significant differences ($p < 0.05$) were mainly observed in the frontal and parietal delta, theta, and high-beta bands.

MWA	F	P-value	q-value	t	df	Mean difference	Std. Error Difference	Confidence Interval 95%	
								Lower	Upper
Frontal Delta	5.84	0.021	0.105	2.42	31	1.14	0.47	0.18	2.11
Frontal theta	6.15	0.018	0.105	2.48	31	1.33	0.53	0.23	2.41
Frontal alpha	2.95	0.095	0.173	1.74	31	0.36	0.21	-0.07	0.79
Frontal beta	3.22	0.081	0.162	1.81	31	0.56	0.31	-0.07	1.19
Frontal HB	7.02	0.012	0.075	2.65	31	0.71	0.27	0.16	1.26
Central Delta	8.11	0.008	0.050	2.85	31	0.56	0.20	0.15	0.97
Central theta	2.94	0.096	0.173	1.72	31	0.44	0.26	-0.09	0.97
Central alpha	1.13	0.296	0.329	1.03	31	-0.05	0.05	-0.14	0.05
Central beta	0.87	0.357	0.372	1.22	31	0.06	0.05	-0.04	0.15
Central HB	5.41	0.026	0.108	2.33	31	0.58	0.25	0.07	1.09
Temporal Delta	1.05	0.191	0.239	1.04	31	0.04	0.04	-0.04	0.11
Temporal theta	2.67	0.110	0.183	1.06	31	0.04	0.03	-0.03	0.10
Temporal alpha	6.89	0.013	0.075	-2.63	31	-1.46	0.56	-2.60	-0.32

Temporal beta	4.19	0.053	0.132	2.05	31	0.29	0.14	0.00	0.58
Temporal HB	0.84	0.365	0.372	1.02	31	0.03	0.03	-0.03	0.08
Parietal Delta	8.36	0.018	0.105	2.89	31	0.80	0.28	0.23	1.37
Parietal theta	14.73	0.001	0.025	3.84	31	0.78	0.20	0.38	1.18
Parietal alpha	3.12	0.060	0.132	1.95	31	-0.81	0.31	-1.66	0.04
Parietal beta	4.11	0.049	0.132	2.03	31	0.43	0.21	0.00	0.86
Parietal HB	5.97	0.020	0.1055	2.44	31	0.79	0.32	0.13	1.45
Occipital Delta	10.28	0.003	0.038	3.21	31	0.65	0.20	0.25	1.05
Occipital theta	9.88	0.004	0.038	3.14	31	0.89	0.28	0.32	1.46
Occipital alpha	3.01	0.090	0.173	1.70	31	-0.25	0.15	-0.54	0.04
Occipital beta	5.36	0.040	0.125	2.05	31	0.31	0.15	0.00	0.62
Occipital HB	2.50	0.040	0.125	2.05	31	0.36	0.17	0.02	0.70

