Investigation and Analysis of Sleep Status in Patients with Vascular Depression

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SUMMARY

Background: To investigate the sleep status of patients with vascular depression (VD) and analyze the characteristics of sleep by polysomnogram.

Methods: 50 VD patients, 30 patients with non-vascular depression (NVD) and 50 normal subjects were enrolled. All subjects were evaluated by the Hamilton Depression Rating Scale (HAM-D), Hamilton Anxiety Scale (HAM-A), Pittsburgh Sleep Quality Index (PSQI) and Epworth sleepiness scale (ESS). All these patients were monitored by polysomnography (PSG).

Results: The PSQI scores of the VD patients didn’t show difference compared with that of NVD, but both of them were significantly higher than the normal. The ESS scores of VD patients were significantly higher than other groups. The total sleep time (TST), sleep efficiency (SE) and sleep maintenance (SMT) in NVD patients and VD patients were poorer, and the waking time after sleep onset (WASO) and sleep latency (SL) were significantly longer. The N1/N2 ratio was longer, and the N3/REM ratio was shorter in NVD patients and VD patients. The HAMD score was negatively correlated with RL and positively with RD, RA and RI. The RL of VD patients was longer than the normal, and RD and RI were higher in VD patients; there was no difference in RA. The obstructivity and apnea-hypopnea index (AHI) significantly increased in VD patients.

Conclusion: The characteristic PSG findings of the NVD patients were shortening REM sleep latency and REM disinhibition. Characteristics of VD with PSG patients were sleep-related breathing disorders (SRBD) and daytime sleepiness, and disorders of 24-h sleep structure.

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1. Introduction

Vascular depression (VD) is a late-life depression with severe harmfulness. VD was first proposed by Alexopoulos and Krishmn in 1997, which included late-onset depression (depression firstly developed in old age), depression with white matter lesions (WML), or cerebral infarction, depression with cardiovascular risk factors and cognitive impairment, and depression with poor sensitivity to antidepressants and electroconvulsive therapy.1-5 VD is common in the elderly. It is characterized by easy fatigue, highlighted somatization, retardation of psychomotor, and lack of pleasure. With the aging of the population and the increase in the incidence of cardiovascular and cerebrovascular disease, the incidence of VD has also increased. Imaging studies have revealed that 94% of patients with late-onset severe depression had developed asymptomatic stroke. Therefore, late-onset depression is mostly VD.6-9 Sleep disorders can be used as predictors of mental diseases, particularly for depression; and disordered sleep can often be used as a precursor and may be the only clinical symptom of 10. Sleep disorders in VD patients mainly manifest as sleep maintenance difficulty, early awakening and excessive sleep.3-11 Since its pathogenesis is different from NVD, sleep disorders in VD patients have different characteristics. The Pittsburgh Sleep Quality Index (PSQI)12 and Epworth sleepiness scale (ESS)15 are the commonly used sleep assessment scales. In this study, the sleep quality of VD patients was investigated, and the sleep situations of VD patients at each stage were analyzed by polysomnography (PSG), in order to summarize the characteristics of sleep in VD patients, and diagnose and treat this disease early.

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2. Materials and Methods

This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of our hospital. Written informed consent was obtained from all participants.

2.1. Subjects

2.1.1. Inclusion criteria

A total of 50 VD patients, who were admitted in our hospital from September 2012 to September 2015, were enrolled into this study. Inclusion criteria for VD patients: patients who met the diagnostic criteria for depression in the Chinese Classification of Mental Disorders, Third Edition (CCMD-3), and had a Hamilton Depression Scale (HAMD) score $\geq 8$ points; patients who met the diagnostic criteria of vascular depression recommended by Alexopoulos et al. 12; depression occurs after the age of 65 years, patients have clinical and (or) laboratory evidence of cerebrovascular disease and vascular risk factors, and depression occurs between 6 and 12 months after the occurrence of cerebrovascular events. In addition, 30 NVD patients, who were outpatients or inpatients during the same period, were enrolled into this study. These patients met criterion 1, but had no clinical and (or) laboratory evidence of cerebrovascular disease and vascular risk factors. The control group comprised of 50 healthy elderly people, who had a HAMD score of $<8$ points. Differences in terms of age, gender, education level and other socio demographic data between these three groups were not statistically significant ($P > 0.05$).

