Research article

Alterations of awake EEG in idiopathic REM sleep behavior disorder without cognitive impairment

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HIGHLIGHTS

• There are EEG changes at waking state in iRBD without cognitive impairment.
• Higher absolute alpha power is found in the frontal region of iRBD compared to control.
• Dominant occipital frequency is lower in iRBD than in controls.

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ABSTRACT

The aim of this study was to find electroencephalographic (EEG) changes in subjects with drug-naive idiopathic rapid eye movement sleep behavior disorder (iRBD) who had no cognitive impairment. A total of 57 iRBD patients confirmed by polysomnography (PSG) and 33 sex and age-matched healthy controls were included and their waking EEG was recorded from five cortical regions for 15 min. Power spectral analyses by fast Fourier transforms were performed on EEG data. In PSG data, the iRBD patients showed sleep disturbances of short total sleep time, decreased sleep efficiency, increased sleep latency and frequent awakening compared to controls. After adjusting for sleep parameters, the absolute alpha power in frontal region in iRBD patients was higher than that in controls (1.2 ± 0.3 vs. 0.9 ± 0.3, p = 0.037). Dominant occipital frequency (DOF) was lower in iRBD patients than in controls after adjusting for the sleep covariates (9.2 ± 0.3 Hz vs. 9.5 ± 0.4 Hz, F = 8, p = 0.006). iRBD patients without cognitive impairment also showed EEG alteration in frontal and occipital cortex at wakefulness, which could be an early marker of cerebral dysfunction in iRBD patients.

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1. Introduction

REM sleep behavior disorder (RBD) is characterized by the absence of muscle atonia during REM sleep, resulting in acting out dreams with sometimes violent or injurious consequences. Idiopathic RBD (iRBD) usually affects older people [1], and many iRBD patients eventually develop a variety of neurodegenerative disorders including dementia with Lewy body disease (DLB), Parkinson’s disease (PD) and multiple system atrophy (MSA) [2–4]. Quantitative electroencephalographic (EEG) analysis has been used to measure cortical activity and assess an underlying pathological process in neurodegenerative disorders, because EEG power analysis provides high temporal resolution and is useful in detecting subtle changes of brain function. Waking EEG slowing is commonly seen in neurodegenerative disorders and various kinds of abnormalities are observed such as global generalized slowing in Alzheimer’s disease (AD), and posterior and temporal cortical slowing in DLB, PD and mild cognitive impairment (MCI) [5–7]. Due to the growing evidence of iRBD as a high risk factor of neurodegenerative disorders, recent studies have been published with regard to EEG abnormalities during wakefulness and sleep in iRBD patients [8–10].

MCI occurs in approximately half of iRBD patients [11], with the remainder retaining grossly normal cognition. Therefore, it could be meaningful to investigate EEG characteristics of iRBD without cognitive impairment to obtain comprehensive understanding of cerebral dysfunction in iRBD. No EEG change in iRBD without MCI has been found so far. However, previous studies had several limitations [9,10]. These comprise a small number of participants and

lack of consideration of total cortical regions with only central and occipital areas examined [9], and inclusion of participants who were taking medications for RBD treatment that can cause changes in the EEG spectrum [10]. Thus, the aim of our study was to investigate waking EEG changes in drug-naïve iRBD patients without cognitive impairment in a relatively large sample.

2. Methods

2.1. Participants

Patients with RBD were enrolled from the sleep clinic at Seoul National University Bundang Hospital from April 2014 to August 2015, and healthy controls were recruited by placing advertisements in the hospital and local newspapers. Patients with degenerative disorders such as PD, MSA, AD and DLB or MCI were excluded from this study. None of the participants showed sleep apnea syndrome, narcolepsy, history of major psychiatric disorders, substance use disorders or any history of brain injury. After evaluation of cognitive function by the Korean version of the Consortium to Establish a Registry for Alzheimer’s Disease Assessment Packet Neuropsychological Assessment Battery (CERAD-K-N) [12], we excluded those who had MCI in all subjects. Additional information about depressive symptoms [13], subjective sleep quality [14] and daytime sleepiness [15] was obtained. All participants were informed of the purpose and procedures of this study and gave written consent. The present study was approved by the Institutional Review Board of Seoul National University Bundang Hospital (B-1408/264-004).

