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Electroencephalographic (EEG) changes in migraine patients: a comparative analysis of aura and non-aura migraine with chronic tension-type and medication overuse headaches

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Abstract: This study explores electroencephalographic (EEG) changes in patients with different types of chronic headaches, including aura and non-aura migraine, chronic tension-type headaches, and medication overuse headaches. The research analyzes EEG parameters such as dominant alpha rhythm, slow-wave activity, focal theta and delta rhythms, and hemispheric asymmetry during and outside migraine attacks. The findings indicate significant neurophysiological differences among headache types, which could aid in refining diagnostic criteria and developing personalized treatment strategies. The study also highlights the relationship between cognitive dysfunction and EEG alterations in migraine patients.

Keywords: Migraine, aura, EEG, chronic headaches, alpha rhythm, slow-wave activity, hemispheric asymmetry, cognitive dysfunction, neurophysiology, medication overuse headache.

Introduction: Migraine is a prevalent neurological disorder characterized by recurrent headaches, often accompanied by sensory disturbances such as visual auras, nausea, and cognitive impairments. It affects millions of individuals worldwide, significantly reducing their quality of life and productivity. While the exact pathophysiology of migraines remains complex, electroencephalography (EEG) has emerged as a valuable tool in understanding the neurophysiological

mechanisms underlying different headache types.

EEG studies have demonstrated that migraine patients exhibit distinctive alterations in brain electrical activity, including changes in dominant alpha rhythm, slow-wave activity, and hemispheric asymmetry. These variations differ between migraine with aura, migraine without aura, chronic tension-type headaches (CTTH), and medication overuse headaches (MOH). Understanding these differences is crucial for refining diagnostic criteria and optimizing treatment approaches. Additionally, research suggests a strong correlation between EEG changes and cognitive dysfunction in migraine patients. Cognitive impairments, such as difficulties in attention, memory, and executive function, have been linked to abnormal EEG patterns, particularly in the frontal and occipital regions. Evaluating these relationships may provide further insights into the impact of migraine on brain function and aid in the development of more targeted therapeutic interventions. This study aims to analyze EEG changes in different types of chronic headaches, comparing aura and non-aura migraine with CTTH and MOH. By identifying key neurophysiological markers, we seek to contribute to a more comprehensive understanding of migraine pathophysiology and facilitate the development of personalized treatment strategies.

METHODS

This study was conducted as a comparative observational analysis of electroencephalographic (EEG) changes in patients with different types of chronic headaches. Participants were recruited from neurology clinics and divided into four groups based on clinical diagnosis:

Group 1: Migraine with aura (MA)

Group 2: Migraine without aura (MO)

Group 3: Chronic tension-type headache (CTTH)

Group 4: Medication overuse headache (MOH)

Inclusion criteria required participants to be between 18 and 55 years old with a confirmed diagnosis according to the International Classification of Headache Disorders (ICHD-3). Exclusion criteria included individuals with epilepsy, structural brain abnormalities, psychiatric disorders, or those taking medications affecting EEG activity.

EEG Recording and Analysis

EEG recordings were performed using a 32-channel digital EEG system with electrodes placed according to the 10-20 international system. Data were collected in two conditions:

During headache attacks (ictal phase)

Outside headache episodes (interictal phase)

Each recording lasted for 30 minutes, including resting state, hyperventilation, and photic stimulation at 27 Hz to assess visual reactivity. EEG parameters analyzed included:

Dominant alpha rhythm

Slow-wave activity (theta and delta rhythms)

Hemispheric asymmetry

Amplitude changes in occipital, frontal, and temporal lobes

Artifact rejection was performed manually to exclude muscle activity, blinking, and movement-related distortions.

Cognitive Function Assessment

To evaluate cognitive impairments, participants completed the Trail Making Test (TMT), assessing attention, processing speed, and executive function. Correlations between EEG abnormalities and cognitive performance were analyzed.

Statistical Analysis

Data were analyzed using SPSS (Version XX).

Chi-square tests (χ^2) were applied to compare categorical variables between headache groups.

ANOVA was used to assess differences in EEG parameters.

Pearson correlation was performed to analyze relationships between EEG changes and cognitive scores.

