International Journal of Health Sciences and Research
www.ijhsr.org ISSN: 2249-9571

Original Research Article

Electroencephalogram (EEG) Changes in Premenstrual Dysphoric Disorder (PMDD)

Binu Shrestha[1], Bishnu Hari Paudel[2], Nirmala Limbu[3], Kopila Agrawal[4], DhanaRatna Shakya[5], Mohan Regmi[6]

[1]Assistant Professor, Neuroscience, Trinity School of Medicine, St. Vincent and the Grenadines.
[2]Professor, Department of Basic and Clinical Physiology, BPKIHS, Dharan, Nepal.
[3]Additional Professor, Department of Basic and Clinical Physiology, BPKIHS, Dharan, Nepal.
[4]Associate Professor, Department of Physiology, Birat Medical College, Biratnagar, Nepal.
[5]Additional Professor, Department of Psychiatry, BPKIHS, Dharan, Nepal.
[6]Associate Professor, Department of Obstetrics & Gynecology, BPKIHS, Dharan, Nepal.

Corresponding Author: Binu Shrestha

Received: 12/08/2016 *Revised: 27/08/2016* *Accepted: 31/08/2016*

ABSTRACT

The prevalence of premenstrual dysphoric disorder (PMDD) is high among the reproductive age group of the female population. The aim of the study was to assess electroencephalogram (EEG) changes in PMDD and compare with healthy controls. The study was conducted on thirty female students of BP Koirala Institute of Health Sciences (BPKIHS), Dharan, Nepal with symptomatic PMDD and thirty female age-matched controls in Clinical Neurophysiology Lab of Department of Basic and Clinical Physiology, BPKIHS, Dharan, Nepal. Subjects were screened with questionnaire and research criteria ofDiagnostic and Statistical Manual of Mental Disorders 4th edition (DSM-IV). EEG recordings of all the participants were done before menstruation start or during the peak of symptoms. EEG was dissected out into its constituent frequency bands by Fast Fourier Transformation and the data of EEG and other variables were compared in between groups. The findings showed increased frontal alpha 2 (p=0.039) in eyes-closed and frontal beta (p=0.014) in eyes-open condition among PMDD group as compared to the control group. Thus, the result is suggestive of the presence of feature anxiety, emotional stress, and poor sleep quality during the symptomatic phase of PMDD.

Keywords: Premenstrual dysphoric disorder (PMDD), Premenstrual syndrome (PMS), Electroencephalogram (EEG),

INTRODUCTION

Premenstrual dysphoric disorder (PMDD) is a severe form of premenstrual syndrome (PMS) that occurs in 3-8% of women, [1] and an additional 19% identified as 'near-threshold' cases that failed to meet the strict PMDD criteria. [2] PMDD is classified as a 'depressive disorder not otherwise specified' in the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV) (American Psychiatric Association, 1994). [3] Although the etiology of PMS and PMDD remains uncertain at present, researchers now concur that these disorders represent biological phenomena rather than purely psychological events. Medical definitions of PMS are limited to a consistent pattern of emotional and physical symptoms occurring only during the luteal phase of the menstrual cycle that are of sufficient severity to interfere with some aspects of life. [1]

Electroencephalogram (EEG) is the recording of electrical activity along the scalp produced by the firing of neurons within the brain. [4] Studies showed that cyclical variation also occurred in the frequency of cortical electrical activity, [5] and also reported an increase in slow-wave activity in the electroencephalogram (EEG) at the onset of menstruation. [6] Study has shown that psychological and physiological changes occur during PMDD has an impact on day-to-day life which adversely affects the emotional well-being, occupational performance, and social relationship. So, the aim of this study is to investigate electroencephalographic changes in the PMDD and compare with controls.

MATERIALS AND METHODS

The Cross-sectional comparative study was conducted among the female students of BPKIHS. The groups PMDD and Controls were selected based on the inclusion and exclusion criteria. Female students of reproductive age between 18 to 30 years with regular menstrual cycle (28±7 days) and fulfilling research criteria DSM-IV were included. Female students with a history of seizure disorder or any psychiatric illness and major medical illness or taking drugs that show changes in EEG and also females on oral contraceptive pills were excluded. All the participants were explained about the study and recording procedures and informed written consents were taken. Documentation of detailed medical history and physical examinations were done. The study group (PMDD) was selected based on DSM-IV criteria. [3] Both the groups had their anthropometric and cardiorespiratory variables measured.