2.2. Diagnosis of RBD and video-polysomnography (vPSG)

RBD was diagnosed according to the standard criteria of the International Classification of Sleep Disorders—second edition (ICSD-2) [16]. In addition, REM sleep without atonia (RSWA) was strictly defined using the American Academy of Sleep Medicine (AASM) manual [17]. All subjects underwent overnight full PSG using the Embla™ N7000 device (Embla, Reykjavik, Iceland) with standard electrodes and sensors. EEG electrodes were applied at F4/A1, F3/A2, C4/A1, C3/A2, O1/A2 and O2/A1 according to the International 10–20 System of Electrode Placement and two electrooculography electrodes were applied. Electromyography electrodes were applied at the submentalis, flexor digitorum superficialis and both anterior tibialis muscles. Strain gages were used for recording chest and abdominal respiratory movements and nasal pressure cannulas were used to record airflow. Electrocardiogram was applied and oxygen saturation was measured using a pulse oximeter applied to the index finger. Sleep was scored at every 30-s epoch of the nocturnal PSG, and sleep stages were scored visually according to current criteria with allowance for REM sleep without atonia [17].

2.3. Quantitative EEG evaluation

EEG was recorded in a sitting position for 15 min and subjects were instructed to relax. For the first 7 min, subjects kept their eyes closed. For the next minute, subjects opened their eyes and for the last 7 min subjects kept their eyes closed again. The subjects were under surveillance of an examiner to check and prevent drowsiness. EEG electrodes were placed according to the international 10–20 system at FP1, FP2, F3, F4, F7, F8, C3, C4, P3, P4, T3, T4, T5, T6, O1 and O2, with an average reference. Recording of EEG started when electrical impedance of all electrodes was below 5 kΩ and EEG signals were sampled at 1000 Hz and digitalized. The high pass filter was set to 100 Hz with the low pass filter set to 0.3 Hz. An artifact-free 120-s EEG recording with the eyes closed (24 epochs of 5-s EEG segments) was selected by visual analysis and artifacts comprised muscle activity, small body movements, eyelid movements and micro-sleep. Spectral analysis was done by the fast Fourier transform. Absolute power values of five bands at each electrode were computed; 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 (25.0–30.0 Hz) bands. The absolute power of each electrode were grouped into five cerebral regions and mean values were calculated. As there was no significant asymmetry in any of the five bands, we averaged left and right electrodes. The five cerebral regions investigated were the frontal (FP1, FP2, F3, F4, F7 and F8), central (C3 and C4), parietal (P3 and P4), temporal (T3, T4, T5, and T6) and occipital (O1 and O2) regions. The dominant occipital frequency (DOF) during waking stage was calculated by averaging peak power of two occipital electrodes. All the EEG analysis was blinded as to the participants. In addition, for evaluating vigilance alterations during the EEG recording, we manually classified vigilance stages of our subjects by using a vigilance related-algorithm; Vigilance Algorithm Leipzig.

2.4. Statistical analyses

Results are reported as means ± standard deviations. Comparisons of demographic, clinical, PSG variables and EEG between two groups were performed using the independent t-test or χ² test, and the Mann-Whitney U test was conducted for data non-normally distributed. Absolute power and mean power were log-transformed to normalize the data distribution. Between group-differences in absolute power were assessed by two-way analysis of variance (ANOVA) for each cortical region, with group as an independent factor and the five frequency bands as repeated measures. We also compared the EEG characteristics after adjusting for differences between two groups as covariates by using two-way analysis of covariance (ANCOVA). All significance tests were two sided and P-value was set at <0.05. SPSS version 22.0 for Windows (SPSS Inc. Chicago, IL, USA) was used for all the analysis.

3. Results

3.1. Demographic, clinical and neuropsychological data

The process of including participants for present study is summarized in Fig. 1. Ninety-five patients were diagnosed with RBD at baseline. Among these 95 RBD patients, 12 were found to have neurodegenerative disorders comprising AD (n = 2), PD (n = 9), MSA (n = 1). One patient did not complete the full evaluation. We further excluded iRBD patients with MCI (n = 10) or sleep apnea syndrome (n = 12). Thirty six sex- and age-matched healthy controls were included; three of them were diagnosed as MCI (8.3%) and were excluded. Three iRBD patients were excluded from the analysis due to QEEG data of poor quality. Finally, 57 iRBD patients (33 men; mean age, 66 ± 6.1) and 33 controls (18 men; mean age, 63.9 ± 5.6) were analyzed. The mean duration of iRBD symptom was 5.7 ± 8.4 years. There was no difference in sex, age, educational level, depressive symptoms, subjective sleep quality, daytime sleepiness and neurocognitive function between iRBD patients and controls (Table 1). Cognitive functions including memory, executive functioning and visuospatial abilities of the iRBD patients were not different from those of controls.

3.2. PSG and EEG data

In PSG data, total sleep time (348.5 ± 49.9 min vs. 389.4 ± 52.9 min, p < 0.001) and sleep efficiency (74 ± 10.3 vs. 82.5 ± 9.2%, p < 0.001) were decreased, and sleep latency (30.6 ± 39 vs. 15.1 ± 21.6 min, p = 0.003) and wake after sleep onset (93 ± 47 vs. 67.2 ± 35.3 min,
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