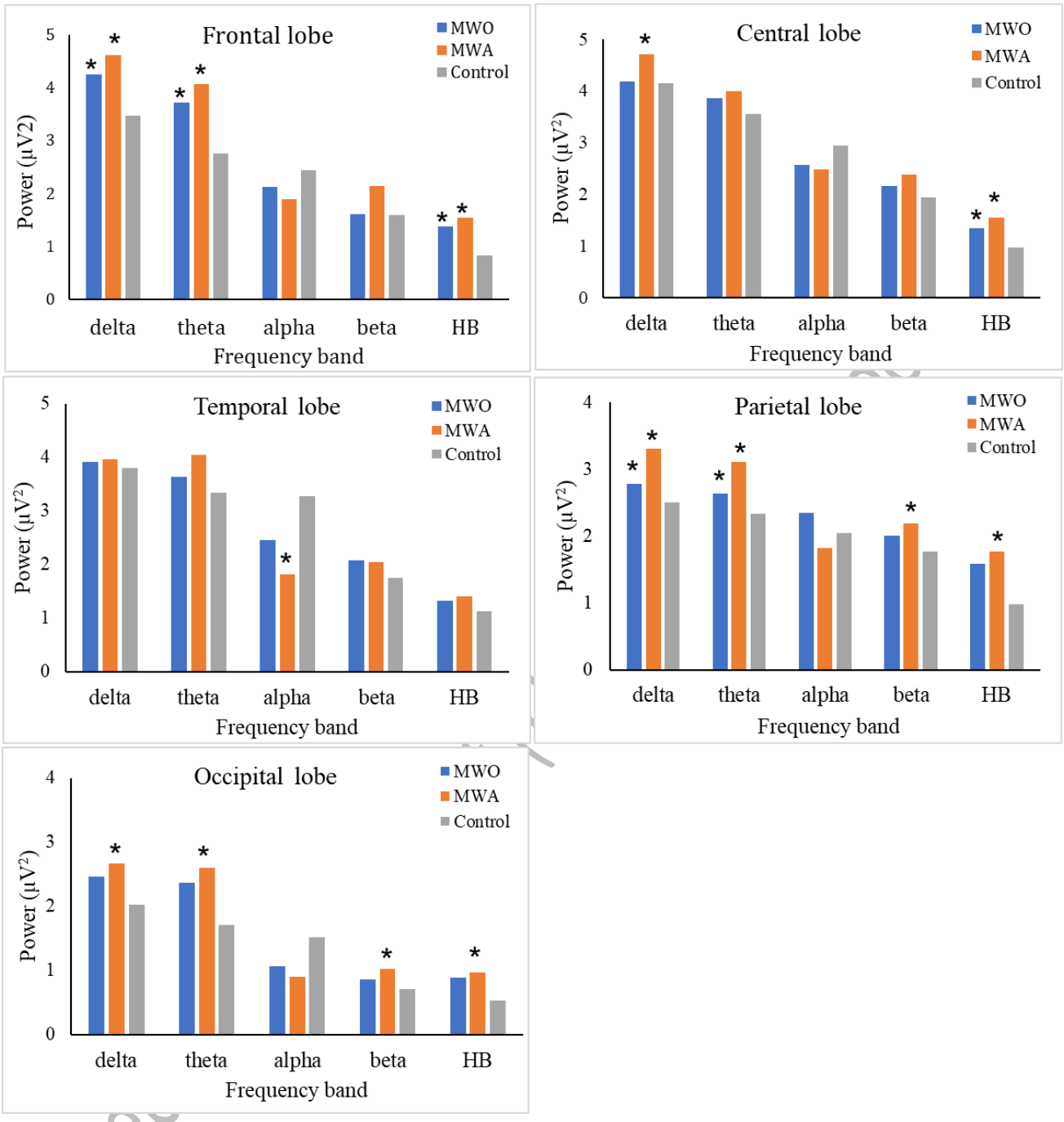

Figure 3. comparison of different frequency band powers in, a: frontal, b: central, c: temporal, d: parietal, and e: occipital lobes.

According to the Spearman rank-order correlation test, as shown in Table 4, in the MWA group, delta power showed strong positive correlations with headache intensity across all examined cortical regions including frontocentral (r = 0.866), temporal (r = 0.803), and parieto-occipital (r = 0.858). Similarly, theta activity exhibited a robust association with headache intensity (r = 0.846–0.874). In contrast, alpha power demonstrated a significant negative correlation with

photophobia in posterior regions (r = –0.803), suggesting that reduced alpha activity may be associated with greater visual sensitivity. Beta and HB power in the parieto-occipital region were also positively correlated with photophobia (r = 0.820 and r = 0.851 respectively). Similarly, in the MWO group, delta power was strongly correlated with headache intensity in the frontocentral (r = 0.836), temporal (r = 0.832), and parieto-occipital (r = 0.884) areas. Beta activity in posterior regions also correlated positively with photophobia (r = 0.860), and high-beta activity demonstrated robust associations with both photophobia (r = 0.886) and headache severity (r = 0.724) (Table 4).

Table 4. Correlation of EEG frequency bands with photophobia, phonophobia, and headache intensity across different brain regions in MWA and MWO groups.

