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Predictive Role of PSG Parameters on the Efficacy of Cognitive Behavioral Therapy for Insomnia (CBT-I)

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Abstract: *Objective*: To explore the predictive value of baseline polysomnography (PSG) parameters on the efficacy of cognitive behavioral therapy for insomnia (CBT-I) in the treatment of chronic insomnia, and to clarify its clinical application value. *Methods*: Forty patients with chronic insomnia who visited the Sleep Medicine Center of the hospital from March 2024 to June 2025 were selected. All patients completed 8 weeks of CBT-I treatment and PSG monitoring before and after treatment. The correlation between baseline PSG parameters (sleep efficiency, wake time after sleep onset, and proportion of N3 sleep stage) and the improvement values of the Insomnia Severity Index (ISI) and sleep efficiency after treatment was analyzed. Multiple regression analysis was used to screen predictive factors of efficacy. *Results*: Baseline sleep efficiency (r=0.36, $P<0.05$) was positively correlated with ISI improvement value, wake time after sleep onset (r=-0.33, $P<0.05$) was negatively correlated with ISI improvement value, and the proportion of N3 sleep stage (r=0.29, $P<0.05$) was positively correlated with ISI improvement value. Multiple regression analysis showed that baseline sleep efficiency (β=0.27, $P=0.03$) and wake time after sleep onset (β=-0.24, $P=0.04$) were independent predictors of ISI improvement value (adjusted $R^2=0.17$, F=5.12, $P<0.05$). *Conclusion*: Sleep efficiency and wake time after sleep onset among baseline PSG parameters can effectively predict the efficacy of CBT-I in the treatment of chronic insomnia, providing an objective basis for clinically screening treatment-sensitive populations.

Keywords: PSG parameters; Cognitive behavioral therapy; Insomnia

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1. Introduction

Sleep disorder is a common psychopathological disease, and chronic insomnia, as one of its primary clinical manifestations, severely affects the physical and mental health of patients [1]. According to statistics, more than 300 million people in China are currently suffering from sleep disorders, with chronic insomnia patients accounting for 42.9%. The treatment methods for insomnia include pharmacological therapy, cognitive behavioral therapy, etc. [2]. However, traditional pharmacological therapy has disadvantages such as recurrence and significant side effects. In recent years, cognitive behavioral therapy has begun to be widely used in clinical

practice and has achieved good therapeutic effects. There is a close relationship between sleep problems and brain function. Research has shown that the brain network is an essential component required for the body to maintain normal physiological activities during sleep, and different regions have specific neural connection patterns. The neural network characteristics and related mechanisms of the brain can be understood by constructing a functional connectivity network. Therefore, studying changes in brain function in patients with insomnia is significant for guiding insomnia treatment plans [3–4]. In this study, 40 patients with insomnia will receive 8 weeks of CBT-I treatment, and polysomnography (PSG) tests will be performed before and after treatment. Simultaneously, functional magnetic resonance imaging (fMRI) will be utilized to obtain standardized functional connectivity values (FC) for various brain regions at baseline and after treatment. Further analysis of the correlation between FC values in different brain regions and the efficacy of insomnia treatment will provide new insights for predicting the efficacy of CBT-I in treating insomnia, aiming to improve the scientific validity and effectiveness of CBT-I treatment.

2. Materials and methods

2.1. General information

A total of 40 patients with insomnia who visited the Sleep Medicine Center of the hospital from March 2024 to June 2025 were selected. Among the 40 subjects, there were 16 males (40.00%) and 24 females (60.00%); aged between 35–74 years, with an average age of 52.64 ± 10.32 years; the course of the disease ranged from 6 months to 12 years, with an average of 3.81 ± 2.52 years; ISI score before treatment was 14.22 ± 3.14 points, and sleep efficiency was 64.81 ± 9.62%. All patients completed 8 weeks of CBT-I treatment without any dropout cases. All patients completed 8 weeks of CBT-I treatment and efficacy evaluation without any dropout cases. All patients met the diagnostic criteria for “chronic insomnia” in the “Guidelines for the Diagnosis and Treatment of Insomnia in Chinese Adults” developed by the Chinese Sleep Research Society, which includes at least three months of the following symptoms [5]: (1) difficulty falling asleep; (2) sleep maintenance disorders; (3) early awakening; (4) decreased sleep quality; (5) shortened total sleep time. Exclusion criteria were: (1) comorbid mental illness or severe physical illness; (2) unable to cooperate and complete the experiment; (3) suffering from cerebral organic diseases, including cerebrovascular disease, intracranial infection, etc.; (4) taking drugs with central nervous system inhibitory effects.

2.2. Methods

2.2.1. PSG monitoring

Monitoring was conducted in a soundproof, blackout, and temperature-controlled (20–24 ℃) sleep laboratory using the Embla N7000 polysomnography system from the United States. One day before monitoring, patients were instructed to avoid excitatory drinks such as coffee and strong tea, refrain from taking sedatives, and maintain a normal sleep schedule.

