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Relationship between the Spectral Power Density of Sleep Electroencephalography and Psychiatric Symptoms in Patients with Breathing-related Sleep Disorder

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Objective: Patients with breathing-related sleep disorder (BRSD) often complain of psychiatric symptoms such as depression in addition to snoring, excessive sleepiness, and disturbed sleep. However, the relationship between psychiatric symptoms and severity of sleep apnea in BRSD is controversial. We conducted this study to investigate the relationship between psychiatric symptoms and sleep electroencephalography (EEG) findings in BRSD patients using spectral analysis.
Methods: All participants underwent polysomnography and evaluation using Symptom Checklist-90-Revised (SCL-90-R) scale. We analyzed the absolute spectral power density values of standard EEG frequency bands in the participants (n = 169) with BRSD during the non-rapid eye movement (NREM) sleep period. We performed correlation analysis between the domain scores of SCL-90-R scale and the absolute values of the EEG frequency bands.
Results: Significant positive correlation was observed between the absolute spectral power density values in the slow oscillation band and the degree of paranoid ideation (r = 0.226, p = 0.028) and depression (r = 0.216, p = 0.044) in SCL-90-R. The multiple linear regression model showed that higher paranoid ideation domain score (B = 0.007, p = 0.020), younger age (B = −0.011, $p < 0.001$), and female sex (B = 0.213, p = 0.004) were associated with higher slow oscillation power during NREM sleep.
Conclusion: The results of the present study suggested a relationship between sleep EEG and psychiatric symptoms in patients with BRSD. This relationship needs to be validated with further studies.

KEY WORDS: Breathing-related sleep disorder; Sleep electroencephalography; Spectral analysis; Symptom Checklist-90-Revised; Polysomnography.

INTRODUCTION

Breathing-related sleep disorder (BRSD) is a common sleep disorder characterized by snoring, recurrent episodes of partial or complete collapse of the upper airway, and witnessed apnea during sleep. BRSD can be clinically diagnosed using the Diagnostic and Statistical Manual of Mental Disorders (fourth edition, text revision) (DSM-IV-TR) [1]. However, polysomnography (PSG) is required for accurate diagnosis. BRSD can be classified into simple snoring (SS), upper airway resistance syndrome (UARS), and obstructive sleep apnea (OSA) according to the apnea-hypopnea index (AHI) scores [2]. OSA is diagnosed when apnea or hypopnea occurs five or more times per hour (AHI ≥ 5) [3]. In the BRSD spectrum, SS is defined as AHI < 5 and UARS is defined as AHI < 5 and respiratory disturbance index > 5.

Common symptoms of BRSD such as snoring, fatigue, and sleepiness can affect daytime functioning. Moreover, psychiatric symptoms such as depression, anxiety, and irritability are frequently reported [4]. Previous studies have explored the relationship between BRSD and psychiatric

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symptoms. OSA could induce depression-like symptoms such as hypersomnolence, fatigue, lack of energy, and difficulty in concentration [4]. The reciprocal effects of BRSD and depression on each other were reported in a large cross-sectional study [5]. The risk of major depressive disorder in patients with OSA compared to normal individuals was 5.2 times and 2.4 times for the female and the male participants, respectively [6]. Depression and anxiety were also found in snorers [7]. However, previous studies have shown inconsistent results regarding the correlation between severity of BRSD (measured with AHI) and severity of psychiatric symptoms such as depression and anxiety. In a previous study, patients with OSA showed more intense somatic and depressive symptoms compared to patients with SS [4,8]. In contrast, other studies showed no significant differences in depression and anxiety between patients with OSA and patients with SS [7,9,10]. We found more severe psychiatric symptoms in the SS group when compared with the OSA group in our previous study [11].

The relationship between severity of BRSD and psychiatric symptoms is still unclear. It is difficult to explain the psychological symptoms of BRSD using the macrostructure of PSG. Many psychiatric disorders including psychotic disorder and depressive disorder are associated with symptoms of sleep disturbance from the early phases of these disorders [12]. Sleep abnormalities have been associated with severity of psychiatric symptoms [13]. Sleep disturbances often precede psychotic or depressive episodes [14,15]. Healthy individuals with a high genetic load for psychiatric disorders showed sleep pattern similar to the sleep pattern in depression observed on PSG. This result suggested the possibility of sleep pattern being a trait marker for vulnerability to psychiatric disorders [16]. In addition, previous studies have analyzed the relationship of sleep electroencephalography (EEG) with mental disorders, psychiatric symptoms, and treatment responses to medication [17-20]. Fast sleep spindle reduction on sleep EEG and its association with cognitive impairment was observed in schizophrenia [17]. Depression is characterized by an overall decrease in the slow-wave activities, which is related to elevated anxiety and depressed mood the following morning [19]. Therefore, we can postulate that the psychiatric symptoms in BRSD may be related to sleep EEG findings.