2.1.2. Exclusion criteria

Patients who had serious body disease, mental disease and other diseases that affect night time sleep were excluded. Patients who had head and body tremors, alcohol and drug abuse history, and developed symptoms of infection and fever within one week were excluded. Patients who had not taken sedative hypnotic drugs and psychoactive substances, or previously taken these drugs but had discontinued its administration for more than three days before detection were included into the study. Patients who had primary sleep disorders were excluded. Patients who could not cooperate with the examination were excluded.

2.2. Methods

2.2.1. Scale tests

The sleep status of patients was evaluated by two professionals using HAMD, Hamilton Anxiety Scale (HAMA), PSQI and ESS. HAMD is the most commonly used scale for depression (24-item edition). A total score of $>35$ points indicates severe depression, a total score between 20 and 35 points moderately severe depression, a total score between 8 and 20 points mild depression, and a total score of $<8$ points indicates normal. The patient’s sleep situations were monitored using the PSG machine produced by Compumedic Siesta (Australia) for a duration of $>7$ h. Within 24 h before the examination, subjects were not allowed to take sedative drugs, or drink wine and tea. This monitoring was performed in the sleep apnea monitoring room. Polysomnography: Subjects were allowed to sleep for two nights in the sleep laboratory. One night was for adaptation, and the other night was for the PSG that was performed the whole night. The main indicators analyzed by the laboratory sleep monitoring system include the following aspects: (A) Sleep measurement and analysis, which includes the following: sleep process, including total sleep time (TST), sleep latency (SL), sleep efficiency (SE), waking time after sleep onset (WASO) and sleep maintenance (SMT); sleep structure, including sleep stages (S1, S2, S3 and REM), sleep duration and percentages; REM sleep indexes, including REM sleep latency (RL), REM activity (RA), REM intensity (RI) and REM density (RD), as well as the number of REM cycles. RL is the interval between the start times of S1 to the appearance of REM sleep. RT is the total time of all stages of REM sleep during the whole night sleep. RA, RI and RD: Each minute of REM sleep was divided into nine units and given a serial number ranging from 0 to 8, the number of rapid eye movement during each REM sleep period was calculated, the time was converted into the number of units, the number of units in all stages were summed up, and the result was RA. RI was the ratio of RA/TST. RD was the ratio of RA/REM sleep time. Breath analysis: Breath airflow was transnasally detected, the abdominal and abdominal breathing motion was detected using a stretch gauge, and blood oxygen saturation was detected at the same time. Apnea and hypopnea were determined according to current international standards, apnea-hypopnea index (AHI) was calculated, and the presence of obstructive sleep apnea syndrome (OSAS) was analyzed.

2.2.2. PSG monitoring

The patient’s sleep situations were monitored using the PSG machine produced by Compumedic Siesta (Australia) for a duration of $>7$ h. Within 24 h before the examination, subjects were not allowed to take sedative drugs, or drink wine and tea. This monitoring was performed in the sleep apnea monitoring room. Polysomnography: Subjects were allowed to sleep for two nights in the sleep laboratory. One night was for adaptation, and the other night was for the PSG that was performed the whole night. The main indicators analyzed by the laboratory sleep monitoring system include the following aspects: (A) Sleep measurement and analysis, which includes the following: sleep process, including total sleep time (TST), sleep latency (SL), sleep efficiency (SE), waking time after sleep onset (WASO) and sleep maintenance (SMT); sleep structure, including sleep stages (S1, S2, S3 and REM), sleep duration and percentages; REM sleep indexes, including REM sleep latency (RL), REM activity (RA), REM intensity (RI) and REM density (RD), as well as the number of REM cycles. RL is the interval between the start times of S1 to the appearance of REM sleep. RT is the total time of all stages of REM sleep during the whole night sleep. RA, RI and RD: Each minute of REM sleep was divided into nine units and given a serial number ranging from 0 to 8, the number of rapid eye movement during each REM sleep period was calculated, the time was converted into the number of units, the number of units in all stages were summed up, and the result was RA. RI was the ratio of RA/TST. RD was the ratio of RA/REM sleep time. Breath analysis: Breath airflow was transnasally detected, the abdominal and abdominal breathing motion was detected using a stretch gauge, and blood oxygen saturation was detected at the same time. Apnea and hypopnea were determined according to current international standards, apnea-hypopnea index (AHI) was calculated, and the presence of obstructive sleep apnea syndrome (OSAS) was analyzed.
90% of depression patients have complained insomnia, which may depression in the early stages. Previous studies have revealed that compared to normal subjects. The proportion of REM in VD patients and the N3/REM ratio was shorter in NVD patients and VD patients, Table 3

