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THE EFFICACY OF COGNITIVE-BEHAVIOURAL THERAPY (CBT) AS RELATED TO SLEEP QUALITY AND HYPERAROUSAL LEVEL IN THE TREATMENT OF PRIMARY INSOMNIA

Maria Gałuszko-Węgielnik, Katarzyna Jakuszkowiak-Wojten, Mariusz Stanisław Wiglusz, Wiesław Jerzy Cubała & Jerzy Landowski

Department of Psychiatry, Medical University of Gdańsk, Gdańsk, Poland

SUMMARY

Background: *Primary insomnia (PI) is a common sleep disorder affecting diurnal functioning. It may contribute to the development of several comorbidities such as major depression or arterial hypertension. It affects about 7% of the adult population. Pharmacotherapy remains the most common treatment for insomnia. However, many studies suggest CBT may be a supreme therapeutic approach resulting in a better long-term outcome.*

The aim of the study was to determine the efficacy of a CBT-protocol in the treatment of PI by means of sleep onset latency and the number of awakenings during night parameters along with sleep quality and the level of psychophysiological hyperarousal. The secondary outcomes were focused on CBT efficacy as determined by the predisposition to insomnia as related to higher vulnerability to stress (measured with FIRST)

Material and methods: *Twenty-six individuals from a tertiary reference sleep disorders outpatients' clinic (22 women; mean age 41.4; 4 men; mean age 42.5) with primary insomnia (DSM-IV-TR) were included in the study. The exclusion covered other primary sleep disorders, secondary insomnia (psychiatric illness, unstable somatic illness, shift work), substance abuse/dependence, high results in HADS-M scale (score above 11). The participants were scored with HADS-M, Ford Insomnia Response to Stress Test (FIRST) at the beginning of the study. The Athens Insomnia Scale (AIS), Hyperarousal Scale, Leeds Sleep Questionnaire (LSEQ) were applied at the beginning, at the end and three months after the end of the study. The participants were also examined by 7 days actigraphic records before and after treatment. During the course of the treatment patients completed a Sleep Diary (SD). The CBT program employed was based on the Perlis protocol. Standard individual sessions of 50 minutes were provided on a weekly basis for 8-10 weeks by a board certified CBT therapist. After 3 months a follow-up session was scheduled.*

Results: *The significant improvement as related to the CBT treatment was present in the measures of sleep onset latency (67.2 vs. 23.4 min.; $p<0.000$), numbers of awakenings during night (2 vs. 0.4; $p<0.000$) and sleep efficiency (77.3 vs. 91%; $p<0.000$) - data from SD, quality of falling asleep (3.2 vs. 6; $p<0.000$), quality of sleep (3.3 vs. 5.8; $p<0.000$) and quality of morning awakening (3.2 vs. 6; $p<0.000$) – data from LSEQ. The improvement reached the significance level in the measure of psychophysiological arousal (52.3 vs. 42.4; $p<0.000$) and AIS (15.7vs. 6.8; $p<0.000$). No significant differences were identified between actigraphic records (light/dark ratio) before and after CBT. FIRST scores allocating patients to high and low stress vulnerability groups were non-contributory to the observed treatment efficacy.*

Conclusion: *CBT is an effective treatment in primary insomnia. No relationship between CBT efficacy and predisposition to insomnia as determined by higher vulnerability to stress was identified.*

Key words: *Cognitive-Behavioural Therapy - primary insomnia - sleep disorders*

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INTRODUCTION

Insomnia is a common disorder affecting 10-20% of adult population (Costa e Silva et al. 1996). It is the third in the order of complaints that patients report to general practitioners (Lugaresi et al. 1983). Insomnia may result from the exposure to stressful events (e.g. journey, hospitalization, problems at work, exams) and usually lasts no longer than 3 weeks (transient and short-term insomnia). Sleep problems lasting longer than 3 weeks are called long-term insomnia (NIMH 1984) or chronic insomnia. Insomnia may be either the primary complaint (primary insomnia - PI) or the symptom of other physical or mental disorder (secondary insomnia). The prevalence of long-term insomnia is estimated at the level if 10% and primary insomnia about 3-7% (Anticoli-Israel & Roth 1991, Ohayon & Lemoine 2002). Primary insomnia is more common in women. The usual age of onset is 20-40 years (Martin & Anticoli-Israel 2002). The Diagnostic and Statistical Manual of Mental Disorder (DSM-IV-TR 2000) defines primary insomnia as a complaint lasting at least 1 month. Patients report problems with onset and maintenance of sleep or poor quality of sleep. Sleep problems affect diurnal activity and do not occur in the course of other physical or mental disorders. ICD-10 defines primary insomnia as nonorganic insomnia (ICD-10 1998).

The diagnosis of PI should be based on a systematic patient interview. Besides, polysomnography and/or actygraphy may be helpful in the differentiation between primary insomnia and other primary sleep disorders (eg. restless leg syndrome).

