Effects of Two-session Group Cognitive Behavioral Therapy for Psychophysiological Insomnia: a preliminary study

ABSTRACT AND KEYWORDS

Abstract

The authors evaluated the effects of brief group cognitive behavioral therapy for insomnia (G-CBT-I) in outpatients with psychophysiological insomnia (PPI). This brief G-CBT-I was designed to yield results in a shorter period of time, because its strategy was intended to lower the dropout rate and enhance the cost performance. And also, it was intended to be easy to make a use of CBT-I for both therapists and patients. This process consists of four components and only two sessions weekly, and a total therapy time is approximately three hours.

Thirty-three participants (including 17 women) with PPI received G-CBT-I therapy. The short-term outcome (four weeks after G-CBT-I) was measured using sleep logs, actigraphy, the Japanese version of the Pittsburgh Sleep Quality Index (PSQI-J), and the Japanese version of the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS-J). The long-term outcome was evaluated by checking medical records at six months after G-CBT-I.

At four weeks after G-CBT-I, subjective sleep onset latency decreased by 32.1%, and objective sleep efficiency increased to approximately 90%. The dissociation between subjective and objective evaluations of sleep decreased. The total score of the PSQI-J and the scores on the DBAS-J (“consequences of insomnia”, “control and predictability of sleep”, and “sleep-promoting practice”) were decreased.

At the long-term follow-up, the amount of hypnotics needed by each participant decreased by 0.6mg (1 being equivalent to 1 mg of flunitrazepam) (33% reduction). These findings suggested that patients with PPI could derive significant benefit from brief G-CBT-I therapy.

Abstract: 246 words

Keywords: "Psychotherapy, Brief", "Cognitive Therapy", "Behavior Therapy", "Sleep Initiation and Maintenance Disorders", "Hypnotics and Sedatives".

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TEXT

Introduction

In the general Japanese population, about 20% of adults reported experiencing symptoms of either transient or chronic insomnia.[1] Insomnia is a prevalent form of sleep difficulty that impairs daytime functioning and reduces the quality of life.[2,3] Currently, pharmacotherapy remains the most frequently selected intervention presented to insomnia patients.[4,5] However, the long-term use of sedative hypnotics can have numerous adverse effects.

Primary insomnia, as defined in the Diagnostic and Statistical Manual of Mental Disorders, 4th ed. text version (DSM-IV-TR),[6] is the most common type of chronic insomnia and is similar to psychophysiological insomnia (PPI) as defined in the International Classification of Sleep Disorders, 2nd ed. (ICSD-2).[7] A well-known non-pharmacological treatment called cognitive behavioral therapy for insomnia (CBT-I) has demonstrated effectiveness for primary insomnia in multiple studies. [8-11] CBT-I may have fewer adverse effects and greater durability of curative effects, when compared to pharmacotherapy.[12] Furthermore, from a long-term perspective, it reduces physical risks to the patient and could be beneficial to the medical economy.[4,13]

CBT-I generally includes various components (e.g., cognitive therapy, relaxation therapy, and sleep restriction therapy), and the majority of the treatment spans a period between four and eight weeks (typically having four to eight sessions).[9,10] Therefore, CBT-I requires a lot of time and manpower, resulting in increased costs. In term of expense and the rapidity of its effect, research has shown that CBT-I is inferior to pharmacotherapy. In addition, CBT-I is largely dependent on the motivation of each individual

patient.[4] Therefore, it may be difficult to secure treatment agreements with insomnia patients who expect improvement from day one, given that CBT-I generally takes longer to show effects. And some studies have reported dropout rates for CBT-I, ranging from 13.7% to 34.0%.[14,15]

Therefore, various types of CBT-I have been investigated. Espie et al. suggested that CBT-I performed by primary care clinic nurses with minimal supervision and without physician involvement affected insomnia patients.[16] Mimeault et al. demonstrated the effectiveness of CBT-I when utilizing only self-help teaching materials and brief phone interventions.[17] These findings suggested that CBT-I experts did not necessarily have to be involved, and the location for CBT did not have to be the hospital or a meeting room.

Based on the concepts mentioned above, the authors designed a brief group CBT-I (G-CBT-I) strategy that could be performed easily and conveniently for both therapists and patients. The strategy was also intended to lower the dropout rate and enhance the cost performance. Therefore, it was essential for the treatment program to efficiently be performed in a short time. This G-CBT-I consisted of four components and only two sessions. The entire treatment period spanned two weeks, with a total therapy time of approximately three hours. And offering the therapy to a group, rather than an individual, leads to a reduction in time and cost.