3.4. Correlation between PSG characteristics in the REM phase and HAMD in VD patients and NVD patients

The total HAMD score was negatively correlated with RL, but was positively correlated with RD, RA and RI. There was no significant correlation between the total HAMD score and changes in sleep structure.

Microarousal in VD patients increased, daytime sleepiness increased, the lowest and mean blood oxygen saturation significantly decreased, and obstructivity and AHI significantly increased, compared with normal subjects and NVD patients; and the differences were statistically significant (P < .05) (Table 4).

4. Discussion

Sleep disorder is one of the most common clinical symptoms of depression in the early stages. Previous studies have revealed that 90% of depression patients have complained insomnia, which may also be an early sign of a new depression attack.3–12 The main symptoms of sleep disorders in depression patients are sleep maintenance difficulty, early awakening and excessive sleep; and its characteristics are not so obvious. The sleep electroencephalogram manifestations of depression patients mainly include the following: prolongation of SL, sleep maintenance disorder, reduction of slow wave sleep (SWS) and inhibitory disorder in REM sleep13–15. Among these, changes in REM sleep indexes have characteristics that mainly include shortened RL of REM sleep, increased duration of REM sleep, increased activity, density and strength of REM sleep, and increased cycles of REM sleep. Among these, RL shortening is a biomarker for characteristic changes in endogenous depression, and is related to relatively stronger negative cognitive and negative emotional experience and terminal insomnia, which reflect the severity of depression. With RL shortening as an indicator, the sensitivity of diagnosing major depressive disorders is 35–95%, and its speciﬁcity is 62–100%. RL shortening is the strongest and most characteristic form of sleep disorder in depression.16–18 This study revealed that some aspects of sleep disorders were similar between NVD patients and VD patients, including prolonged SL, sleep maintenance disorder, reduction of SWS and sleep efficiency. These were consistent with the results of other studies.19,20 Our further research revealed that the total score of HAMD in VD patients was negatively correlated with RL, and was positively correlated with RD, RA and RI. This study also revealed the following differences between sleep disorders induced by VD and NVD: RL in VD patients was longer than in NVD patients; RA, RD and RI in VD patients were less than those in NVD patients, that is, the increase in EEC bewaking at REM phase in VD patients was not as obvious as that on NVD patients. Compared with NVD
patients, the daytime sleepiness of VD patients was more obvious, the ESS scores and AHI of VD patients were higher, VD patients were more likely to develop SRBD (apnea or hypopnea occurs in the sleep stage), patients had more obvious disorders of 24-h sleep structure, and there was no significant correlation between the total score of HAMD and changes in sleep structure. For the reasons of the difference in PSG between these two groups, it is considered to include the following aspects: VD patients have risk factors of cerebrovascular disease, and SRBD such as OSAS is one of the risk factors of cerebrovascular disease, which can cause daytime sleepiness, and also increase the disorder of sleep structure; Although there was no significant difference in MMSE scores between VD patients and NVD patients, MCI patients are usually characterized by RL prolongation in PSG. The nature of REM sleep remains unelucidated. Some scholars believe that REM sleep is a process to remove brain tissue metabolic wastes, while some scholars believe that this period is the process to prepare proteins and neurotransmitters that nerve cells need in the daytime. We speculate that the activity and intensity of REM sleep may be related to cognitive function to a certain extent. The specific reasons need to be further studied.

Although we achieved interesting results, there were limitations in our study. We did not quantify the breath analysis of PSG in patients with sleep apnea. The size of studied population may also be a limitation. Although we made some observations on cognitive function, the results are not satisfactory. Considering these facts, further investigation is still a necessity in this area.

5. Conclusion

The characteristic PSG findings of the NVD patients was shortening REM sleep latency and REM disinhibition. Characteristics of VD with PSG patients were sleep-related breathing disorders (SRBD) and daytime sleepiness, and disorders of 24-h sleep structure.

References