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Quantitative Electroencephalographic Analysis in Women with Migraine during the Luteal Phase

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Abstract: Migraine is a common, headache disorder characterized by recurrent episodes of headache often associated with nausea, vomiting, photophobia, and phonophobia. Prior to puberty, boys and girls are equally affected. Female preponderance emerges after puberty. Migraine pathophysiology is not fully understood, and although the hormonal effect of estrogen is significant, it is not clear how hormonal phases affect brain excitability and EEG patterns in women with migraine. The objective of this research was to study the effect of migraine on the resting-state EEG activity of women during the luteal phase. This work compares electroencephalographic (EEG) absolute power in different frequency bands and scalp areas between young women who suffer from migraine and had a migraine attack within 24 h prior to EEG recording (experimental) and ten age-matched young healthy women (controls), all with normal menstrual cycles. For women with migraine, we found a significant decrease/increase in alpha power in the occipitoparietal/frontocentral area, significant decrease in beta power for all areas, significant decrease in delta power in the temporal area, and significant decrease in theta power in the frontocentral and occipitoparietal area. We concluded that women with migraine have a distinct electroencephalographic pattern during the luteal phase in comparison with control women. A possible explanation might be an intermittent rhythmic activity linked to pain.

Keywords: EEG; migraine; luteal phase; absolute power

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1. Introduction

Migraine is a form of neurovascular headache [1], with a high incidence (>12%). Predominantly women (3:1 compared with men) are affected and intensity is variable [2]. Migraine is a public health problem, and it is also one of the main causes of incapacity for work because fifty percent of migraine patients interrupt their daily activities due to attacks and most of them require rest at home in dark places [3].

Migraine symptoms negatively affect quality of life as well as academic and work performance and limit the realization of daily activities [4]. In general, it is defined as an episodic attack of intense, pulsating, and unilateral headache that may last from 4 to 72 h ranging from once a week to once a year [5]. The age group with the highest prevalence of migraine is between 25 and 55 years old [6], however, there are reports that indicate that the number of episodes decreases as the age of the patients increases, or at least the prevalence

of unilateral and pulsating pain [7]. With regard to triggers that induce migraine, it has been reported that drinking alcohol, smoking, living with stress, neck pain, and hormonal changes are the main stimuli that play a role [8].

Migraine's etiology involves a neurovascular mechanism and is characterized by cortical hypersensitivity [9]. However, for some authors, it is mainly a neuronal pathology with secondary vascular effects [10].

Migraine has been associated with hormonal changes; several authors have reported that there is an increase in the reproductive years, and menstruation specifically is considered one of the most common triggers for migraine, also affecting the level of pain, the duration of symptoms, and the response to treatment [11–13].

The menstrual cycle is a series of natural changes in hormone production and the structures of the uterus and ovaries of the female reproductive system that makes pregnancy possible. The four phases of the menstrual cycle are menstruation, the follicular phase, ovulation, and the luteal phase. Common menstrual problems include heavy or painful periods and premenstrual syndrome. Migraines that occur during the menstrual period tend to be more disabling than those that happen at different times of the month [14].

Moreover, hormonal contraceptive use during the reproductive years and hormone replacement in menopause alter the levels of sex hormones, and these events and interventions are associated with a change in the prevalence and intensity of headaches [14].

Regarding the mechanism for menstrual migraine, it was reported that an estrogen level drop during the late luteal phase may be a marker of vulnerability to migraine symptoms in women that experience migraine [15]. In line with this, others reported a drop in estrogen levels prior to menstruation, which was explained by the effects of estrogens and progestogens on central serotonergic and opioidergic neurons, modulating their neuronal activity and the density of the 5-HT1 type receptor [16,17].

Although there are some techniques for studying the central mechanisms involved in chronic pain like migraine, electroencephalography (EEG) stands out as a valuable, non-invasive tool because it provides reliable and relevant information about brain functioning during rest, sensory stimulation, and cognitive tasks. In addition, this technique is safe, low-cost, and employs a straightforward methodology, thus making it an appropriate tool for use in clinical practice.