	MWO			MWA		
	frontocentral	**temporal**	**parietooccipital**	**frontocentral**	**temporal**	**parietooccipital**
delta& photophobia	0.499	0.296	0.702	0.630	0.416	0.760
delta& phonophobia	-0.059	0.754	0.157	-0.186	0.762	-0.051
delta& headache intensity	0.866	0.803	0.858	0.836	0.832	0.884
theta& photophobia	0.382	0.396	0.810	0.376	0.218	0.72
theta& phonophobia	0.044	0.464	0.078	-0.065	0.778	0.079
theta& headache intensity	0.8468	0.811	0.839	0.840	0.8460	0.874
alpha& photophobia	-0.261	-0.313	-0.79	-0.649	-0.217	-0.803
alpha& phonophobia	-0.282	-0.825	-0.055	-0.227	-0.868	-0.019
alpha& headache intensity	-0.307	-0.374	-0.668	-0.7201	-0.099	-0.747
beta& photophobia	0.442	0.130	0.82	0.582	0.522	0.86
beta& phonophobia	0.160	0.702	-0.117	0.220	0.711	-0.047
beta& headache intensity	0.599	0.306	0.541	0.638	0.425	0.684
HB & photophobia	0.604	0.081	0.851	0.617	0.110	0.886
HB & phonophobia	0.120	0.68	0.166	-0.180	0.818	0.090
HB & headache intensity	0.526	0.174	0.718	0.514	0.174	0.724