The four components included stimulus control therapy, sleep restriction therapy, cognitive therapy, and sleep hygiene education. The authors did not adapt the relaxation therapy that is frequently used as a component of CBT-I. One reason was to shorten the treatment period. Another was that relaxation therapy might be less effective than stimulus control and sleep restriction therapies, especially with in patients of advanced age.[5]

The necessary session number of times was twice to accomplish the four components. Although Edinger et al. suggested that the optimal number of CBT was four sessions biweekly[18], the authors gave priority to shortening a

treatment period to maintain the motivation of patients and to decrease a dropout by setting this brief CBT two sessions weekly. And the authors supplemented the treatment with the individual booster sessions performing once or twice after the second session.

The aim of the present study was to evaluate the effects of the brief G-CBT-I.

Methods

Study Participants

The eligible subjects were male and female 20 years of age or older, were consecutive PPI patients who had been diagnosed by ICSD-2,[7] and who attended Jikei University Hospital as outpatients between 2009 and 2013, and who already had been using hypnotics for over a month when recruiting, and wished to receive G-CBT-I.

The participants were excluded in the following conditions: they (1) met the DSM-IV-TR[6] criteria for an axis I diagnosis of 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.

The participants continued to take any medication that had already been prescribed before their enrollment in the trial. This was done to avoid any impact of the medication withdrawal during the treatment.

Measurements

During the pre- and post-treatment periods, the authors conducted measurements including sleep logs, actigraphy, the Pittsburgh Sleep Quality Index (PSQI)[19], the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS)[20,21], and the Beck Depression Inventory (BDI).[22]

At six months after the G-CBT-I treatments, each participant's medical record was reviewed as part of the long-term follow-up. The authors investigated the daily dosage of hypnotics.

Sleep Logs

During the pre- and post-treatment periods, the participants were asked to keep sleep logs, which they completed just after getting up in the morning for 7 days. The authors averaged the participants' data for bedtime, rising time, sleep-onset time (SONT), sleep-offset time (SOFT), sleep onset latency time (SOL), total sleep time (TST), and total time in bed (TIB).

Actigraphy

During the pre- and post-treatment periods, participants were required to wear an actigraph (mini-motion logger actigraph; Ambulatory Monitoring, Inc) on their non-dominant wrist at all times for 7 days. Based on the participant's rest/activity data, their sleep was estimated using the algorithm devised by Cole et al.[23]. From this result, the authors obtained the averaged 7-day data for objective SONT, SOFT, SOL, and the number of awaking episodes lasting more than 5 min (NOA), awaking time after sleep onset (WASO), TST, sleep efficiency (SE), and moving time during sleep (MT). SE was also calculated as the percentage of the objective TST for each participant's actigraphy chart per TIB, which was recorded objectively on the sleep log by the family member, as indicated above.

Questionnaire

The authors used questionnaires as follows: the Japanese version of the PSQI (PSQI-J) to assess sleep quality and quantity, the Japanese version of the DBAS (DBAS-J) to grasp the participants' faulty understandings about sleep, and the Japanese version of the BDI (BDI-J) as a screening tool for

depression.
The reliability and validity of these Japanese versions has been previously confirmed. The high level of internal consistency of these was indicated, each Cronbach's α coefficient was 0.74 in the PSQI-J, 0.89 in the DBAS-J, and 0.87 in the BDI-J. [24-26]

Treatment

The four therapists (all men) conducted G-CBT-I, and all of them work as clinical psychiatrists and certified physicians for a Japanese society involved in sleep research. The first author conducted the CBT-I, and the other authors supervised its contents. The components of the G-CBT-I consisted of stimulus control therapy,[9,10] sleep restriction therapy,[9,10] cognitive therapy,[9,10] and sleep hygiene education.[9,10] The G-CBT-I protocol was as follows.

- During the pre-treatment period (defined as the seven days prior to the first G-CBT-I session), the participants were required to wear an actigraph and keep sleep logs for 7 consecutive days. The participants also completed the PSQI-J, DBAS-J, and BDI-J.
- At the first session, the participants underwent sleep hygiene education. The actigraphy and the sleep logs were collected after the session.
- The second session was conducted 1 week after the first. After reviewing the sleep hygiene education, the participants underwent cognitive therapy, stimulus control therapy, and sleep restriction therapy.
- As part of the two group sessions (60–90 minutes, 3–5 patients per group), participants listened to a lecture by a therapist and undertook a CBT-I group discussion.
- After the second session, individual booster sessions (once or twice, for durations of 10 minutes) were planned during the four-week follow-up period. The therapist confirmed whether the participants received

stimulus control therapy and sleep restriction therapy definitely, and answered questions about sleep hygiene education from the participants.