Researchers have previously attempted to investigate the microstructure of PSG-derived sleep as well as waking quantitative EEG (qEEG) in BRSD [21-25]. The sigma (13–15 Hz) power of sleep EEG predicted next-day sleepiness, as measured by the multiple sleep latency test [21]. Low-resolution electromagnetic tomography and sleep and wake qEEG analyses showed decreased alpha activity in the right posterior cingulate gyrus in patients with severe OSA than in patients having mild OSA [24]. Despite the results of many spectral analyses of sleep and wake qEEG in OSA or BRSD patients, no study has analyzed the association between psychiatric symptoms and spectral power densities of sleep EEG in patients with BRSD, to the best of our knowledge.

Our goal was to study the relationship between the spectral power density of sleep EEG and the degree of psychiatric symptoms in BRSD patients. If a significant correlation between the power spectra in a specific sleep EEG band and psychiatric symptoms could be found, we also aimed to find whether severity of BRSD and demographic characteristics in addition to psychiatric symptoms could predict EEG power of the specific spectrum.

METHODS

Participants

We recruited adults (aged 18–65 years) with BRSD from the Gachon Sleep Medicine Center in Gil Medical Center from March 2012 to February 2016. Among the 205 participants recruited through screening scales and structured clinical interviews, 169 were included in the final analyses. All participants had symptoms of BRSD such as frequent snoring, daytime sleepiness, and witnessed apnea. All participants met the BRSD diagnostic criteria according to DSM-IV-TR [1]. Diagnoses of BRSD of the participants were confirmed by interviews with board-certified medical doctors in the departments of otolaryngology, psychiatry, and neurology who had experience of over 5 years in sleep medicine and BRSD.

We excluded participants with following conditions: 1) current or past history of major psychiatric disorders; 2) comorbidities of severe medical conditions; 3) previous diagnosis with OSA; 4) history of uvulopalatopharyngoplasty; and 5) other suspected major sleep disorders such as rapid eye movement (REM) sleep behavior disorder, restless legs syndrome, circadian rhythm sleep disorder, or narcolepsy.

All participants provided written informed consent and the Institutional Review Board of Gil Medical Center approved the study protocols (GIRBA2764-2012). We followed the relevant guidelines and regulations for all experiments throughout the study period.

Questionnaire for the Evaluation of the Psychiatric Symptoms

All participants completed a questionnaire about psychiatric symptoms, namely the Symptom Checklist-90-Revised (SCL-90-R) scale comprising of 90 items [26]. SCL-90-R is a self-report inventory designed to evaluate patterns of psychological symptoms in psychiatric and medical patients. The results are shown as nine domain scores of primary symptom dimensions including somatization, obsessive-compulsive, depression, interpersonal sensitivity, anxiety, hostility, phobic anxiety, paranoia, and psychotic symptoms and three global indices, namely the global severity index, the positive symptoms total, and the positive symptoms distress index. Each question is scored based on a 5-point Likert scale from 0 (none) to 4 (extreme). We used the Korean version of SCL-90-R validated in Korean population [27].

Polysomnography

All participants underwent laboratory night PSG in accordance with the recommendations of American Academy of Sleep Medicine (AASM) [28]. Standard PSG recordings were conducted using the COMET system (Grass-Telefactor Corporation, West Warwick, RI, USA) with six EEG leads (F3, F4, C3, C4, O1, and O2), two electrooculogram channels (E1-M2 and E2-M2), three electromyography channels, and an electrocardiography channel. The PSG parameters were scored based on the criteria in the AASM manual [28]. According to the recommended rules of the AASM manual, we defined hypopnea as $\geq$ 30% reduction in nasal pressure signal excursions from baseline lasting $\geq$ 10 seconds associated with $\geq$ 4% desaturation from the pre-event baseline [28]. All PSG recordings were conducted by experienced PSG technologists who underwent and completed the interscorer reliability program (http://www.aasmnet.org/isr/) using the AASM sleep scoring criteria [28]. A board-certified neurologist and sleep specialist medical doctor (K.H.P.) confirmed all PSG data.