Electroencephalogram (EEG) Recording

After screening with a questionnaire, anthropometric variables were measured. The participants were advised to rest for 10 minutes in a supine position on the dental chair after adjusting the head and back on the chair according to their comfort. The EEG recordings were undertaken in a quiet, relaxed environment, with the subject lying comfortably in supine position. Premenstrual EEG recordings were done for 6 minutes during eyes-closed, eyes-open conditions for 3 minutes each.

Data acquisition

EEG records were visually inspected on the computer to check for eye blink, detectable eye movement, and body movement artifacts. After visual inspection, three artifact-free-5-second epochs selected from just before the end of 1st, 2nd, and 3rd minute of EEG recording during eyes-closed and eyes-open phase. Thereafter, Fast Fourier Transformation (FFT) method was applied to these data for decomposition of EEG waveforms into sine wave components in terms of respective frequencies.

These components were used to estimate spectral power for frequency in the ranges of delta (0.5-4.0 Hz), theta (4.0-7.0 Hz), alpha1 (7.0-10.0 Hz), alpha2 (10.0-13.0 Hz), and beta (13.0-32.0 Hz) bands. The spectral power for each band thus obtained for different regions of the brain (as provided by the selected montage) was exported to Microsoft Excel worksheet files for further analysis. The powers from three epochs were averaged for each subject.

Statistical analysis

The data obtained for anthropometric variables, cardio-respiratory variables, and EEG power spectra exported to SPSS (*version 18*) and tested for normal distribution. The data of anthropometric variables, cardio-respiratory variables, were normally distributed and expressed in terms of mean ± standard deviation (SD).

The data of EEG power spectra were non-normally distributed, hence subjected to log transformation and statistical analysis was done. The EEG power spectra were expressed as mean ± SD. Independent t-test was used for comparison of anthropometric variables, cardio-respiratory variables, and EEG power spectra between the study group and healthy controls. A p-value of ≤ 0.05 was considered statistically significant.

RESULTS

The anthropometric variables (age, weight, height, and BMI) and cardiorespiratory variables (systolic and diastolic blood pressure, pulse rate, respiratory rate, and arterial oxygen saturation), when compared between PMDD group and control,did not show any statistical significance.

EEG power spectra during eyes-close condition (Table 1) showed there was significantly more alpha2 activity in PMDD group than healthy controls at frontal (p=0.039) and temporal (p=0.010) sites.

EEG power spectra during eyes-open condition (Table 2) showed there was significantly more beta activity at Frontal (p=0.014) in EEG of PMDD than control before menstruation.

Table 1: Comparison of EEG power spectra between PMDD (n=30) and controls (n=30) Eyes-close condition

		Delta		Theta		Alpha 1		Alpha 2		Beta	
Electrode sites	Group	Mean ± SD	p	Mean ± SD	p	Mean ± SD	p	Mean ± SD	p	Mean ± SD	p
Frontal	PMDD	40.89 ± 34.23	0.861	6.99 ± 6.37	0.773	20.72 ± 36.58	0.262	8.72 ± 6.42	0.038*	10.22 ± 6.14	0.417
	Control	38.94 ± 32.48		5.84 ± 3.95		22.92 ± 30.74		5.00 ± 2.83		8.14 ± 3.40	
Temporal	PMDD	30.37 ± 24.11	0.247	10.19 ± 8.43	0.720	60.74 ± 93.34	0.575	47.83 ± 41.67	0.010*	26.65 ± 18.50	0.242
	Control	22.02 ± 14.10		10.43 ± 9.23		93.11 ± 103.72		19.67 ± 14.83		20.10 ± 12.47	
Midline	PMDD	79.01 ± 34.03	0.973	38.70 ± 33.79	0.815	93.90 ± 143.64	0.268	47.00 ± 38.45	0.390	39.29 ± 30.25	0.813
	Control	80.38 ± 40.96		36.25 ± 19.89		115.56 ± 137.07		33.69 ± 22.66		37.14 ± 25.78	
Parietal	PMDD	64.28 ± 52.14	0.516	23.64 ± 25.62	0.601	88.43 ± 131.67	0.405	84.78 ± 75.83	0.487	37.80 ± 27.99	0.864
	Control	55.49 ± 38.59		25.88 ± 20.48		156.83 ± 289.62		58.27 ± 49.48		31.95 ± 19.50	
Occipital	PMDD	52.54 ± 53.96	0.438	20.50 ± 24.39	0.551	110.46 ± 218.43	0.063	69.09 ± 73.14	0.544	35.19 ± 24.09	0.469
	Control	46.37 ± 30.84		17.94 ± 14.15		180.23 ± 225.50		53.03 ± 46.98		26.07 ± 13.33	

Table 2: Comparison of EEG power spectra between PMDD (n=30) and controls (n=30) Eyes-open condition.