EEG has been applied to assess brain function in several chronic pain syndromes. The American Neurology Academy has suggested that EEG can be used in people with symptoms associated with headaches and migraine [18]. One of the first studies in which EEG alterations were associated with headache dates back to 1959, when Golla and Winter [19], described two main types of EEG frequency response during flickering light: (a) a peak in the alpha band and a rapid decline with an increase in stimulus frequency above 14 f/s, and (b) a flat-top showing a response maintained up to or above 20 f/s. These authors concluded that the spatial distribution of the cerebral mechanisms involved in the flicker response resulted from the disturbance in the cardio-vascular barostatic mechanisms.

Several years later, Wasler et al. [20], reported frontal intermittent rhythmic delta activity (FIRDA) in EEG during and shortly after migraine episodes in migraine patients with episodes of impairment of consciousness and neurological deficit, indicating dysfunction of the upper brainstem and occipital and medial temporal lobes. Schoenen et al. [21], identified markedly reduced alpha activity in one occipital area on the side of the headache in 19 of 22 patients with migraine. Sixteen of these patients had a concomitant reduction in theta activity in the same location. In all patients except one, when they were re-examined seven days after a migraine attack, the EEG asymmetries had disappeared. According to the authors, unilateral EEG changes can thus be detected during migraine attacks and could be associated with unilateral disturbances of cortical electrogenesis. Later, Nyrke et al. [22], found an increase in higher alpha rhythm variability within 72 h following a migraine attack. Bjork et al. [23], found increased relative theta activity and attenuated medium-frequency photic responses in migraineurs without aura compared to controls. On the other hand, O'Hare et al. [24] found the lower alpha band (8 to 10 Hz) power was increased in

the migraine group compared with the control group, which may provide a mechanism for increased multiplicative noise.

Silberstein [25], described that migraine was more present in women than in men, suggesting that changes in estrogen levels at menarche, menstruation, pregnancy, and menopause may trigger or change the prevalence of migraine. For example, the fall in the levels of estrogen that occurs during menstruation triggers menstrual migraine, whereas the sustained high estrogen levels during pregnancy frequently result in headache relief. The same author argued that estrogen produces changes in prostaglandins, hypothalamic opioids, and prolactin secretion, which may, in part, account for the genesis of headache. For this reason, one might assume that EEG patterns could be influenced by the menstrual cycle and the pain experienced. Becker et al. [26], reported mean alpha frequency cyclic changes in EEG activity, i.e., slower alpha waves during the follicular phase and faster alpha waves during luteal phase, as well as theta and beta small cyclic changes in women during both spontaneous and oral contraceptive-controlled menstrual cycles. Solís-Ortiz et al. [27], studied the effect of the menstrual cycle on EEG power during rest (eyes open and closed) in healthy women with no oral contraceptive effects. They reported lower EEG absolute power during the follicular phase; high power in delta, theta, and alpha 1 (7.5–9.5 Hz) during the luteal phase; high alpha 2 (9.5–12.5 Hz), beta 1 (12.5–17.5 Hz), and beta 2 (17.5–30 Hz) during the menstrual phase; and lower relative power in low alpha and higher in high alpha during the luteal phase. In addition, there was higher interhemispheric correlation between frontal regions during ovulation and between occipitals during the luteal phase, with no significant asymmetries. Thus, the authors concluded with the observation of a lower activation of frontal regions during the luteal phase and higher activation of central–parietal regions during the menstrual phase. Furthermore, Baehr et al. [28], found EEG frontal alpha asymmetry in a group of women suffering from premenstrual dysphoric disorder in comparison to a control group during the luteal period. Haraguchi et al. [29], reported lower alpha, theta, and gamma MEG power during the menstrual phase in comparison with outside this phase in healthy women.

Platzer et al. [30], investigated the effect of the menstrual cycle on brain activation and connectivity patterns by using fMRI in naturally cycling women performing cognitive tasks (spatial navigation and verbal fluency). The authors found no significant difference in task performance throughout the menstrual cycle, and changes in brain activation patterns were similar during both tasks. They also reported a hippocampal activation during the follicular phase and a boosting effect of progesterone in fronto-striatal activation during the luteal phase. Moreover, right-hemispheric frontal activation was suggested to result from inter-hemispheric decoupling and to be involved in the down-regulation of hippocampal activation. Hidalgo and Pletzer [31], assessed brain activation during an N-back verbal memory task in women with a regular menstrual cycle. They were able to corroborate a hormone-mediated inter-hemispheric decoupling that enhanced frontal activity and the disinhibition of the salience brain network and striatum during the luteal phase. The authors interpreted these results in relation to a top-down differential regulation in higher hormone level phases and a hyperactive bottom-up network during the luteal phase, which could explain the vulnerability of this phase to menstrual cycle-associated disorders.