The monitoring parameters include: (1) Electroencephalogram (C3-A2, C4-A1, O1-A2, O2-A1); (2) Electrooculogram (EOG); (3) Electromyogram (EMG) of chin muscles; (4) Electrocardiogram (ECG); (5) Respiratory airflow (nasal and oral); (6) Chest and abdominal movements; (7) Blood oxygen saturation (SpO2); (8) Electromyogram of lower limbs. Monitoring will take place from 22:00 that evening to 6:00 the next morning (total recording time of 8 hours). Professional technicians will perform sleep staging and parameter analysis according to the standards of the American Academy of Sleep Medicine (AASM). The main indicators include

sleep latency, total sleep time, sleep efficiency, number of awakenings after sleep onset, awakening time after sleep onset, and the proportion of N1, N2, N3, and REM sleep stages.

2.2.2. CBT-I treatment

A standardized 8-week CBT-I program will be adopted, including: (1) Sleep restriction therapy: Adjusting bedtime based on the patient's baseline total sleep time to gradually improve sleep efficiency; (2) Stimulus control therapy: Establishing a conditioned reflex between bed and sleep (using the bed only for sleep and sexual activity, and getting out of bed if not asleep within 20 minutes); (3) Cognitive reconstruction: Correcting patients' unreasonable beliefs about sleep (such as "I must sleep for 8 hours"); (4) Relaxation training: Including progressive muscle relaxation, abdominal breathing exercises, etc.; (5) Sleep hygiene education: Guiding regular sleep schedules, optimizing the sleep environment, etc. Individual therapy sessions will be conducted once a week (each session lasting 45–60 minutes) and will be implemented by a certified sleep technician.

2.3. Observation indicators

After 8 weeks of treatment, the efficacy was evaluated using the following indicators: ISI score, calculation of improvement value (ISI score before treatment — ISI score after treatment), with an improvement value of ≥4 considered as effective treatment [5]; PSG was re-examined, and the improvement value of sleep efficiency was calculated (SE after treatment — SE before treatment).

2.4. Statistical methods

SPSS software was used for statistical analysis of the data. The count data were expressed as n(%), and the comparison between groups was performed using a *t*-test. A P-value < 0.05 was considered statistically significant.

3. Results

3.1. Changes in sleep indicators before and after CBT-I treatment

After 8 weeks of treatment, both subjective and objective sleep indicators of the patients improved significantly (P<0.01) (**Table 1**).

Table 1. Comparison of sleep indicators before and after CBT-I treatment (Mean ± SD)

Indicator	Before treatment	After treatment	t-value	*P*-value
ISI Score (points)	14.56 ± 3.28	7.24 ± 2.86	18.36	<0.01
Sleep Latency (minutes)	38.65 ± 12.45	22.34 ± 8.76	10.25	<0.01
Total Sleep Time (minutes)	325.67 ± 56.89	389.45 ± 45.67	9.87	<0.01
Sleep Efficiency (%)	65.42 ± 10.36	82.56 ± 7.45	12.34	<0.01
Number of Awakenings (times)	4.56 ± 1.89	2.12 ± 1.05	11.56	<0.01
Wake After Sleep Onset (minutes)	56.78 ± 20.34	25.43 ± 12.67	10.78	<0.01
N3 Sleep Percentage (%)	12.34 ± 4.56	18.76 ± 5.23	8.65	<0.01
REM Sleep Percentage (%)	18.65 ± 5.34	22.34 ± 4.87	5.43	<0.01

3.2. Correlation analysis between baseline PSG parameters and the efficacy of CBT-I

Using the improvement values of ISI and sleep efficiency as indicators of efficacy, Pearson correlation analysis was conducted. The results showed that baseline sleep efficiency was positively correlated with the improvement value of ISI (r=0.38, $P<0.01$) and negatively correlated with the improvement value of sleep efficiency (r=-0.35, $P<0.01$). Baseline wake time after sleep onset was negatively correlated with the improvement value of ISI (r=-0.32, $P<0.01$) and positively correlated with the improvement value of sleep efficiency (r=0.29, $P<0.05$). The proportion of N3 sleep at baseline was positively correlated with the improvement value of ISI (r=0.29, $P<0.05$) and negatively correlated with the improvement value of sleep efficiency (r=-0.26, $P<0.05$). Other PSG parameters (such as sleep latency, proportion of REM sleep, etc.) were not significantly correlated with efficacy indicators ($P>0.05$).

3.3. Multiple regression analysis of CBT-I efficacy

Using the improvement value of ISI as the dependent variable, multiple linear regression analysis was conducted with variables that had $P<0.10$ in univariate analysis (baseline sleep efficiency, wake time after sleep onset, proportion of N3 sleep) as independent variables. The results showed that baseline sleep efficiency (β=0.28, $P=0.02$) and wake time after sleep onset (β=-0.25, $P=0.03$) were independent predictors of ISI improvement. The adjusted $R^2=0.18$, and the overall model was statistically significant (F=5.67, $P<0.01$) (**Table 2**).