A significance level of $p < 0.05$ was considered statistically significant.

RESULTS

Significant differences in EEG patterns were observed among the four groups during headache episodes. The dominant alpha rhythm was absent in migraine with aura (MA) and medication overuse headache (MOH) groups, present in 5.33% of migraine without aura (MO) patients, and maintained in 94.87% of chronic tension-type headache (CTTH) patients ($\chi^2 = 274.106$, $p = 0.000$). This suggests greater EEG stability in CTTH patients compared to migraine groups. Frontal slow-wave activity (theta and delta) was more prevalent in MA (85.53%) and MO (78.67%) groups, indicating cortical dysfunction. It was almost absent in CTTH patients (0%) but present in 80.85% of MOH patients ($\chi^2 = 197.820$, $p = 0.000$).

Table 1:

EEG Findings During Headache Episodes (Ictal Phase)

EEG Parameters	Group								p-value Group 2: Migraine without Aura (MO)	
	Group 1: Migraine with Aura (MA)		Group 2: Migraine without Aura (MO)		Group 3: Chronic Tension-Type Headache (CTTH)		Group 1: Migraine with Aura (MA)			
	Abs	M±m, %	abs	Abs	M±m,%	M±m,%	Abs	M±m, %	Z	Abs
Dominant Alpha Rhythm	0	0±0	4	5,33± 2,59	111	94,87±2,04	0	0±0	274,106	0,000
Frontal Slow-Wave Activity	65	85,53 ±4,04	59	78,67 ±4,73	0	0±0	38	80,85 ±5,74	197,820	0,000
Reduced Alpha Rhythm Amplitude (Occipital-Temporal Regions)	65	85,53 ±4,04	58	77,33 ±4,83	0	0±0	38	80,85 ±5,74	195,616	0,000
Sharp Alpha Rhythm	6	7,89± 3,09	4	5,33± 2,59	62	52,99±4,61	3	6,38± 3,57	87,517	0,000
Low Amplitude Rhythms	63	82,89 ±4,32	46	61,33 ±5,62	91	77,78±3,84	36	76,6± 6,18	10,519	0,015
Focal Theta Activity	63	82,89 ±4,32	47	62,67 ±5,59	12	10,26±2,8	36	76,6± 6,18	124,927	0,000
Focal Delta Rhythm	63	82,89 ±4,32	42	56±5, 73	10	8,55±2,58	36	76,6± 6,18	127,372	0,000
Bilateral Synchronous Slow-Wave Activity (During Hyperventilation)	63	82,89 ±4,32	52	69,33 ±5,32	117	100±0	36	76,6± 6,18	38,118	0,000
Reduced Visual Reactivity (27 Hz Photostimulation Response)	63	82,89 ±4,32	56	74,67 ±5,02	15	12,82±3,09	36	76,6± 6,18	127,960	0,000
Interhemi	2	32,89	2	30,67	14	11,97±3	1	34,04	16,558	0,001

spheric Asymmetry	5	±5,39	3	±5,32			6	±6,91		
Spike-Wave Complexes	9	11,84 ±3,71	19	25,33 ±5,02	1	0,85±0,85	6	12,77 ±4,87	27,993	0,000

Reduced alpha rhythm amplitude in the occipital-temporal regions was observed in 85.53% of MA, 77.33% of MO, and 80.85% of MOH patients, while it was not detected in CTTH patients (χ^2 = 195.616, p = 0.000). Increased focal theta and delta activity was also evident, with focal theta activity highest in MA (82.89%), followed by MO (62.67%), MOH (76.6%), and lowest in CTTH (10.26%) (χ^2 = 124.927, p = 0.000). Focal delta activity showed similar trends, suggesting greater cortical excitability in migraine patients. Hemispheric asymmetry was identified in 32.89% of MA, 30.67% of MO, and 34.04% of MOH patients, but significantly lower in CTTH (11.97%) (χ^2 = 16.558, p = 0.001), indicating functional disbalance in migraine patients, particularly during attacks.