- In the post-treatment period (defined as the seven days after 4 weeks of the second session), the participants were required to do the same thing that they did in the pre-treatment period.

Stimulus Control Therapy

Stimulus control attempts to break the association between sleep environment and wakefulness. This is achieved by teaching the participants how to not engage in activities that might disturb their sleep. The instructions the therapists gave were as follows: (1) go to bed only when becoming sleepy, (2) do not use the bedroom for anything except sleep or sex, and (3) get out of bed and go to another room whenever unable to fall asleep over a period of 30 minutes, returning to bed only when sleepy again.

Sleep Restriction Therapy

This treatment seeks to increase the homeostatic sleep drive through partial deprivation thereby improving the ability to sleep. A bedtime and rising time schedule was prescribed in an attempt to improve the sleep quality and decrease the time spent awake during the night. Time in bed was reduced based on the total sleep time, as recorded in the sleep logs. In addition, the time of rising was always fixed to a set time. The time that the participant went to bed was adjusted on the basis of sleep efficiency. Although the authors were not absolutely strict when administering sleep restriction therapy, the therapists emphasized the importance of spending time in bed only when sleepy.

Cognitive Therapy

The therapists calculated the dissociation between the participants'

subjective sleep evaluation, taken from their sleep logs, and the participants' objective sleep data, measured by an actigraph during the pre-treatment period. The therapists showed the participants the amount of dissociation between the two parameters, as an indicator of sleep state misperceptions. Subsequently, cognitive therapy was conducted to identify the incorrect perceptions about sleep specific to each participant. By doing so, the therapists could correct any dysfunctions that were present.

Sleep Hygiene Education

Sleep hygiene education included instructions about health practices as well as environmental factors that may be beneficial to maintain sufficient sleep. Details were also given regarding the homeostatic drive for sleep, circadian factors, and the effects of drugs and other habits prior to sleep.

Statistical Analysis

Data were analyzed using the Stat View-J 5.0 software for Windows (SAS Institute Inc.). The authors examined the changes in the parameters, such as the sleep logs and actigraphy measurements, using the paired t-test. The Wilcoxon signed-rank test was used to examine the changes in the parameters, such as PSQI-J, DBAS-J, and the dosage of hypnotics. Statistical significance was determined at $p < 0.05$.

Approval of the Study

The study protocol and therapy regimen were approved by the Jikei University School of Medicine Ethics Committee. 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. All of the procedures were carried out in accordance with the Good Clinical Practice guidelines, the Helsinki Declaration, and related laws.

Results

The Number and Characteristics of the Participants at Baseline

A total of 39 participants gave their written informed consent to take part in the study. During the treatment, however, six participants withdrew from the trial. These 6 participants were excluded because of lack of interest (n = 3) or physical problems (n = 3). These physical problems were the operation of the cataract, the admission by the acute pancreatitis, and the difficulty in participation for the aggravation of the lumbago. Data for these participants were excluded from the statistical analysis.

Ultimately, 33 participants were included. Of the 33 participants, 17 (51.5%) were women and 16 (48.5%) were men, with an average age of 58.4 ± 13.8 years (range: 30-81 years). The average education level was 14.7 ± 1.9 years (range: 9-18 years). The average duration of insomnia, which was the subjectively reported period from the initial appearance of insomnia to the time of the G-CBT-I, was 6.8 ± 6.2 years (range: 0.08-21 years) and 33 (100%) had previously used hypnotics. The average nightly dosage of hypnotics before the trial was 1.8 ± 1.0 mg (1 being equivalent to 1 mg of flunitrazepam) (range: 0.33-4.0 mg). This was calculated using the Dose Equivalence of Psychotropic Drugs: 2006-Version.[27] The average score on the BDI-J was 9.5 ± 5.8. The values represented the mean ± standard deviation. Table 1 presents the demographic data and clinical variables.

Sleep Data

As shown in Table 2, evaluation of the sleep logs indicated that there was a significant reduction of SOL ($p < 0.001$) and significant extension of TST ($p < 0.01$) in the post-treatment period compared with those in the pre-treatment period. In the post-treatment period, bedtime ($p < 0.005$) was at a later time and the rising time ($p < 0.001$) was significantly earlier. Objective results also showed that the WASO ($p < 0.05$) measured on actigraphy became

shorter in the post-treatment period. The SE ($p < 0.001$) increased to approximately 90% (Table 2).