To the best of our knowledge, this is the first study that addresses the effect of migraine on the resting-state EEG of women during the luteal phase. We focused on the luteal phase as it is characterized by a change in estrogen levels, which may trigger migraine symptoms. Based on the mentioned migraine studies that reported EEG power changes in different frequency bands (delta, theta, alpha), we expected a difference in EEG power between the with and without migraine conditions in women during the luteal phase.

2. Material and Methods

2.1. Subjects

Twenty female right-handed subjects participated in our study. There were ten women with migraine: mean age 25.4 ± SD 1.9 years, mean years of condition 4.9 (±SD 2.6), mean

schooling 14.5 ± SD 2.1 years. In the case of healthy women: mean age 26.7 ± SD 1.9, mean schooling 15.5 ± SD 3.5.

We used the Oldfield questionnaire [32], to test the handedness.

A local Ethics Committee of Maestría en Diagnóstico y Rehabilitación Neuropsicológica from the Faculty of Psychology of Benemérita Universidad Autónoma de Puebla (DCECEN) approved the experimental protocol. All women participated in accordance with the Declaration of Helsinki as it was established by the World Medical Association in 1964 [33]. Subjects participated with understanding and informed consent.

We collected data through a semi-structured interview (Table 1). The experimental group described pain originating from all over the frontal and central areas.

Table 1. Questionnaire results.

Patient	Pain Place L (Left), R (Right)	Symptoms
1	Frontal L	Dizziness
2	Fronto-Parietal R	Blurred vision
3	Frontal R	None
4	Frontal L–R	None
5	Frontal L	Dizziness
6	Frontal L-R	Dizziness
7	Frontal L-R	None
8	Frontal L	Dizziness
9	Frontal R	Dizziness
10	Frontal L	Dizziness, blurred vision

Nausea was the most frequent symptom associated with pain during a migraine attack. Vomiting and dizziness were also present, as well as blurred vision.

For convenience, our study was cross-correlational with non-probability statistical sampling. The migraine of participants was diagnosed by a neurologist, according to 'the International Classification of Headache Disorders, 3rd edition [34]. Patients were recruited through a request from the neurology service to complete the neurological assessment. We also used semi-structured interviews to collect demographic data, as well as information on how migraine episodes occur: triggering (beginning), episode duration, pain location, frequency, and associated symptoms.

The control group was formed based on the voluntary participation of women awaiting gynecological consultation (control group).

Inclusion Criteria for All Participants

Patients with migraine (experimental group) had the following signs: headache attacks lasting 4–72 h, pulsating quality, moderate or severe pain intensity, aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs), nausea, vomiting, photophobia, or phonophobia. We selected patients with migraine who reported a migraine attack in the last 24 h prior to the EEG session.

Volunteer women had no signs of migraine or headache.

All women with regular spontaneous menstrual cycles were screened in a standardized interview.

All women in the luteal phase were selected with the use of a calendar to establish the first day of menstrual bleeding, the average cycle length, and the length of the luteal phase.

Exclusion criteria:

Anxiety or depression;

Neuroleptic or drug use for chronic pain, depression, or epilepsy;

Alcohol or drug use;

Report of irregular menstrual cycles;

Pregnant or lactating during the last 12 months;

Taking oral contraceptives during the last four months.

2.2. EEG Session

Subjects sat comfortably in an electrically shielded, dimly lit room. We recorded EEGs in rest conditions with closed eyes for three minutes. Subjects did not report having anxiety during the experimental session, but women with migraine (experimental group), reported migraine attack in the last 24 h prior to the EEG session.

2.3. Recordings

We collected EEGs (bandpass DC-200 Hz, sampling rate 250 Hz, NicVue System, Nicolet Biomedical Inc., Middleton, Wisconsin, USA) from 20 scalp positions (according to 10–20 System) referenced to the ear lobes, with the ground electrode at the forehead. We set the Notch filter at 60 Hz, and we kept electrode impedances under 5 kOhm. For further analysis, we recorded electrooculograms (using the same bandpass and sampling rate as for the EEG) to exclude trials contaminated by eye movements. We stored data and analyzed them offline.