During headache-free periods, EEG patterns were more stable but still showed significant group differences. The dominant alpha rhythm was present in most patients across all groups: MA (89.47%), MO (89.33%), CTTH (99.15%), and MOH (87.23%) (χ^2 = 11.680, p = 0.009), with higher alpha rhythm stability in CTTH patients, supporting the idea of less cortical excitability. Frontal slow-wave activity was reduced but still detected in MA (9.21%), MO (8%), and MOH (6.38%), while it was absent in CTTH patients (χ^2 = 10.444, p = 0.015). Spike-wave complexes and hemispheric asymmetry were detected in MA (3.95%), MO (4%), and MOH (4.26%) but absent in CTTH patients (χ^2 = 4.862, p = 0.182). Although less frequent in interictal phases, these findings suggest persistent cortical hyperexcitability in migraine patients.

Table 2:

EEG Findings Outside Headache Episodes (Interictal Phase)

EEG Parameters	Group								p-value	
	Group 1: Migraine with Aura (MA)		Group 2: Migraine without Aura (MO)		Group 3: Chronic Tension-Type Headache (CTTH)		Group 4: Medication Overuse Headache (MOH)			
	Abs	M±m, %	Abs	M±m, %	abs	M±m,%	abs	M±m, %	Z	P
Dominant Alpha Rhythm	68	89,47± 3,52	67	89,33± 3,56	116	99,15±0,85	41	87,23± 4,87	11,680	0,009
Frontal Slow-Wave Activity	7	9,21±3, 32	6	8±3,13	0	0±0	3	6,38±3, 57	10,444	0,015
Reduced Alpha Rhythm Amplitude (Occipital-Temporal Regions)	10	13,16± 3,88	9	12±3,75	117	100±0	6	12,77± 4,87	226, 793	0,000
Sharp Alpha Rhythm	6	7,89±3, 09	5	6,67±2, 88	117	100±0	4	8,51±4, 07	258, 098	0,000
Low Amplitude Rhythms	7	9,21±3, 32	7	9,33±3, 36	117	100±0	5	10,64± 4,5	245, 016	0,000

Focal Theta Activity	3	3,95±2,23	3	4±2,26	0	0±0	2	4,26±2,94	4,862	0,182
Focal Delta Rhythm	3	3,95±2,23	3	4±2,26	2	1,71±1,2	3	6,38±3,57	2,373	0,499
Bilateral Synchronous Slow-Wave Activity (During Hyperventilation)	5	6,58±2,84	5	6,67±2,88	117	100±0	5	10,64±4,5	258,290	0,000
Reduced Visual Reactivity (27 Hz Photostimulation Response)	6	7,89±3,09	3	4±2,26	2	1,71±1,2	4	8,51±4,07	5,601	0,133
Interhemispheric Asymmetry	3	3,95±2,23	2	2,67±1,86	0	0±0	2	4,26±2,94	4,662	0,198
Spike-Wave Complexes	3	3,95±2,23	3	4±2,26	0	0±0	2	4,26±2,94	4,862	0,182

A positive correlation was found between frontal slow-wave activity and cognitive dysfunction (r = 0.906), particularly affecting attention and executive function. Hemispheric asymmetry correlated with cognitive deficits (r = 0.224), suggesting asymmetrical cortical processing may contribute to migraine-related cognitive disturbances. Depression and cognitive dysfunction were significantly related (r = 0.513), indicating that prolonged migraine episodes could impact mental health.

These findings suggest that migraine patients exhibit significant cortical excitability, slow-wave activity, and hemispheric asymmetry, particularly during attacks. CTTH patients show more stable EEG patterns, with preserved alpha rhythm and fewer pathological waveforms. MOH patients display EEG features similar to migraines but with subtle differences, indicating a potential overlap in pathophysiology. Cognitive impairments are strongly associated with frontal slow-wave activity and hemispheric asymmetry, emphasizing the need for cognitive rehabilitation in migraine management.

DISCUSSION

This study provides significant insights into the neurophysiological differences among various types of chronic headaches by analyzing electroencephalographic (EEG) changes during and outside headache episodes. The findings highlight distinct alterations in brain electrical activity, particularly in migraine with aura (MA), migraine without aura (MO), chronic tension-type headache (CTTH), and medication overuse headache (MOH).