		Delta		Theta		Alpha 1		Alpha 2		Beta	
Electrode sites	Group	Mean ± SD	p	Mean ± SD	p	Mean ± SD	p	Mean ± SD	p	Mean ± SD	p
Frontal	PMDD	21.65 ± 17.13	0.509	3.78 ± 1.92	0.481	3.49 ± 2.60	0.857	3.00 ± 2.78	0.622	8.76 ± 5.17	0.013*
	Control	20.06 ± 18.42		3.90 ± 1.50		3.54 ± 2.55		2.26 ± 1.51		5.79 ± 2.62	
Temporal	PMDD	23.67 ± 23.72	0.299	4.07 ± 2.25	0.832	6.91 ± 8.99	0.286	8.87 ± 10.84	0.583	14.34 ± 11.95	0.560
	Control	17.37 ± 9.48		3.87 ± 1.84		10.36 ± 12.72		6.23 ± 7.88		12.04 ± 11.05	
Midline	PMDD	68.07 ± 23.71	0.239	18.20 ± 7.37	0.777	18.93 ± 14.04	0.082	21.88 ± 26.69	0.574	28.25 ± 20.60	0.938
	Control	61.39 ± 25.24		17.65 ± 6.57		34.06 ± 39.50		22.00 ± 21.63		26.36 ± 18.72	
Parietal	PMDD	48.92 ± 17.42	0.306	12.61 ± 5.33	0.813	16.98 ± 18.09	0.096	22.70 ± 31.59	0.458	24.92 ± 20.95	0.762
	Control	45.27 ± 20.67		12.26 ± 4.60		30.17 ± 36.62		30.32 ± 51.22		24.08 ± 18.87	
Occipital	PMDD	45.81 ± 52.67	0.538	8.20 ± 4.64	0.659	13.06 ± 17.16	0.110	15.54 ± 18.21	0.749	22.60 ± 16.05	0.195
	Control	34.82 ± 16.77		8.03 ± 2.88		22.07 ± 26.15		15.02 ± 18.35		16.99 ± 14.04	

*p<0.05

DISCUSSION

The aim of the study was to investigate EEG changes among PMDD and controls. The results showed more EEG spectral power of alpha 2 activity at the frontotemporal site, during eye close state and more beta activity at the frontal site during eyes-open state, in the premenstrual recording of PMDD group as compared to healthy controls. Thesefindings are consistent with Pollock and Schneider, 1990 who reported increased in alpha and beta1 power during waking EEG in a patient with depression [7] and increased beta activity during waking is common in patient with psychiatric disorders. [8] So it can infer that study group must have symptoms of depression which are also included in

research criteria for PMDD. This is also supported by the study done by C. Buchpiguel *et al.* [9] to determine changes in regional cerebral blood flow (rCBF) associated with PMS, which concluded that SPECT (Single photon emission computed tomography) imaging demonstrates modest decreases in rCBF in the temporal lobes that correlate with the level of depression in subject with PMS.

The present results are also consistent with the statement given by Niedermeyer E and da Silva FL. [10] EEG beta1 power during waking state indicates cortical arousal. [11] Another study also found a trend for increased power density in the 12-13 Hz narrow band during Non-REM sleep in the same group of women with severe PMS compared with controls in both the follicular and lateluteal phases of the menstrual cycle. [12] Though there is increased beta1 activity, as shown by the present study, this study was done during sleep, thus discrepancy with our study seems natural.

The finding is in the line of a study done by I-Mein Lin *et al*. [13] which showed higher frontal alpha asymmetry among the participant with PMDD than without PMDD in depressive induction and relaxation condition during the luteal phase. A study done by Scherzer et al [14] found a well-developed alpha rhythm in the emotionally very tense state.