3. Data Analysis

3.1. EEG Spectral Power Analysis

We performed offline visual artifact rejection to exclude contaminated segments. After that, we concatenated segments of 60 s (15,000 points) from each participant. We computed spectral power (SP) for the considered frequency bands by using customized software scripts programed in MATLAB (Mathworks, 2019), using the following formula:

$$SP_C(f) = \frac{1}{n}\sum_{i=1}^{n} C_i(f)C_i^*(f)$$

where C_i represents the Fourier-transformed channel c for a given segment number ($i = 1, n$) and "*" indicates the complex conjugate.

We calculated the SP in the following frequencies: delta (0.5–3.5 Hz), theta (4–7.5 Hz), alpha (8–12 Hz), and beta (13–30 Hz). The frequency resolution we selected was 0.1 Hz.

3.2. Statistical Analysis

We calculated the areas under the curve for each SP frequency band. To test for any statistical difference in the SP, we normalized data values between 0 and 1. Because our data were not normally distributed (Shapiro–Wilk normality test $p < 0.05$), we used a nonparametric statistical analysis for two independent groups (U Mann–Whitney test) for the absolute power comparisons in each band per topographic area. In each band, we grouped the electrodes' signals to the average by cortical areas. In the frontocentral topographical area, we averaged the following electrodes: Fp1, Fp2, F7, F3, Fz, F4, F8, C3, Cz, and C4. In the occipitoparietal area, we averaged the following electrodes: P3, Pz, P4, O1, Oz, and O2. In the temporal area, we averaged the following electrodes: T3, T4, T5, and T6.

The null hypothesis was that the dependent variables were the same across the factors. We report effects as significant (two-tailed) if $p \leq 0.05$. The statistical analysis was performed using the software SPSS 25.

4. Results

The task was performed for all subjects according to the instructions. None of the participants reported fatigue during the experiment, but some showed anxiety and signs of irritability, such as sweating, agitation, and claustrophobic sensation.

4.1. Topographical Analysis

We obtained the following results for the absolute power analysis in different cortical areas and frequency bands.

4.2. Alpha

We found a significantly higher normalized power in the experimental group for the alpha band in the frontocentral area (U = 23, z = −2.04, r = 0.45, $p < 0.05$) in comparison with the control group. In contrast, we found a significantly higher normalized power in the occipitoparietal area in the control group (U = 6, z = −1.92, r = 0.43, $p < 0.05$) in comparison with the experimental group (Figure 1).

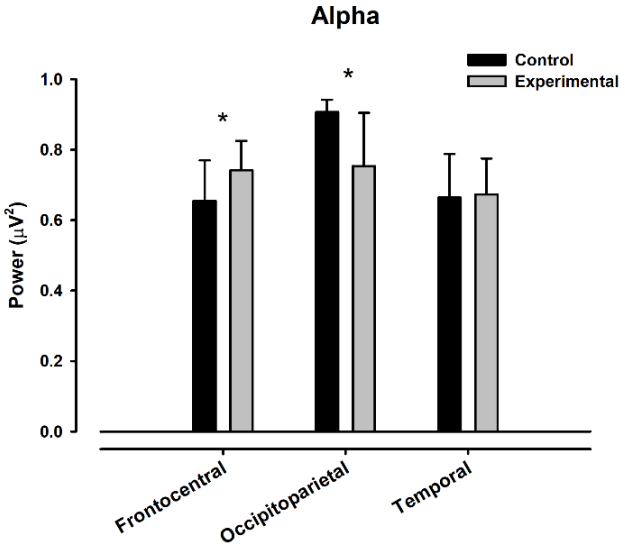

Figure 1. Topographic comparisons in alpha band. The * means a statistically significant difference ($p < 0.05$).

4.3. Beta

We found a significantly higher normalized power in the control group for the beta band in the frontocentral area (U = 19, z = −2.34, r = 0.52, $p < 0.05$), occipitoparietal area (U = 2, z = −2.56, r = 0.57, $p < 0.05$), and temporal area (U = 0, z = −2.31, r = 0.52, $p < 0.05$) in comparison with the experimental group (Figure 2).