Spectral Analysis

We computed power spectra for each EEG frequency band, namely slow oscillation band (0.5−1 Hz), delta (1−4 Hz), theta (4−8 Hz), alpha (8−12 Hz), sigma (12−15 Hz), and beta (15−20 Hz). In the present analysis, we used the data derived from the average non-REM (NREM) central EEG electrode ([C3/A2 + C4/A1] / 2).

We used SpectralTrainFig (National Sleep Research Resource, Boston, MI, USA) for the spectral power analysis (https://github.com/nsrr/SpectralTrainFig) [29,30]. This open-source Matlab graphic interface is designed for the spectral analysis of sleep EEG in PSG. It automatically detects and deletes epochs with artifacts and yields summary figures for visual adjudication [31,32]. According to Welch's method, it calculates the spectral power density using ten overlapping 4-second sub-epochs for each 30-second epoch with a 50% tapered cosine window. It removes the artifact resulting from electrocardiogram interference using a template subtraction method [33]. Manual visual adjudication and exclusion of spectral data with significant artifacts were conducted by a researcher who was blinded to the information about the participants.

Statistical Analysis

Descriptive statistics were used for the characterization of the participants and Pearson correlation analysis was used to find the correlation between the SCL-90-R score and the absolute spectral EEG power in the participants. For the SCL-90-R variables that deviated from normal distribution, log transformation was conducted before the correlation analysis. Significance was defined as $p < 0.050$ after Bonferroni correction calculated by uncorrected p value × 9, as 9 was the number of domains on the SCL-90-R scale. The linear multiple regression model with stepwise method was used to find the best predictive factor for the specific EEG band significantly correlated with SCL-90-R. In the regression model, the independent variables included demographic information (age and sex), severity of BRSD (AHI), and psychiatric symptom domain scores (SCL-90-R) with significant correlation. IBM SPSS Statistics for Windows ver. 23 (IBM Corp., Armonk, NY, USA) was used for all analyses with statistical significance set at $p < 0.050$ (two-tailed).

RESULTS

Demographic and PSG Characteristics of the Participants

Demographic and PSG characteristics of the participants are presented in Table 1. The average age was 45.7 years and male subjects constituted 84.6% of the total sample (total 169 participants). The description of the PSG data is shown in Table 1. The average total sleep time was 338.0 minutes and sleep efficiency was 82.1%; average AHI was 24.5 per hour and arousal index was 31.1 per hour.

Table 1. Demographic data, polysomnographic findings, psychiatric symptoms, and spectral power density of participants (n = 169)

Variable	Statistics
Demographics	
Age (yr)	45.7 ± 11.3
Sex (male)	143 (84.6)
Body mass index (kg/m^2)	25.7 ± 3.5
Polysomnographic data	
Sleep and wake time	
Time in bed (min)	412.1 ± 28.9
Total sleep time (min)	338.0 ± 56.0
Sleep latency (min)	13.5 ± 21.9
Sleep efficiency (%)	82.1 ± 13.6
WASO (min)	59.8 ± 48.8
REM sleep latency (min)	124.4 ± 62.3
Sleep stage (%)	
N1	27.4 ± 15.8
N2	53.0 ± 14.4
N3	3.4 ± 5.6
R	15.9 ± 7.1
Respiration	
AHI (events per hour)	24.5 ± 22.6
Oxygen desaturation index	20.6 ± 20.2
Lowest O_2 saturation	80.9 ± 8.8
Arousal index	31.1 ± 17.9
SCL-90-R scores	
Somatization	50.8 ± 11.9
Obsessive-compulsive	44.5 ± 9.3
Interpersonal sensitivity	41.0 ± 9.8
Depression	45.4 ± 11.0
Anxiety	44.1 ± 9.1
Hostility	44.2 ± 7.7
Phobic anxiety	43.8 ± 7.4
Paranoid ideation	42.5 ± 9.0
Psychoticism	44.4 ± 7.7
Global severity index	32.5 ± 0.5
Positive symptom total	42.6 ± 11.3
Positive symptom distress index	43.8 ± 12.6
Spectral power density[a] during NREM sleep	
Slow oscillation (0.5−1 Hz)	2.07 ± 0.36
Delta (1−4 Hz)	1.19 ± 0.19
Theta (4−8 Hz)	0.63 ± 0.22
Alpha (8−12 Hz)	0.34 ± 0.24
Sigma (12−15 Hz)	0.12 ± 0.22
Beta (15−20 Hz)	−0.38 ± 0.23

Values are presented as mean ± standard deviation or number (%).
WASO, wake time after sleep onset; REM, rapid eye movement; N1, non-REM (NREM) stage 1; N2, NREM stage 2; N3, NREM stage 3; R, REM stage; AHI, apnea hypopnea index; SCL-90-R, Symptom Checklist-90-Revised NREM, non-rapid eye movement.
[a]Log-transformed spectral power density (log10 μV2).