The etiology of primary insomnia is mulitifactoral. According to Spielman et al. PI is the effect of predisposing, precipitating and perpetuating factors

(Spielman et al. 1987). The predisposing factors are in the biopsychosocial spectrum (eg. hyperarousal/hyper-reactivity, congenital poor sleep generating system, cognitive tendency to rumination). The precipitating factors interact with ones predisposition for insomnia causing transient sleep problem or maintenance insomnia (eg. medical illnesses, shift work, interpersonal problems). The perpetuating factors include a wide spectrum of maladaptive strategies that patients develop in order to diminish insomnia (e.g. excessive time in bed, naps, failure to keep sleep hygiene rules, maladaptive strategies for thoughts control) (Spielman et al. 1987, Ebben & Spielman 2009, Schmidt et al. 2010).

Hyperarousal plays the central role in PI etiology. It includes cortical arousal, somatic arousal and cognitive arousal with typically negative, intrusive thoughts and pathological worrying associated with sleep problems and day activity. Hyperarousal is a vital part of insomnia models. Harvey proposed the model of insomnia based on cognitive arousal in the evening and during the day (Harvey 2002). Espie et al. proposed the attention-intention-effort pathway in the development of insomnia where sleep is an automatic process which is vulnerable and may be inhibited by any activity aimed to control its expression (eg. focused attention) (Espie et al. 2006).

Untreated insomnia worsens the quality of life patients and may predispose the individual for the development of comorbid medical conditions or worsening the existing ones (Agargun et al. 1997, Liu & Tanaka 2002, Wallander et al. 2007, Narkiewicz 2001).

Pharmacotherapy remains the most common treatment for insomnia. However, many studies suggest CBT may be a supreme therapeutic approach resulting in better long-term outcome (Morin et al. 2006, Wu et al. 2006).

The aim of the study was to determine the efficacy of a CBT-protocol in the treatment of PI by means of sleep onset latency and number of awakenings during night parameters along with sleep quality and the level of psychophysiological hyperarousal. The secondary outcomes were focused on CBT efficacy as determined by the predisposition to insomnia as related to higher vulnerability to stress (measured with FIRST)

SUBJECTS AND METHODS

Twenty six individuals with primary insomnia (DSM-IV-TR) were enrolled in the study (22 women; mean age 41.4; 4 men; mean age 42.5). The patients were recruited at the regional sleep disorders out-patients' clinic. The exclusion covered other primary sleep disorders, secondary insomnia, substance abuse/dependence, high results in HADS-M scale (above 11 points). Thirty patients were screened at baseline visit and 4 of them were excluded or dropped out from the study.

At the beginning of the study and after systematic psychiatric evaluation participants were scored with the Polish modification of the Hospital Anxiety and Depression Scale (HADS-M) (Zigmond & Snaith 1983, Majkowicz 2000), Ford Insomnia Response to Stress Test (FIRST) (Drake et al. 2004, Drake et al. 2006). The Athens Insomnia Scale (AIS) (Soldatos et al. 2000, Soldatos et al. 2003), Hyperarousal Scale (HS) (Regestein et al. 1996), Leeds Sleep Evaluation Questionnaire (LSEQ) (Parrott & Hindmarch 1980) were applied at the beginning, at the end and three months after the end of the study. The participants were also assessed with 7-days-long actimetry before and after treatment. Across the treatment patients were asked to complete a Sleep Diary (SD). The CBT intervention program was based on the Perlis protocol (Perlis et al. 2005) Standard individual sessions of 50 minutes were provided on a weekly basis for 8-10 weeks by a board certified CBT therapist under supervision. After 3 months a follow-up session was scheduled.

The statistical analysis was performed using ANOVA with repeated measures test, post hoc LSD Fisher's test and Pearson's r correlation with Statistica v.8.0 software.

The study protocol was approved by the local bioethics committee at the Medical University of Gdańsk.

RESULTS

The improvement reached significance level in the measure of psychophysiological arousal (52.3 vs. 42.4; $p<0.000$) and AIS average scores reduced about 8.9 points (before treatment-15.7; after–9.2; follow up-6.8) $p<0.000$ (Figure 1). The significant improvement as related to the CBT treatment was present in data from sleep diary records with improvement in sleep onset latency being reduced from 67.2 min. at the beginning of the intervention to 23.4 min. after treatment $p<0.000$ (Figure 2), numbers of awakenings during night with 2 vs. 0.4 respectively; $p<0.000$ (Figure 3), increased sleep efficiency from 77.3% to 91% after treatment, $p<0.000$ (Figure 4). LSEQ also indicated better sleep quality (3.3 vs. 5.8; $p<0.000$) (Figure 5), improved quality of falling asleep (3.2 vs.6; $p<0.000$), and the quality of morning awakening (3.2 vs. 6; $p<0.000$). The improvement reached the significance level in the measure of psychophysiological arousal – 52.3vs. 42.4; $p<0.000$ (Figure 6). No significant differences were identified between actigraphic records (light/dark ratio) before and after CBT (Figure 7).