The participants subjectively assessed that the SOL was longer and TST shorter than the objective values during the pre-treatment period. In the same way, the subjective SONT was later and the subjective SOFT was earlier compared with the objective evaluations. Thus, the dissociation between subjective and objective estimation of sleep was confirmed. In the post-treatment period, the differences between the sleep logs and actigraphy for SOL ($p < 0.001$) and SONT ($p < 0.001$) were significantly decreased, whereas those for TST ($p < 0.001$) and SOFT ($p < 0.001$) were significantly increased (Table 3).

Questionnaire Measures and Ratings

On the PSQI-J, not only the total score ($p < 0.001$), but also the scores for "overall sleep quality" ($p < 0.001$) and "sleep latency" ($p < 0.005$) were decreased significantly by the post-treatment period (Table 4).

On the DBAS-J, the scores for "consequences of insomnia" ($p < 0.005$), "control and predictability of sleep" ($p < 0.001$), and "sleep-promoting practice" ($p < 0.05$) were decreased significantly at the post-treatment period (Table 4).

On the BDI-J, the total score was varied from 9.5 ± 5.8 to 9.0 ± 6.2 during the post-treatment period ($p = 0.471$).

The Daily Dosage of Hypnotics at the Long-term Follow-up

Table 1 presents the data for demographic and clinical variables at the long-term follow-up (N=31). The details of two of 33 participants were unclear, because they stopped the ambulatory continuation during the six months after the completion of G-CBT-I.

The daily dosage of hypnotics significantly decreased ($p < 0.001$). Compared to pre-treatment levels, it had fallen from 1.8 ± 1.2 mg to 1.2 ± 1.4 mg at six months after the G-CBT-I.

Discussion

This study evaluated the clinical efficacy of a brief G-CBT-I that was designed to be easily administered. This two-session CBT-I took less time than the generally-recognized CBT-I.
Stimulus control therapy, sleep restriction therapy, and sleep hygiene education were given in a lecture format, and participants could refer to their notes and the provided documents if they wished to revisit the content. The method that the authors adopted for the cognitive therapy is similar to the feedback technique performed by Tang and Harvey, rather than the standard format of cognitive therapy[28]. This method could only be incorporated by keeping sleep logs for one week, and by using actigraphy for one week.

In the post-treatment period, the subjective evaluations improved significantly as indicated by the sleep logs, especially SOL and TST; however, objective SOL recorded by actigraphy did not improve significantly, and the objective TST had shortened. The dissociation between the subjective and objective estimation of SOL and TST was also reduced. Thus, the overestimation of SOL and the underestimation of TST were corrected after G-CBT-I. This brief G-CBT-I strategy could be effective for the alteration of sleep state misperceptions that may play a pathological role in the mechanism of insomnia.

Cognitive therapy, using the feedback technique, may strongly influence this improvement. Observing their sleep status using actigraphy and visually confirming their distorted perceptions about sleep may have a big impact for the participants. Tang and Harvey have suggested that, compared with insomnia patients who were simply told about the discrepancy between their

subjective and objective sleep estimates, those who were shown the estimates in visible form through this technique thought and felt more positively about their sleep.[28]

In terms of the questionnaire measures and ratings, the sleep quality and sleep latency were improved significantly at one month after G-CBT-I on the PSQI-J. On the other hand, there was no large or significant changes in the index of total sleep time. This showed that the sleep restriction therapy was effective. In actuality, the TIB was more than thirty minutes shorter after G-CBT-I; this suggested that sleep restriction therapy was appropriately utilized. Consequently, the subjective sleep efficiency was also improved.

On the DBAS-J, the scores for all five indexes were decreased after the G-CBT-I, and anxiety about the consequences of insomnia and anxiety about the control and predictability of sleep had greatly improved in particular. Acquiring accurate knowledge about sleep through an educational component may contribute to this improvement. However, the recent studies have determined that therapy that only incorporated sleep hygiene education was inferior to sleep restriction therapy alone or a multi-component therapy.[29-32] Hence, the other components may have complemented the sleep hygiene education for the improvement of DBAS-J in this study as well.

As described above, this brief G-CBT-I did have some effect. However, for the subjective evaluations as indicated by the sleep logs in the post-treatment period, the TST was less than 6 hours (352 min) and the SE was approximately 82%. These results were lower than normative levels (TST > 6 hours, SE > 85%). The total score for PSQI-J at one month after G-CBT-I was 10.2, which is higher than the 5.5 considered to be the cut-off level.