SCL-90-R Scores and Absolute Spectral EEG Power of Participants

Table 1 presents the description of the domain scores for nine primary symptom dimensions and three global indices of the SCL-90-R scale. The mean domain scores of the nine dimensions ranged from 41.0 to 50.8.

The absolute spectral EEG power of the participants is presented in Table 1. The log-transformed absolute spectral power densities were 2.07 ± 0.36, 1.19 ± 0.19, 0.63 ± 0.22, 0.34 ± 0.24, 0.12 ± 0.22, and −0.38 ± 0.23 for slow oscillation band, delta, theta, alpha, sigma, and beta band, respectively.

Correlation Analysis between Absolute Spectral Power Density and Domain Scores of SCL-90-R

Table 2 presents the results of the correlation analyses between absolute spectral power density and domain scores of the nine primary symptom dimensions in the SCL-90-R scale. Log transformation for domain scores of SCL-90-R was performed before Pearson correlation analysis when the variables deviated from normal distribution. Slow oscillation during NREM sleep significantly correlated with paranoid ideation score ($r = 0.226$, $p = 0.003$, corrected $p = 0.028$; Table 2) and with depression score ($r = 0.216$, $p = 0.005$, corrected $p = 0.044$; Table 2).

Multiple Linear Regression Models for Slow Oscillation Power during NREM Sleep

The results of the multiple linear regression models using slow oscillation power that significantly correlated with paranoid ideation and depression scores of SCL-90-R as the dependent variable are presented in Table 3. In the multiple regression model, higher slow oscillation power was found to be predicted by higher paranoid ideation domain score (B = 0.007, $p = 0.020$), younger age (B = −0.011, $p < 0.001$), and female sex (B = 0.213, $p = 0.004$). AHI ($p = 0.13$) and depression domain score ($p = 0.77$) were excluded from the stepwise multiple linear regression model.

Table 2. Correlation analysis between SCL-90-R domain scores and absolute spectral power densities of sleep EEG during NREM sleep

Variable	Slow oscillation			Delta			Theta		
	r^a	p value	p corr	r^a	p value	p corr	r^a	p value	p corr
SOM	−0.023	0.77	> 0.99	0.119	0.12	> 0.99	0.135	0.080	0.72
O-C	0.203	0.008	0.072	0.144	0.061	0.54	0.096	0.21	> 0.99
I-S	0.204	0.008	0.072	0.088	0.25	> 0.99	0.032	0.67	> 0.99
DEP	0.216	0.005	0.044[b]	0.129	0.095	0.85	0.086	0.26	> 0.99
ANX	0.160	0.038	0.34	0.084	0.27	> 0.99	0.082	0.29	> 0.99
HOS	0.180	0.019	0.17	0.130	0.093	0.83	0.115	0.13	> 0.99
PHOB	0.173	0.025	0.22	0.075	0.33	> 0.99	0.098	0.20	> 0.99
PAR	0.226	0.003	0.028[b]	0.061	0.43	> 0.99	0.021	0.78	> 0.99
PSY	0.099	0.20	> 0.99	0.078	0.31	> 0.99	0.080	0.30	> 0.99
	Alpha			Sigma			Beta		
	r^a	p value	p corr	r^a	p value	p corr	r^a	p value	p corr
SOM	0.146	0.058	0.52	0.167	0.030	0.27	0.111	0.15	> 0.99
O-C	0.111	0.15	> 0.99	0.130	0.093	0.83	0.059	0.44	> 0.99
I-S	0.042	0.59	> 0.99	0.092	0.23	> 0.99	0.041	0.59	> 0.99
DEP	0.081	0.29	> 0.99	0.088	0.25	> 0.99	0.054	0.48	> 0.99
ANX	0.099	0.20	> 0.99	0.126	0.10	0.92	0.080	0.30	> 0.99
HOS	0.077	0.32	> 0.99	0.100	0.19	> 0.99	0.067	0.38	> 0.99
PHOB	0.065	0.39	> 0.99	0.086	0.26	> 0.99	0.084	0.27	> 0.99
PAR	−0.006	0.94	> 0.99	0.026	0.73	> 0.99	0.007	0.93	> 0.99
PSY	0.081	0.29	> 0.99	0.130	0.092	0.82	0.082	0.28	> 0.99