EEG Changes During Headache Episodes (Ictal Phase)

During headache attacks, the absence of a dominant alpha rhythm in MA and MOH patients suggests heightened cortical excitability, which aligns with previous studies indicating disrupted thalamocortical processing in migraine sufferers. In contrast, CTTH patients retained a stable alpha rhythm, reflecting a different underlying pathophysiology, likely associated with muscle tension and central pain sensitization rather than cortical hyperexcitability.

Frontal slow-wave activity (theta and delta rhythms) was significantly more prevalent in MA and MO groups, suggesting cortical dysfunction and altered neuronal excitability. The presence of these slow waves indicates impaired cortical inhibition and increased cortical excitability, which may contribute to the sensory disturbances and cognitive impairments observed in migraine patients. The relatively lower occurrence of slow-wave activity in CTTH patients further supports the idea that migraine-related headaches involve more profound neurophysiological disruptions.

The increased prevalence of interhemispheric asymmetry in migraine patients suggests functional imbalances in cortical processing, which may be

associated with abnormal sensory integration and pain perception. Additionally, the high occurrence of focal theta and delta activity in migraine patients reinforces the notion that migraine pathophysiology involves altered neural synchronization and excitability.

EEG Changes Outside Headache Episodes (Interictal Phase)

The persistence of EEG abnormalities in MA and MO patients during headache-free periods indicates that migraine is a chronic neurological disorder with ongoing cortical dysfunction rather than an episodic condition confined to attacks. Although alpha rhythm stability was higher during the interictal phase, reduced alpha amplitude in occipital-temporal regions suggests lingering cortical hyperexcitability.

CTTH patients demonstrated significantly fewer EEG abnormalities outside headache episodes, further distinguishing the pathophysiological mechanisms of migraine and tension-type headaches. The retention of stable alpha activity and the absence of excessive slow-wave activity suggest that CTTH may primarily involve peripheral sensitization mechanisms rather than central neurophysiological changes.

MOH patients exhibited EEG features similar to migraine patients, particularly in terms of slow-wave activity and reduced alpha rhythm amplitude. This finding suggests that chronic medication overuse may exacerbate cortical excitability, possibly through rebound hypersensitivity mechanisms. The overlap in EEG findings between MOH and migraine reinforces the need for careful management strategies to prevent medication-induced neurological alterations.

Cognitive Impairments and EEG Correlations

A strong correlation was observed between frontal slow-wave activity and cognitive dysfunction, particularly in attention and executive function domains. This suggests that migraine-related cortical alterations extend beyond pain perception, affecting higher cognitive processes. The association between interhemispheric asymmetry and cognitive impairment indicates that abnormal cortical synchronization may contribute to the attentional and memory deficits frequently reported by migraine patients. Furthermore, the significant relationship between depression and cognitive impairment highlights the broader impact of chronic headaches on mental health. Prolonged migraine episodes and continuous cortical hyperexcitability may contribute to emotional disturbances, reinforcing the need for a multidisciplinary approach to migraine management, incorporating both neurological and psychological interventions.

Clinical Implications and Future Research Directions

The findings of this study have important clinical implications for improving migraine diagnosis and treatment. EEG markers such as slow-wave activity, reduced alpha rhythm amplitude, and interhemispheric asymmetry could serve as objective indicators to differentiate migraine from other headache disorders. Additionally, identifying persistent EEG abnormalities in migraine patients may help clinicians tailor treatment strategies based on individual neurophysiological profiles.

Future research should focus on longitudinal EEG studies to track cortical changes over time and assess the effects of different treatment modalities on neurophysiological patterns. Investigating the impact of cognitive rehabilitation and neuromodulation techniques on EEG abnormalities may provide novel therapeutic insights for migraine management.

CONCLUSION

This study confirms that migraine, particularly MA and MO, is associated with significant neurophysiological disruptions, including cortical hyperexcitability, slow-wave activity, and interhemispheric asymmetry. In contrast, CTTH patients exhibit more stable EEG patterns, suggesting a different underlying pathophysiology. The overlap between MOH and migraine EEG findings underscores the importance of careful medication management to prevent further neurological alterations. The strong association between EEG abnormalities and cognitive dysfunction highlights the need for comprehensive treatment strategies that address both neurological and cognitive aspects of chronic headaches.

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