The study done by Leary and Batho had compared between the EEG recorded in the premenstrual phase and those recorded at mid-cycle also had the result of a significant increase in mean alpha frequency and increase in mean alpha amplitude in premenstrual records. These findings may correlate with subjective mood changes commonly experienced by females in the premenstrual phase. [15]

In contradictory to present result, Toner et al in 1995 found increased in delta activity during PMS along with a suggestive, but not necessarily significant decreased in beta activity, [16] in their study sample size was 6 women of age ranged from 30 to 43. They were not grouped based on research criteria but were self-reported. They had 21- channel QEEG (quantitative EEG) recording and P300 evoked potentials measured during mid-cycle and premenstrual phase. Thus, the differences between this study and the study by Toner et al seem obvious.

CONCLUSION

The result of the study showed increased alpha 2 and beta activity in premenstrual EEG at the front temporal region among the participants with symptoms of PMDD which is suggestive of the presence of depression, anxiety, poor sleep quality, emotional stress in premenstrual dysphoric disorder patients.

ACKNOWLEDGEMENT

We acknowledge it to all the faculty members, postgraduates students, laboratory technician, staffs of and all the participants for their help and support in conducting this research project.

REFERENCES

1. Steiner M. Premenstrual syndrome and premenstrual dysphoric disorder: Guidelines for management. J Psychiatry Neurosci2000; 25:459-68.
2. Witttchen HU, Becker E, Leib R. Prevalence, incidence and stability of premenstrual dysphoric disorder in the community. Psychol Med 2002; 32(1): 119-32.
3. The American Psychiatric Association, Diagnostic and Statistical manual of Mental disorders, Fourth Edition, Text Revision, Washington DC, American Psychiatric Association. 2000:717-8.
4. Niedermeyer E, da Silva FL. Electroencephalography: Basic Principles, Clinical Applications, and Related Fields. Lippincott Williams & Wilkins; 2004.
5. Lindsley, D. B., and Rubenstein, B. B. Relationship between brain potentials and some other physiological variables, Proc Soc expo Bio.1937; 35, 558.
6. De Barenne, D. D., and Gibbs, F. A. Variations in the electroencephalogram during the menstrual cycle. Amer J

Obstet Gynec.1942; 44, 687.
7. Pollock VE, Schneider LS. Quantitative, waking EEG research on depression. Biol Psychiatry. 1990; 27:757-780.
8. Coutin-Churchman P, Añez Y, Uzcátegui M, et al. Quantitativespectral analysis of EEG in psychiatry revisited: drawing signs out of numbers in a clinical setting. Clin Neurophysiol. 2003; 114:2294-2306.
9. Buchpiguel C, Alavi A, Crawford D, Freeman E, Newberg A. Changes in cerebral blood flow associated with premenstrual syndrome. J Psychosom Obstet Gynaecol. 2000; 21(3):157-165.
10. Niedermeyer E, da Silva F L. Electroencephalography Basic Principles, Clinical Applications & Related fields. Baltimore: Urban & Schwarzenberg; 1982.
11. De Valck E, Cluydts R, PirreraS. Effect of cognitive arousal on sleep latency, somatic and cortical arousal following partial sleep deprivation. J Sleep Res. 2004; 13:295-304.
12. Baker FC, Kahan TL, Trinder J, Colrain IM. Sleep quality and the sleep electroencephalogram in women with severe premenstrual syndrome. *Sleep.* 2007; 30(10):1283-1291.
13. Lin IM, Tsai YC, Peper E, Yen CF. Depressive mood and frontal alpha asymmetry during the luteal phase in premenstrual dysphoric disorder. J Obstet Gynaecol Res. 2013; 39(5):998-1006.
14. Scherzer E, Das F. The flat EEG as bioelectric expression of expectation stress (psychogenic Alpha reduction). Psychiatr Neurol (Basel). 1966; 152: 207-212.
15. Leary PM, Batho K. Changes in the electroencephalogram related to the menstrual cycle. S Afr Med J. 1979; 55(17):666.
16. Toner I, Pedab C, Carol S, Hayden M, Stone J, Vuciecevic V. P300 and QEEG changes during menstrual cycle. (Abstract) Int J Psychophysiol. V (1995).

How to cite this article: Shrestha B, Paudel BH, Limbu N et al. Electroencephalogram (EEG) changes in premenstrual dysphoric disorder (PMDD). Int J Health Sci Res. 2016; 6(9):174-178.

****