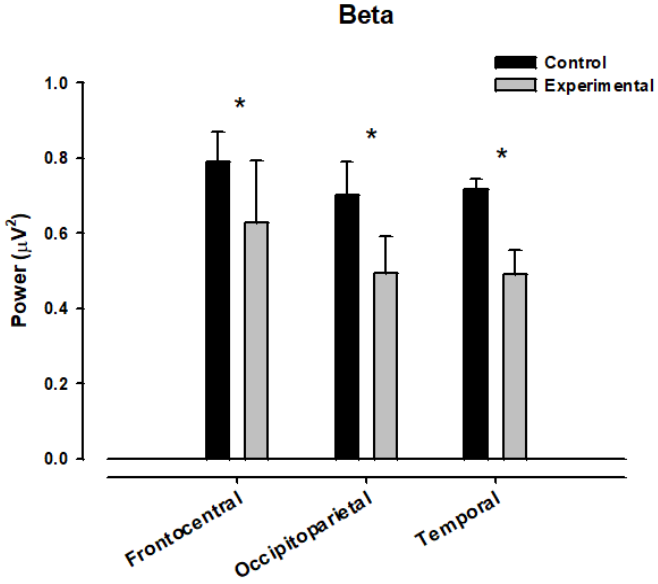

Figure 2. Topographic comparisons in beta band. The * means a statistically significant difference ($p < 0.05$).

4.4. Delta

We found a significantly higher normalized power in the control group for the delta band in the temporal area (U = 0, z = −2.31, r = 0.52, $p < 0.05$) in comparison with the experimental group (Figure 3).

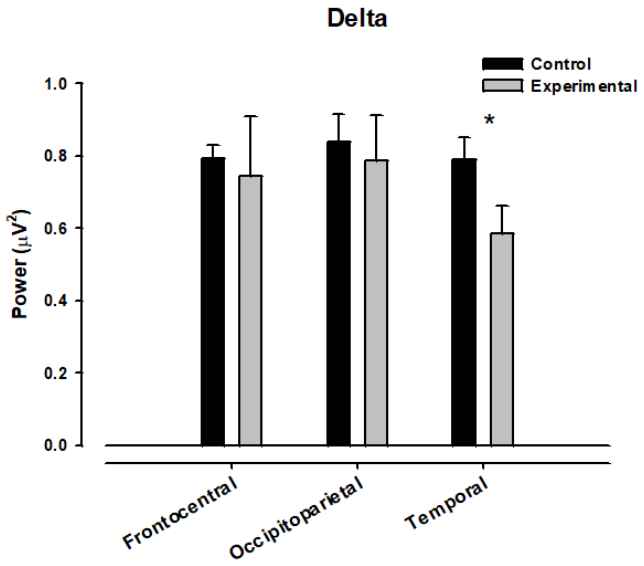

Figure 3. Topographic comparisons in delta band. The * means a statistically significant difference ($p < 0.05$).

4.5. Theta

We found a significantly higher normalized power in the control group for the theta band in the frontocentral area (U = 24, z = −1.97, r = 0.44, $p < 0.05$) and occipitoparietal area (U = 5, z = −2.10, r = 0.47, $p < 0.05$) in comparison with the experimental group (Figure 4).

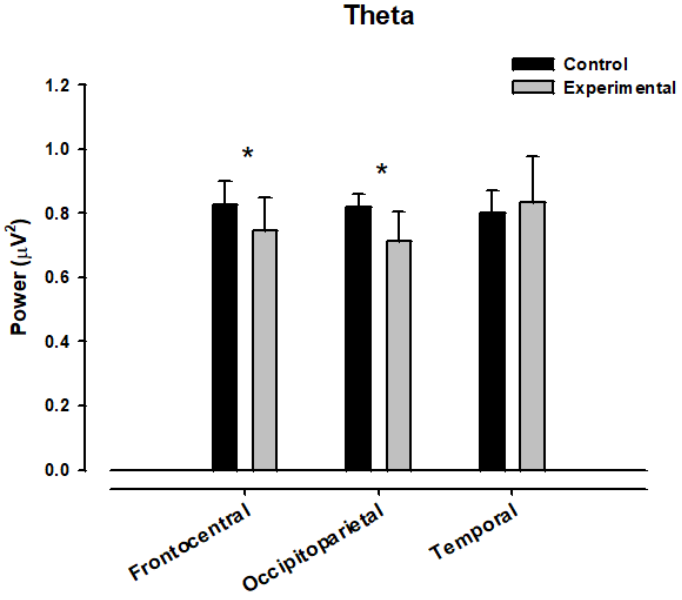

Figure 4. Topographic comparisons in theta band. The * means a statistically significant difference ($p < 0.05$).