At the long-term follow-up, the daily dosage of hypnotics was decreased by 0.6 mg (33% reduction). This finding suggested that this brief G-CBT-I could be an effective intervention for PPI patients who would like to discontinue or

decrease their use of benzodiazepines. And reducing hypnotic drug use could lead to the reduction of medical economy. On the other hand, it is important to note that there can be many relapses while using hypnotics after discontinuation in the treatment outcome by using only CBT-I.[33]

The present study has several limitations. First, there was no control group. In the absence of a control group, it will be impossible to rule out other causes for improvements over time. In addition, there was no randomization of participants in this study. All participants wanted to receive the brief G-CBT-I and therefore might have been very motivated. Second, the authors did not conduct measurements including sleep logs, actigraphy, PSQI-J, DBAS-J, and BDI-J at the long-time follow-up. It will be necessary to confirm changes in cognition and behavior because insomnia patients who benefit from short-term evaluation might remain vulnerable to recurrent insomnia episodes over the long-term. Third, the authors did not quantify about the influence that individual booster sessions gave to curative effect. It might be possible that the participants took benefit from individual booster sessions, not from two group sessions that they all received equally. Fourth, the participants did not undergo nocturnal polysomnography. The authors judged the patient data from clinical interviews, so occult sleep pathology like sleep apnea or periodic limb movement might not have been excluded.

In consideration of these limitations, further research will be needed to determine how long the effect of brief G-CBT-I can be sustained.
It will be significant to investigate the sleep data, the perception about sleep, and the daily dosage of hypnotics at the long-term follow-up (e.g., six months or one year after the treatment).
Further investigation will also be needed to determine the most suitable choice of components to be included in the brief CBT-I protocol.
The authors did not adopt the relaxation therapy as a component of the brief G-CBT-I in this study. However, it was necessary for the effect of brief CBT-I comprised of the combination of various techniques (e.g., stimulus control, sleep restriction, muscle relaxation, and paradoxical intention) to be

weighed.

Edinger et al. suggested that not only the number of treatment sessions but also the length of the inter-session interval were factors worthy of investigation.[18] They also suggested that biweekly individual CBT sessions may be a suitable interval. The present study scheduled the brief G-CBT-I over two consecutive weeks. Hence, it will be necessary to study the influence that different lengths of inter-session intervals (e.g., weekly versus biweekly) have on the effect.

DISCLOSURE STATEMENT

This was not an industry-supported study. The authors have indicated no financial conflicts of interests.

Text: 3435 words

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Table 1
Demographic and clinical characteristics before G-CBT-I and at long-term follow-up

	Total	
Characteristics	Pretreatment (N=33)	Long-trem follow-up (N=31)
Age, yr	58.4 (13.8)	58.3 (13.9)
Sex, male/female	16 / 17	15/16
Education, yr	14.7 (1.9)	14.7 (1.9)
Insomnia duration, yr	6.8 (6.2)	7.1 (6.2)
Benzodiazepine intake		
Average nightly quantity, mg-flunitrazepam equivalent	1.8 (1.0)	1.2 (1.4) *
BDI-J total score	9.5 (5.8)	-

All data are mean (SD). BDI-J: the Japanese version of the Beck Depression Inventory.
* : a significant decrease of Benzodiazepine intake at 6 months after CBT by Wilcoxon signed-rank test. ($p < 0.0001$