SCL-90-R, Symptom Checklist-90-Revised; EEG, electroencephalography; NREM, non-rapid eye movement r, Pearson's r p corr, p value after Bonferroni correction (uncorrected p value × 9) for correction of multiple comparisons; SOM, somatization; O-C, obsessive-compulsive; I-S, interpersonal sensitivity; DEP, depression; ANX, anxiety; HOS, hostility; PHOB, phobic anxiety; PAR, paranoid ideation; PSY, psychoticism.
[a]Log transformation for domain scores of SCL-90-R was performed before Pearson correlation analysis when the variables deviated from normal distribution. [b]Significant after Bonferroni correction ($p < 0.050$).

Table 3. Multiple linear regression models for sleep oscillation power during NREM sleep

Independent variable	Slow oscillation				
	B	Standard error	β	p value	R^2
Age	−0.011	0.002	−0.356	< 0.001[a]	0.20
Sex	0.213	0.072	0.213	0.004[a]	
PAR of SCL-90-R	0.007	0.003	0.167	0.020[a]	
Excluded variable					
AHI				0.13	
DEP of SCL-90-R				0.77	

Dependent variable: slow oscillation power during NREM sleep.
Multiple regression model: independent variables included age, sex, PAR domain score of SCL-90-R, DEP domain score of SCL-90-R, and AHI. AHI and DEP scores were excluded from the stepwise multiple linear regression model.
NREM, non-rapid eye movement PAR, paranoid ideation; SCL-90-R, Symptom Checklist-90-Revised; AHI, apnea-hypopnea index; DEP, depression.
[a]$p < 0.050$.

DISCUSSION

We conducted spectral power analysis of PSG-derived sleep EEG in patients with BRSD and analyzed the correlation between spectral power density and domain scores of SCL-90-R. The significant finding of the present study was the positive correlation between slow oscillation during NREM sleep and paranoid ideation and depression domain scores of SCL-90-R. In addition, multiple linear regression analysis showed that younger age, female sex, and paranoid ideation domain score had a significant effect on increased slow oscillation during NREM sleep.

The domain scores of SCL-90-R in our study were mostly between 40 and 50. Only the domain score for somatization was over 50 points, which is the cut-off score of abnormality [27]. The domain scores were similar to those reported in a previous Korean study that compared the SCL-90-R scores between tinnitus patients without psychiatric illness and control subjects [34].

In the present study, slow oscillation during NREM

sleep was significantly correlated with paranoid ideation and depressive symptoms. Slow oscillation has not been sufficiently studied in previous human studies. Hence, its clinical significance has not been established. Slow oscillations (< 1 Hz) in the NREM sleep EEG originate from the cortical neurons of the brain and are known to alternate between a depolarized up-state and a hyperpolarized down-state in association with slow membrane potential fluctuation [35]. A previous study has reported slow oscillation increases while sleeping after prolonged wakefulness [35]. NREM sleep in patients with post-traumatic stress disorder showed decreased slow oscillation power and increased higher frequency activity compared to that in control subjects. On the contrary, the slow oscillation power during REM sleep increased in the occipital area [36]. In addition, a study comparing sleep EEG among subjects with primary insomnia, chronic fatigue syndrome, and normal controls showed consistently lower proportion of slow oscillation power during slow wave sleep in primary insomnia and chronic fatigue syndrome, although the proportion of slow wave sleep duration varied among the groups [37]. A large-scale computer model study of the thalamocortical system during sleep found that a decrease in the cortical synaptic strength could explain the decrease in the slow-wave activity including slow oscillation during sleep [38]. In the present study, as the domain scores of paranoid ideation and depression increased, the spectral power density in slow oscillation increased during NREM sleep and paranoid ideation significantly predicted slow oscillation in the regression analyses. Little is known about the relationship between paranoia and BRSD. A large previous cohort study has reported increased prevalence of psychotic disorder in patients with OSA [39], and case studies have found increased psychotic depression [40], hallucination, and delirium in OSA patients [41], and improved psychotic symptoms after treating sleep apnea [42]. Association between psychotic symptoms and sleep EEG has not been investigated in BRSD patients; however, low delta [43] and high alpha power [44] in schizophrenia patients have been reported. The association between psychiatric symptoms and spectral power density in BRSD has not been investigated, and our results seem inconsistent with the results of previous studies in schizophrenia patients. This inconsistency may be attributed to the exclusion of subjects with major psychiatric disorders in our study. In addition, we believe that slow oscillation increased as a compensatory mechanism to overcome sleep insufficiency due to increased psychiatric symptoms. However, it is difficult to draw inferences regarding the cause-effect relationship due to the limitations posed by the cross-sectional nature of this study.

