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Psychophysiological Evaluations of Clinical Efficacy in Outpatients Morita Therapy for Psychophysiological Insomnia

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Introduction

Psychophysiological insomnia (PPI) is a clinical subtype of chronic insomnia disorder in International Classification of Sleep Disorders (ICSD-3) [1]. PPI is the most common insomnia in clinical setting.

PPI is an objectively verifiable insomnia that develops as a consequence of two mutually factors: 1) somatization tension due to the fear for insomnia and 2) learned sleep-preventing associations.

On the other hand, morbid fear of insomnia in Morita therapy develops as a consequence of the following factors: 1) psychic interaction based on hypochondriacal temperament, 2) autosuggestion which caused by psychic interaction and 3) anxiety regarding adaptation with subjective fabrication of symptom.

Therefore, the disease concept of PPI in ICSD-3 and neurotic insomnia in Morita therapy is seemed to be very similar2).

The reconstruction of sleep-preventing association through the education of sleep hygiene is the main therapeutic strategy for PPI. Sleep hygiene instructions consist of the following factors: 1) homeostatic drive for sleep, 2) circadian factors, 3) drug effects and 4) arousal in sleep setting.

The point of Morita therapy for morbid fear for insomnia is to accept insomnia as it is and lead a constructive life with the guidance of Morita therapy.

The authors supposed that the guidance of Morita therapy adding some psychopharmacological and bed room environmental findings is corresponded with the current concept of sleep hygiene [2].

The aim of this study [3] was to investigate the clinical efficacy of Morita therapy for PPI.

Subjects and Methods

The clinical efficacy of outpatients Morita therapy [4] for PPI was evaluated in terms of psychophysiological factors. The subjects, 13 outpatients (mean age: 47.6 ± 17.7, male/female: 6/7), were given the diagnosis of PPI at ICSD-3 who wished to receive Morita therapy.

The participants were excluded in the following conditions: they 1) met the DSM-IV-TR [5] criteria for any psychiatric disorder and/or substance abuse, 2) required psychotropic medication for psychiatric symptoms, or 3) had symptoms suggestive of sleep apnea syndrome, narcolepsy, or restless legs syndrome as judged from clinical interviews.

For each one week of pre-treatment (PRE) and post-treatment (POST; 2.0 ± 1.1 months), the examinations were performed consecutively, using by objective (actigraphy) and subjective (sleep logs) measurements.

The participants continued to take any medication that had already been prescribed before their enrollment in the trial.Data are presented as means ± SD, and were analyzed by the Wilcoxon rank sum sign test.

All analyses were performed using Stat View-J5.0 for Windows (SAS Institute Inc.). A P value of <0.05 was considered statistically significant.The study protocol and therapy regimen were approved by the Jikei University School of Medicine Ethics Committee (No. 11-21(2721)).

Written informed consent to participate in the study was obtained from all the participants after they were given an explanation of the study and its potential risks.

Results

Subjectively, total sleep time increased and sleep latency shortened significantly at POST compared with PRE.

Objectively, numbers of awakening decreased, sleep efficiency increased and moving time in sleep decreased significantly at POST compared with PRE.

The participants subjectively assessed that the sleep latency was longer and total sleep time shorter than the objective values during the PRE period (Table 1).

N=13	Pre-treatment	Post-treatment	P value
Sleep logs			
SONT (h)	24.1 (0.9)	24.2 (1.1)	n.s
SOFT (h)	7.6 (5.0)	7.1 (1.7)	n.s

TST (hours)	5.1 (1.9)	6.6 (1.6)	P < 0.05
SOL (min)	73.7 (42.0)	44.8 (31.5)	P < 0.05
Actigraphy			
SONT (h)	23.3 (1.0)	23.6 (0.8)	n.s
SOFT (h)	7.3 (0.8)	7.7 (1.0)	n.s
TST (hours)	7.4 (1.2)	7.9 (1.4)	n.s
SOL (min)	34.4 (21.2)	37.2 (31.8)	n.s
NOA (times)	5.8 (6.4)	3.1 (2.6)	P < 0.05
SE (%)	89.9 (8.6)	93.4 (7.1)	P < 0.05
MT (counts/epoch)	12.5 (6.5)	8.7 (5.6)	P < 0.01

Table 1: Sleep logs and actigraphy pre and posttreatment Morita therapy. All data are mean (SD). P: Wilcoxon rank sum sign test. SONT: sleep onset time, SOFT: sleep offset time, SOL: Sleep Onset Latency, TST: Total Sleep Time, SE: Sleep Efficiency, NOA: number of awakening episodes lasting more than 5 min, MT: Moving Time During Sleeping.

In the same way, the subjective sleep onset time was later and the subjective sleep offset time was earlier compared with the objective evaluations. Thus, the dissociation between subjective and objective estimation of sleep was confirmed. In the POST period, the differences between the sleep logs and actigraphy for sleep latency, total sleep time and sleep offset time were significantly decreased compared with the PRE period. Dissociations between subjective and objective evaluations about awakening time, total sleep time and sleep latency at PRE improved significantly at POST (Table 2).

N=13	Pretreatment	Posttreatment	P value
SONT (hours)	1.0 (0.8)	0.7 (0.9)	n.s
SOFT (hours)	2.7 (4.6)	0.8 (1.0)	P < 0.01
TST (hours)	2.5 (1.7)	1.5 (1.6)	P < 0.05
SOL (min)	42.2 (37.7)	14.4 (13.2)	P < 0.01

Table 2: Dissociation between subjective (sleep logs) and objective (actigraphy) estimation. All data are mean (SD) of the difference between subjective and objective measurement ([sleep logs minus actigraphy]) in the same night. P: Wilcoxon rank sum test. SONT: sleep-onset time, SOFT: sleep-offset time, TST: total sleep time, SOL: sleep onset latency.

Discussion

From the above-mentioned results, it was suggested that the improvement of dissociations between subjective and objective evaluations at POST showed psychophysiologically the reconstruction of sleep preventing association and getting out of the entrapment about insomnia. This finding suggested that outpatients Morita therapy for PPI was effective that subjects in this study accepted insomnia as it was and ware led a constructive life.

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