4.6. Grand Average Band Analysis

For each band, we averaged the normalized absolute power for all electrodes (Figure 5). We found a significantly higher amplitude in the theta (U = 5, Z= −0.32, $p < 0.05$, r = 0.07)

and beta (U = 2, Z= −2.56, $p < 0.05$, r = 0.57) bands in the control group in comparison with the experimental group.

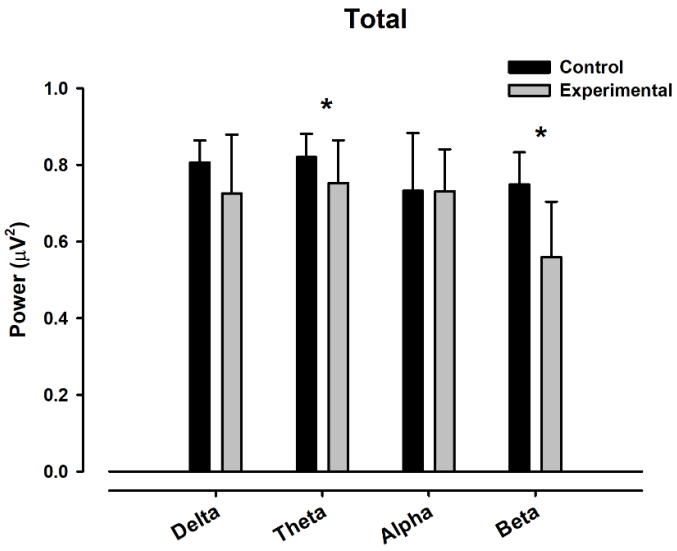

Figure 5. Total averaged absolute power for delta, theta, alpha, and beta bands. The * means a statistically significant difference ($p < 0.05$).

4.7. Independent Channel Analysis

We performed the same analysis for each electrode as for cortical areas. Because the statistics to compare all electrodes with all the bands in the two groups would require a multidimensional analysis, we do not present comparisons of these results. For this reason, we simplified the analysis by areas. We only describe qualitatively what we see in the corresponding averaged areas The mean absolute power figure, for individual electrodes in the 10–20 system (both groups) is presented in the Supplementary Material because we believe it might provide new data on the distribution of frequency bands in the scalp.

We found more specific differences in the frontal areas: the theta band was larger in Fp1 and Fp2 in the control group than in the experimental group. In F7 and F8, beta was larger in the control group than in the experimental group. Meanwhile, the alpha band in the same areas was larger in the experimental group than in the control group. In F3 and F4, beta was larger in the control group than in the experimental group (see Figure S1).

We also found that in the somatosensory-related leads C3 and C4, the beta band was larger in the control group than the experimental group. We also found that the theta band over C3 was larger in the control group than in the experimental group. In contrast, the theta band over C4 was larger in the experimental group than in the control group (see Figure S1).

5. Discussion

It has been reported that migraine is three times more frequent in women than in men of reproductive age. This fact is associated with the changes of hormonal mechanisms that occur in females throughout the menstrual cycle [35].

A specific phase of the menstrual cycle that is related to psychological, cognitive, and physical changes is the luteal phase. The symptoms and signs of this period are distinct and have been named pre-menstrual syndrome. During this period, a persistent headache of the migraine type that decreases after three days of menstruation has been reported [36].

The present study showed that women with no migraine presented the following relevant encephalographic characteristics during the luteal phase: alpha occurrence over the parieto-occipital area, a predominance of beta rhythm over the whole scalp, theta increase over the frontocentral and parietooccipital area, and an increase in delta for the temporal areas.