Results of the regression analysis revealed that slow oscillation decreased with age and increased in female sex. Previous studies have reported that the number and the amplitude of slow oscillation decreases with increasing age [45-47]. In addition, it seems that slow oscillation and sleep spindle generation and their coupling is also impaired in old age [48]. This is presumably a secondary result of normal age-related changes in the brain structure [46,49]. Young adult female have shown greater amount of slow wave sleep during NREM sleep than male in a previous study [50]. Older female showed more amount of slow wave sleep than older male of the same age during their sleep [51,52], although females usually complain of insomnia more often and no interaction effect of age and sex on slow wave sleep emerged in another sleep qEEG study [53]. The relationship among slow oscillation, age, and sex observed in the regression analysis of the present study is consistent with the results of the previous studies.

In the present study, AHI was not associated with slow oscillation during NREM sleep in BRSD patients. This result is in accordance with the results of previous studies showing weak relationship between AHI and symptoms of BRSD such as daytime sleepiness and sleep perception [54-56]. It has also been reported that psychiatric symptoms such as depression and anxiety were elevated in SS patients when compared with those in OSA patients [7]. We have also reported comparable results in previous studies and found no association among psychiatric symptoms, quality of life, and AHI in BRSD [11,57]. Thus, the results of the present result may suggest that the objective severity of BRSD is not associated even with the microstructural characteristics of BRSD patients.

To the best of our knowledge, there have been no spectral analysis studies regarding the relationship between psychiatric symptoms and sleep EEG in patients with BRSD. In a previous spectral analysis study of BRSD, sleep qEEG was used to investigate the EEG changes during transient hypoxia induced by apnea or hypopnea [58]. In another study that investigated EEG during periods of sleep apnea in REM and NREM sleep, the delta activity in-

creased progressively and was generalized over the posterior frontal, central, and parietal regions during NREM apneas [59]. A previous study has found EEG slowing in REM sleep during apnea over the frontal, central, and parietal regions in OSA patients when compared with normal controls [60]. It was also reported that decreased brain activity was associated with apnea events [61]. We found that an increase in slow oscillation was associated with paranoid ideation of the BRSD patients. This finding may suggest the compensatory role of slow oscillation activity in psychiatric symptoms of BRSD.

This study has several limitations. The cross-sectional design might limit conclusions regarding a causal relationship. We suggest future studies with a longitudinal design to investigate the effect of psychiatric symptoms on the microstructure of BRSD patients' sleep. Modest effect sizes (r = 0.226 for correlation and β = 0.167 for regression analyses) and relatively low values of paranoid ideation are other limitations of the results of the present study that might limit the explanatory power. Future studies with a large sample size and thorough evaluation of psychiatric symptoms, which would increase the power of the investigation, will be necessary.

This is the first study to investigate the relationship between psychiatric symptoms and sleep EEG in BRSD patients. We found a significant relationship between slow oscillation and psychiatric symptoms such as paranoid ideation and depression during NREM sleep. Factors associated with increased slow oscillation were younger age, female sex, and greater paranoid ideation, suggesting the compensatory role of slow oscillation in psychiatric symptoms of BRSD. Further prospective studies are needed in future to elucidate the relationship among varied phenotypes of BRSD, microstructure of PSG, and the mechanism of sleep EEG activation.

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■ Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

■ Author Contributions

Conceptualization: Seung-Gul Kang. Data curation: Seon Tae Kim, Gun Bae Lee, Kee Hyung Park, and Seung-Gul Kang. Formal analysis: Jae Myeong Kang and Seung-Gul Kang. Funding acquisition: Seung-Gul Kang. Investigation: Jae Myeong Kang, Seo-Eun Cho, and Seung-Gul Kang. Methodology: Seon Tae Kim, Kee Hyung Park, and Seung-Gul Kang. Resources: Seon Tae Kim, Seo-Eun Cho, Gun Bae Lee, Kee Hyung Park, and Seung-Gul Kang. Supervision: Seung-Gul Kang.

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