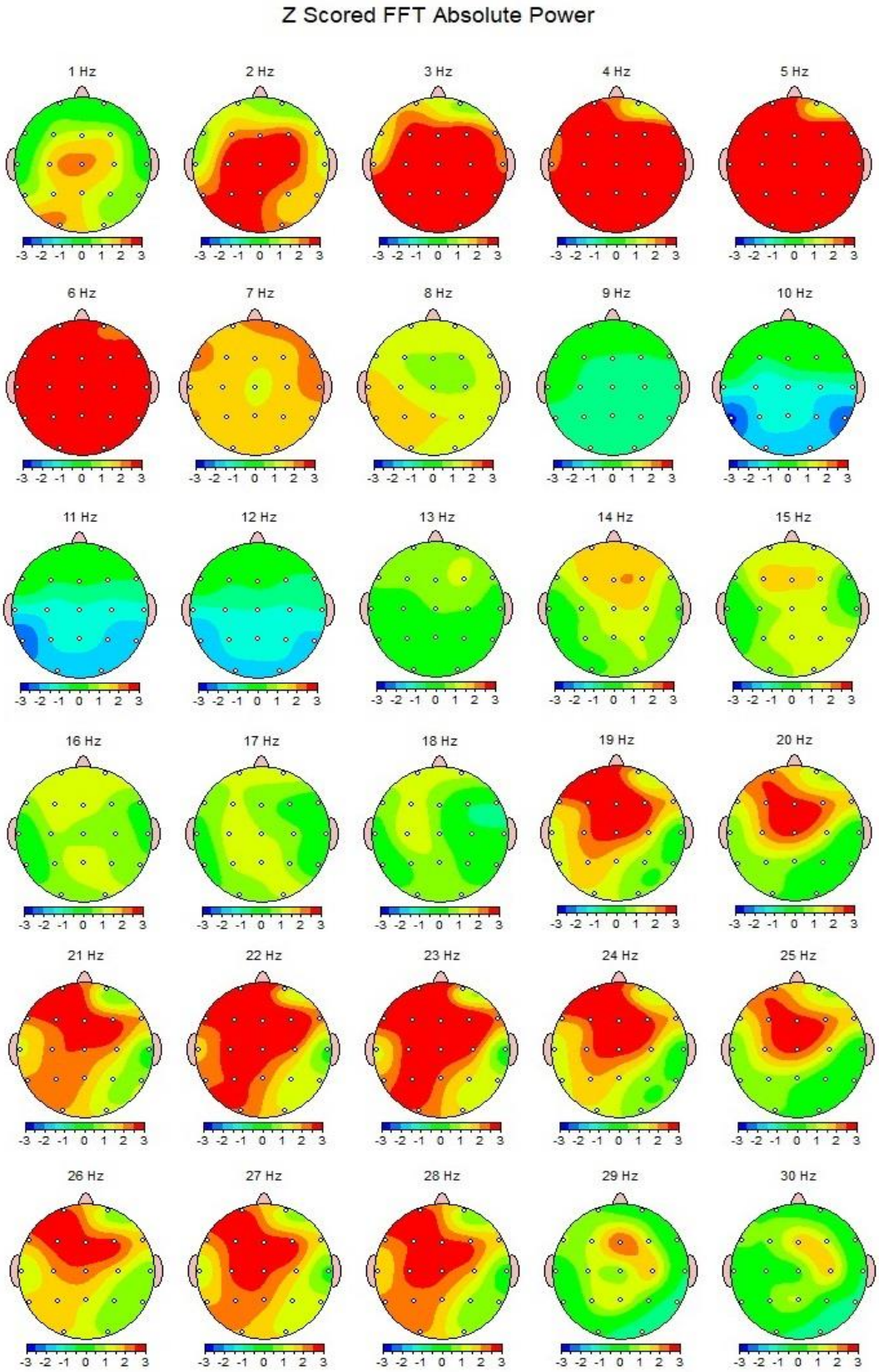

Figure 4. An example of the Z-scored absolute power spectrum of EEG across different frequency bands in an MWA patient

Discussion

The primary objective of this study was to investigate QEEG patterns in migraine patients and to compare them with healthy controls to characterize neurophysiological patterns potentially related to the underlying pathophysiology and clinical manifestations of migraine. Our results demonstrate that migraine patients exhibit increased absolute power across multiple EEG frequency bands, with the most pronounced elevations in the beta band. These increases were observed across all cortical regions and were more robust and spatially extensive in patients with aura compared to those without aura. These findings align with prior research proposing that heightened cortical excitability may contribute to migraine pathophysiology, particularly in relation to aura-related mechanisms (Aurora and Wilkinson 2007, Noseda and Burstein 2013, Charles 2018). QEEG provides a non-invasive method for characterizing neuronal and cortical excitability patterns by analyzing brain activity through diverse electrophysiological parameters. Among these, elevated absolute power has been proposed as a potential electrophysiological indicator of cortical hyperexcitability and neuronal dysregulation in migraine, although its clinical utility for objective monitoring remains to be established (Minnerly, Shokry et al. 2021). Another noteworthy observation in our study was the elevated theta power across multiple brain regions, particularly within the temporal and parieto-occipital lobes. This increase was more pronounced in MWA patients, consistent with prior reports of heightened theta activity in both migraine subtypes (Lia, Carenini et al. 1995, Hughes and Crunelli 2005, Bjørk, Stovner et al. 2009). Theta oscillations are implicated in sensory processing, cortical network regulation, and thalamocortical communication, suggesting that abnormal theta activity may be involved in mechanisms related to sensory hypersensitivity and pain perception in migraine. Our correlation analysis demonstrated that in both MWA and MWO groups, enhanced slow-wave (delta and theta) activity might be associated with headache intensity, consistent with the involvement of pain-related cortical and thalamocortical networks, whereas decreased alpha and increased high-frequency (beta and high-beta) activities, particularly in posterior regions, are associated with visual hypersensitivity and photophobia. Delta power was considerably elevated in frontocentral and parieto-occipital regions in MWA patients, with strong correlations to headache severity. These findings are consistent with Bjork et al. (2011) results indicating increased theta activity and interictal cortical dysfunction are closely linked to sensory hypersensitivity and photophobia in migraine patients, based on QEEG frequency analysis and visual evoked potentials. This supports the hypothesis that abnormal theta oscillations may serve as a neurophysiological substrate for sensory symptoms in migraine (Bjørk, Stovner et al. 2011). Theta activity is typically