In the case of women with migraine, we observed an increase in alpha in the frontocentral area and a reduction in alpha in occipitoparietal area. Few studies have been carried out in patients with migraine, and the results have not been very consistent. However, the first studies reported an increase in alpha rhythm variability in the headache-free phase [21,22,37–40].

In our study, we observed a reduction in beta in all the considered cortical areas except the frontopolar leads. This result is in contrast with studies that reported an excess of beta activity during attacks [41], in migraine patients. Walker [42], reported an excess of high-frequency beta activity (21–30 Hz) in all four cortical areas in a group of migraine patients in neurofeedback therapy in comparison with a group of patients using drug therapy.

We also found a focal decrease in the theta band in the left central–parietal leads (C3 and P3) and a local delta decrease in the left temporal and right occipital leads (T3 and O2). Past research has also shown that individuals with chronic pain exhibit increased beta and decreased alpha activity, with additional increased theta/delta [43]. Much evidence suggests the involvement of delta oscillations during sustained pain as a reflex of autonomic processes linked to efforts of homeostatic processes [44]. However, our study is more specifically focused on a phase of the menstrual period that begins with pain and not the pain itself.

Xie et al. [45], suggested that EEG activity in migraine patients is related to a predisposition to painful or high-risk stimuli. In line with Xie et al. [45], we observed power differences between the considered groups in the frontal channels (see Figure S1).

Some authors suggest that migraine is related to a lack of habituation to environmental stimuli due to inadequate information processing [46]. Our study supports the suggestion that the differences in EEG patterns in women with migraine could be involved in pain related to migraine and the regulation of input processing. In other words, chronic pain can change the responsiveness of brain regulatory systems and the emotional processing of somatosensory information; this could be a reason why we found that in somatosensory areas such as C3 and C4, the delta and beta band were larger in the control group than the experimental group. This type of EEG pattern could be related to allostatic load, which also includes the participation of the hypothalamic–pituitary–gonadal axis and the negative feedback regulation of the endogenous opioid system during the release of luteinizing hormone. The cyclic surges of gonadal hormones may directly alter neuronal, glial, and astrocyte function throughout the brain [47].

The irregular EEG patterns found in our study could be used not only for diagnosis criteria, but also for the improvement of pharmacological and psychological therapeutic targets.

Future work should consider EEG data as a part of the physiological changes related to the menstrual cycle in relation to behavioral and emotional traits. As there is increasing evidence that migraine symptoms could be ameliorated by a multimodal approach that includes behavioral interventions like biofeedback, cognitive behavioral therapy, and relaxation, at first, women may consider the use of medication, which may be complemented by an integrated approach that includes exercise, relaxation, and biofeedback. Future studies should investigate the effect of such interventions as reflected in neurophysiological data.

It is also important to address the recognition of premonitory symptoms in migraine, because most patients in the present work were able to correctly predict the onset of migraine headaches. The pain prediction may represent an essential treatment paradigm, where the risk of headache is treated prior to the experience of pain.

6. Conclusions

Finally, we state that more conclusive research is needed to understand the physiology of migraine in greater detail. Similarly, research involving various levels of analysis is needed in order to be more explanatory and less descriptive. It is important to consider that the sample size may have been a limitation in our study. We suggest that the sample size should be increased to have external validation. In addition, carrying out hemisphere

correlation studies and coherence analysis will be essential to analyze the diffuse asymmetry of EEG patterns in patients with migraine.

Even though the signs and symptoms of migraine guide the clinical course of diagnosis, the electroencephalographic data can improve or increase the sensitivity of the diagnosis according to the specific phases of the menstrual cycle. Therefore, the results derived from EEG can function as biomarkers of the condition of the patient.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/app13137443/s1, Figure S1: Distribution of the mean absolute power for delta, theta, alpha, and beta bands for each location of scalp electrodes in the 10–20 system.

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Informed Consent Statement: The studies involving human participants were reviewed and approved by Local Ethics Committee of Maestría en Diagnóstico y Rehabilitación Neuropsicológica from the Faculty of Psychology of Benemérita Universidad Autónoma de Puebla (DCECEN 254546 OMD). The patients/participants provided their written informed consent to participate in this study.

Data Availability Statement: The original contributions presented in the study are included in the article, further inquiries can be directed to the corresponding author.

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