Table 2
Sleep logs and actigraphy before and after G-CBT-I

N=33	Sleep logs				N=33	Actigraphy			
	Pretreatment	Posttreatment	Change [95% CI]	p		Pretreatment	Posttreatment	Change [95% CI]	p
SONT (hr)	24.4 (1.4)	24.3 (1.1)	0.10 [−0.07 ; 0.27]	0.2449	SONT (hr)	23.9 (1.3)	24.1 (1.1)	−0.86 [−1.73 ; 0.01]	0.0519
SOFT (hr)	6.1 (1.5)	6.2 (1.2)	−0.16 [−0.31 ; −0.02]	0.0296	SOFT (hr)	6.9 (1.3)	6.7 (1.3)	0.11 [0.003 ; 0.22]	0.0434
SOL (min)	50.2 (34.0)	34.1 (17.1)	16.37 [8.25 ; 24.48]	<0.0001	SOL (min)	18.5 (11.2)	17.8 (13.0)	0.60 [−3.65 ; 4.84]	0.7823
TST (min)	335.5 (60.0)	352.1 (40.6)	−16.80 [−28.92 ; −4.69]	0.0068	TST (min)	401.6 (57.1)	383.1 (47.7)	16.58 [7.35 ; 25.81]	0.0005
SE (%)	72.0 (9.7)	81.8 (7.2)	−9.08 [−11.65 ; −6.50]	<0.0001	SE (%)	87.0 (7.8)	89.8 (6.7)	−2.76 [−4.16 ; −1.35]	0.0001
Bedtime (hh;mm)(hr)	23:36 (01:18)	23:48 (01:06)	−0.18 [−0.30 ; −0.07]	0.0014	NOA (times)	1.6 (1.0)	1.3 (0.8)	0.25 [−0.01 ; 0.51]	0.0601
Risingtime (hh;mm)(hr)	07:18 (01:06)	07.00 (01:12)	0.37 [0.26 ; 0.48]	<0.0001	WASO (min)	15.6 (12.3)	12.2 (9.6)	2.87 [0.16 ; 5.58]	0.0381
TIB (min)	464.8 (56.7)	430.5 (43.9)	34.30 [25.25 ; 43.36]	<0.0001	MT (counts/min)	8.2 (3.9)	7.8 (3.5)	0.40 [−0.33 ; 1.13]	0.2789

All data are mean (SD). G-CBT-I: group cognitive behavioral therapy for insomnia. SONT: sleep onset time, SOFT: sleep offset time,
SOL: sleep onset latency, TST: total sleep time, SE: sleep efficiency, TIB: total time in bed, NOA: number of awakening episodes lasting more than
WASO: awakening time after sleep onset, MT: moving time during sleeping, CI: confidence interval. p: paired-t test.
Bedtime, Risingtime: The values at pre- and post-treatment are clock indication(hh:mm), and the unit of amount of change is hour (hr).

Table 3
Dissociation between subjective (sleep logs) and objective (actigraphy) estimation

N=33	Pretreatment	Posttreatment	Change [95% CI]	p
SONT, min	31.4 (30.7)	15.9 (21.4)	15.57 [7.31 ; 23.83]	0.0003
SOFT, min	−45.3 (41.9)	−27.8 (25.4)	−15.95 [−24.32 ; −7.59]	0.0002
SOL, min	34.0 (30.5)	16.2 (19.3)	18.40 [10.27 ; 26.52]	<0.0001
TST, min	−68.4 (47.8)	−33.9 (34.6)	−33.26 [−44.36 ; −22.15]	<0.0001

All data are mean (SD) of the difference between subjective and objective measurement (sleep logs minus actigraphy) in the same night. CI: confidence interval. p: paired-t test.
SONT: sleep onset time, SOFT: sleep offset time, SOL: sleep onset latency, TST: total sleep

Table 4
PSQI-J and DBAS-J before and after G-CBT-I

N=33	Pretreatment	Posttreatment	p
PSQI-J			
Overall sleep quality	2.1 (0.6)	1.5 (0.6)	0.0008
Sleep latency	2.0 (0.8)	1.5 (1.0)	0.0041
Duration of actual sleep time	1.8 (0.9)	1.7 (0.8)	0.4796
Sleep efficiency	1.5 (1.2)	1.1 (1.1)	0.1144
Sleep disturbance	1.1 (0.4)	0.9 (0.6)	0.0833
Medication necessary to sleep	2.8 (0.5)	2.7 (0.8)	0.3657
Day dysfunction due to sleepines	1.0 (0.8)	0.7 (0.6)	0.1009
Total score	12.3 (2.7)	10.2 (3.6)	0.0008
DBAS-J			
Consequences of insomnia	57.1 (21.6)	47.0 (23.0)	0.0049
Control and predictability of slee	47.8 (17.3)	38.1 (20.4)	0.0002
Sleep requirement expectations	37.6 (17.4)	31.5 (16.7)	0.1298
Causal attributions of insomnia	34.0 (24.9)	31.7 (21.4)	0.6334
Sleep-promoting practices	32.0 (14.2)	28.0 (15.5)	0.0444

All data are mean (SD). G-CBT-I: group cognitive behavioral therapy for insomnia.
PSQI-J: the Japanese version of the Pittsburgh Sleep Quality Index,
DBAS-J: the Japanese version of the Dysfunctional Beliefs and Attitudes about Sleep Scale
p: Wilcoxon signed-rank test.