associated with the hippocampal-limbic circuitry and is known to play a key role in emotional regulation, memory formation, and cognitive processing. In the healthy brain, theta waves are most commonly linked to relaxed wakefulness and drowsiness. However, elevated theta power in awake individuals such as observed in migraine patients suggests cortical network dysfunction, particularly in regions involved in sensory integration, pain modulation, and attentional control. This abnormal increase in theta activity may be interpreted as consistent with thalamocortical dysrhythmia model, which arises from decreased activity of thalamic nuclei and impaired thalamocortical inhibition. Such dysregulation has been proposed as a potential mechanism underlying sensory disintegration and heightened cortical sensitivity to external stimuli, potentially manifesting as photophobia and phonophobia in migraine. Another important observation in our study was the significant elevation of delta power in the frontocentral and parieto-occipital regions during the interictal period in MWA patients. Correlation analyses further revealed that this increase in delta activity was strongly associated with headache intensity. These findings are consistent with previous hypotheses suggesting that repetitive activation of trigeminovascular pathways may be associated with long-term functional alterations in pain-processing networks. Such changes may manifest as increased slow-wave (delta) activity, which correlates with the severity of migraine symptoms (Bigal, Ferrari et al. 2009). Also, the results of some previous studies show that brain white matter lesions and cortical changes obtained in neuroimaging studies are related to the severity of headache attacks in migraine patients (Kruit, van Buchem et al. 2004, Valfrè, Rainero et al. 2008). Although increased delta activity has been reported in association with structural or metabolic alterations in several neurological disorders, scalp EEG findings in awake individuals provide indirect evidence and should not be interpreted as direct markers of structural integrity. Low-frequency delta oscillations, particularly around 1 Hz, are closely linked to thalamocortical communication pathways. Pathological delta activity has been associated with metabolic or structural brain abnormalities, with focal delta often emerging in cases involving subcortical white matter, thalamic, or brainstem lesions. In migraine patients, elevated delta power are thought to be involved in cortical dysregulation and hyperexcitability, as well as impaired functioning of pain-related thalamocortical circuits.

Alpha-band reductions were observed at a nominal level in MWA and were not statistically significant in MWO; none of the temporal or parieto-occipital alpha reductions survived FDR correction. Furthermore, our correlation analysis revealed a significant negative association between alpha power in the temporal and parieto-occipital regions and the presence of

photophobia and phonophobia in migraine patients, particularly those with aura, when compared to controls. Nevertheless, the consistent direction of change and QEEG topographical patterns indicate potential impairment in cortical inhibitory mechanisms, particularly in visual cortical networks, which may contribute to heightened photophobia and phonophobia in migraine patients (Hughes and Crunelli 2005, Roux, Wibral et al. 2013, Lange, Keil et al. 2014). Specifically, the dynamic interaction between the thalamic reticular nucleus and lateral thalamic nuclei is thought to be central to cortical alpha oscillations. Disruption in this thalamocortical interplay has been hypothesized to contribute to impaired sensory gating and increased cortical sensitivity to visual and auditory stimuli in migraine (Hughes and Crunelli 2005). Roux et al. (2013) demonstrated that alpha oscillations are critically involved in the functional inhibition of the sensory cortex, serving to regulate the influx of sensory stimuli by suppressing cortical excitability and reducing the firing rates of cortical neurons (Roux, Wibral et al. 2013). Cortical excitability is inversely related to the amplitude of alpha oscillations, which play a critical role in modulating cortical responsiveness and are essential for the accurate perception and filtering of sensory stimuli (O'Hare, Tarasi et al. 2023). Emerging evidence suggests that neural oscillatory patterns are disrupted in migraine patients, with thalamic dysrhythmia proposed as a key mechanism underlying the imbalance between cortical excitation and inhibition (De Tommaso, Ambrosini et al. 2014). In migraine patients, alpha oscillations have been proposed to engage inhibitory interneuronal networks that modulate cortical excitability, thereby suppressing excessive visual sensory input. In this regard, reduced alpha-band activity has been associated with alterations in inhibitory control mechanisms and may contribute to increased sensory cortical responsiveness, although such interpretations remain inferential in scalp EEG studies (Lange, Keil et al. 2014). These findings are consistent with our correlation analysis, demonstrating a significant relationship between alpha power reduction and the severity of photophobia in migraine patients (Lange, Keil et al. 2014). In summary, reduced alpha oscillations may be associated with increased visual cortical responsiveness and heightened sensitivity to visual stimuli in migraine patients. These findings provide exploratory correlational insight into mechanisms that warrant further experimental validation. Overall, our findings indicate that MWA patients exhibit more robust and spatially extensive cortical oscillatory alterations compared to MWO patients, while the latter shows consistent but subtler electrophysiological trends. These results are consistent with theoretical models proposing thalamocortical dysrhythmia and altered cortical excitability as contributing mechanisms in migraine pathophysiology.