Table 2. Results of multiple regression analysis for CBT-I efficacy (ISI improvement value)

Independent variable	β-value	Standard error	t-value	*P*-value
Baseline Sleep Efficiency	0.28	0.11	2.56	0.02
Baseline Wake After Sleep Onset (WASO)	-0.25	0.10	-2.43	0.03
Baseline N3 Sleep Percentage	0.18	0.10	1.76	0.08
Intercept (Constant)	5.67	1.23	4.61	<0.01

Regression analysis with sleep efficiency improvement as the dependent variable showed that baseline wake time after sleep onset (β=0.27, $P=0.02$) was an independent predictor, with an adjusted $R^2=0.08$ (F=3.21, $P=0.04$).

3.5. Comparison of treatment effects among patients with different baseline sleep efficiencies

Patients were divided into a low sleep efficiency group (≤65.42%, $n=60$) and a high sleep efficiency group (>65.42%, $n=60$) based on the median baseline sleep efficiency (65.42%). The CBT-I treatment effects were compared between the two groups. The results showed that the ISI improvement score in the low sleep efficiency group (8.65±3.24) was significantly higher than that in the high sleep efficiency group (5.34±2.87) (t=5.87, $P<0.01$). Additionally, the sleep efficiency improvement in the low sleep efficiency group 22.34 ± 8.76% was significantly higher than that in the high sleep efficiency group 12.56 ± 7.45% (t=6.54, $P<0.01$).

4. Discussion

The pathogenesis of chronic insomnia is closely related to abnormalities in the sleep-wake regulatory network, with functional imbalances in nuclei such as the suprachiasmatic nucleus of the hypothalamus (biological rhythm

center), the raphe nuclei (5-hydroxytryptamine neurons), and the locus coeruleus (norepinephrine neurons) being the core links [6]. CBT-I can improve the regulatory function of these nuclei and restore normal sleep structure by adjusting sleep behavior and correcting cognitive biases [7]. This study focused on the predictive role of baseline PSG parameters on CBT-I efficacy, providing objective evidence for precision clinical treatment.

This study found that patients with lower baseline sleep efficiency showed more significant improvement after CBT-I treatment, which is consistent with the mechanism of sleep restriction therapy. Patients with low sleep efficiency often have the problem of "spending too much time in bed but actually sleeping for a short duration." Sleep restriction can quickly improve sleep continuity by reducing time spent in bed and increasing sleep pressure (increasing the accumulation of sleep-promoting substances such as adenosine) [8]. On the other hand, patients with high sleep efficiency may have already formed a relatively stable sleep pattern and are less sensitive to intervention, suggesting that CBT-I can be preferentially adopted for patients with low sleep efficiency in clinical practice to achieve more significant initial efficacy.

Patients with longer baseline wake time after sleep onset responded better to CBT-I, which may be related to the synergistic effects of stimulus control therapy and relaxation training. Frequent awakenings after falling asleep are often associated with excessive attention to the sleep environment (such as "worrying about not being able to sleep") and increased autonomic nervous system excitability. Stimulus control therapy can reduce wake time while in bed, and relaxation training can lower sympathetic nervous system activity (such as reducing heart rate and blood pressure fluctuations), thereby improving sleep maintenance [9]. Additionally, this type of patient often has a lower proportion of N3 sleep, and CBT-I can increase deep sleep (N3 stage) to improve sleep stability, further validating the predictive value of wake time after sleep onset.

Regarding the predictive role of the proportion of N3 sleep, this study showed a positive correlation with treatment efficacy, but it did not enter the final regression model (P=0.10), which may be related to the small sample size. N3 sleep is regulated by the ventrolateral preoptic area of the hypothalamus and is closely related to physical recovery and sleep stability [10]. Patients who retain a good proportion of N3 sleep at baseline may have less impairment in their sleep regulatory mechanisms and may be more likely to benefit from CBT-I [11]. This conclusion needs to be further validated with a larger sample size.

The strengths of this study lie in: (1) strictly following PSG monitoring protocols to ensure data objectivity; (2) the time frame of the cases aligns with the hospital's opening time (March 2024 to June 2025), ensuring a reliable and authentic sample source; (3) focusing on the middle-aged and elderly population aged 35–75, which is more aligned with the characteristics of the clinical population prone to chronic insomnia. The limitations include: (1) being a single-center study with a small sample size (40 cases), which may introduce selection bias; (2) the absence of long-term follow-up data, making it impossible to evaluate the impact of predictive factors on the durability of treatment effect; (3) not combining neuroimaging techniques (such as fMRI) to further explore brain mechanisms. Future multi-center studies can be conducted, combining dynamic PSG monitoring and brain functional imaging to further refine the prediction model.

Sleep efficiency and wake time after sleep onset among baseline PSG parameters can serve as independent predictors of the efficacy of CBT-I in treating chronic insomnia. Patients with low baseline sleep efficiency and long wake time after sleep onset show a more significant treatment response. Clinically, these objective indicators can be used to screen CBT-I sensitive populations, optimize treatment strategies, and improve the precision of insomnia management.

5. Conclusion

The baseline PSG parameters, including sleep efficiency and wake after sleep onset (WASO), can effectively predict the efficacy of CBT-I in treating chronic insomnia, providing an objective basis for clinically identifying treatment-sensitive populations.

Disclosure statement

The author declares no conflict of interest.

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