Limitations of the study

The findings of this study should be interpreted with caution due to the use of multiple statistical comparisons across cortical regions and frequency bands. After applying FDR correction, several nominally significant effects, particularly within the MWO group and in alpha-band reductions, did not remain statistically robust. This indicates that some observed differences may represent subtle trends rather than stable effects. Although the overall patterns were physiologically coherent and consistent with QEEG topographical findings, these results should be considered exploratory and require confirmation in independent datasets.

Another important limitation of the present study is the relatively modest sample size. The a priori power analysis was based on previously reported large interictal spectral differences in migraine, particularly in slow-frequency bands, suggesting adequate power to detect large effect sizes. However, the study may have been underpowered to reliably detect small-to-moderate oscillatory alterations. Consequently, while the significant findings likely reflect robust interictal neurophysiological differences, more subtle spectral changes may have gone undetected. Future studies with larger cohorts will be essential to further clarify the magnitude and spatial specificity of these oscillatory alterations.

Although patients were instructed to discontinue migraine-related medications at least 48 hours prior to EEG recording, and recordings were obtained during the interictal phase, residual confounding effects related to prior medication exposure or variability in proximity to subsequent migraine attacks cannot be entirely excluded. Differences in individual pharmacokinetics, subclinical pre-ictal changes, or variability in attack cycles may have influenced oscillatory activity. Future longitudinal studies with stricter monitoring of medication washout periods and precise tracking of attack timing would help to further distinguish state-dependent from trait-related electrophysiological alterations.

Conclusion

This study demonstrates distinct QEEG alterations in migraine patients, particularly those with aura, characterized by elevated absolute power in theta, delta, and high beta bands, alongside a significant reduction in alpha power in key cortical regions. These neurophysiological changes can be associated with clinical symptoms such as photophobia, phonophobia, and headache intensity, suggesting the involvement of cortical hyperexcitability and

thalamocortical dysrhythmia in migraine pathophysiology. While the observed increases in theta and delta activities likely reflect disruptions in sensory integration and pain processing networks, the reduction in alpha power points to impaired inhibitory control, contributing to heightened cortical excitability and sensory hypersensitivity. Collectively, these findings not only enhance our understanding of migraine's underlying neural mechanisms but also suggest that QEEG biomarkers, particularly alpha and theta oscillations, could serve as valuable targets for monitoring disease progression and developing novel therapeutic interventions.

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Data Availability

All datasets used and /or analyzed in this study are not publicly available, but are available from the corresponding author on reasonable request.

Ethics Approval

The study protocol was approved by the Ethics Committee of Ahvaz Jundishapur University of Medical Sciences (AJUMS) (IR.AJUMS.REC.1404.223) and followed the ethical principles of the Declaration of Helsinki. An informed consent was also obtained from all patients and their legal guardians before QEEG test.

Declaration

Competing Interests

The authors have no relevant financial or non-financial interests to disclose.

Consent to participate

Informed consent was obtained from all individual participants included in the study.

Consent to publish

The authors affirm that human research participants provided informed consent for publication of the images in the study Figure(s).

Author Contribution

Conceptualization and methodology were made by Siamak Askari and Mostafa Jalilifar. Software usage by Mostafa Jalilifar, validation of results by Jafar Fatahi-Asl and Shahram Nasiri. Formal analyses were completed by Mostafa Jalilifar and Siamak Askari, investigations made by Jafar Fatahi-Asl, resources arranged by Siamak Askari and Shahram Nasiri. The manuscript was written by Mostafa Jalilifar and reviewed and edited by Siamak Askari. Data were visualized by Jafar Fatahi-Asl and Shahram Nasiri. The project administrator was Mostafa Jalilifar and he was responsible for funding acquisition.

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