FIRST scores allocating patients to high and low stress vulnerability groups were non-contributory to the observed treatment efficacy.

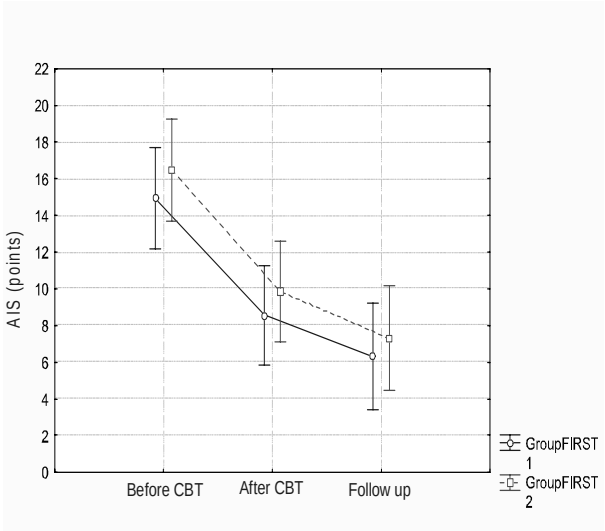

Figure 1. Athens' Insomnia Scale. (0.95 confidence interval)

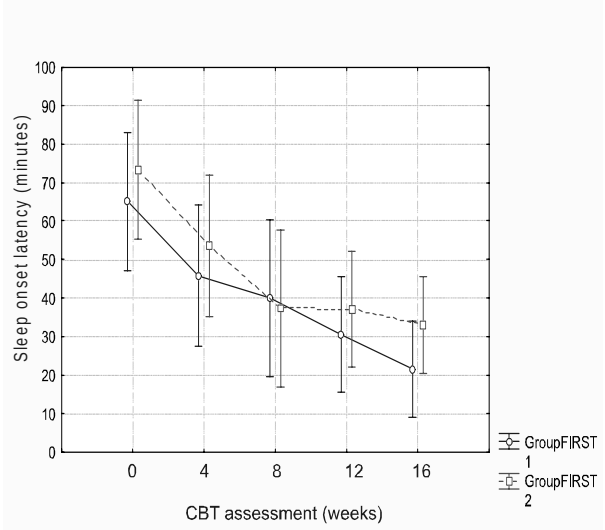

Figure 2. Sleep onset latency. (0.95 confidence interval)

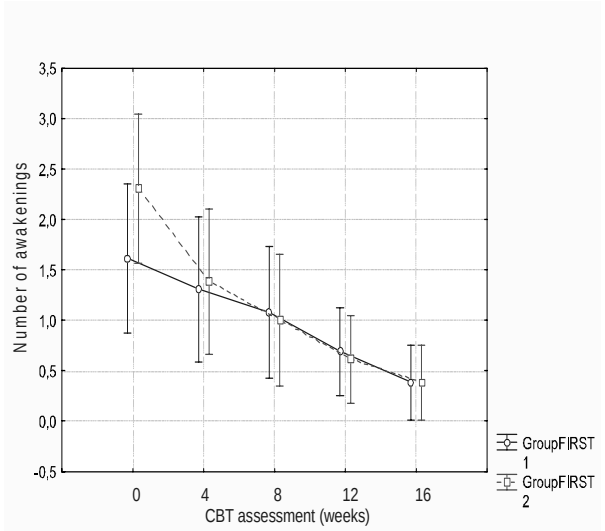

Figure 3. Number of awakenings. (0.95 confidence interval)

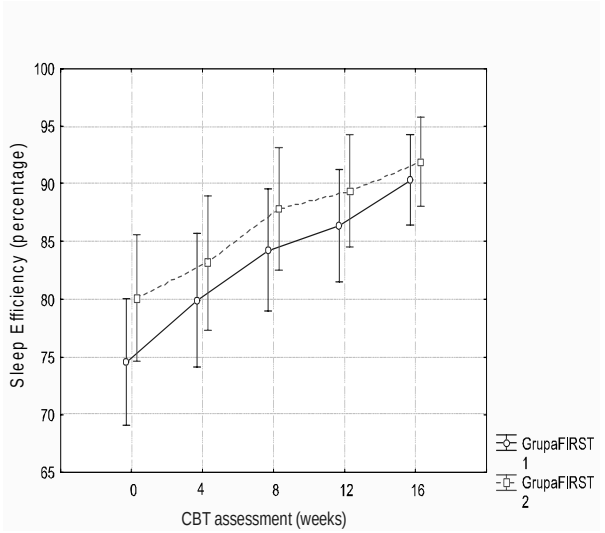

Figure 4. Sleep efficiency. (0.95 confidence interval)

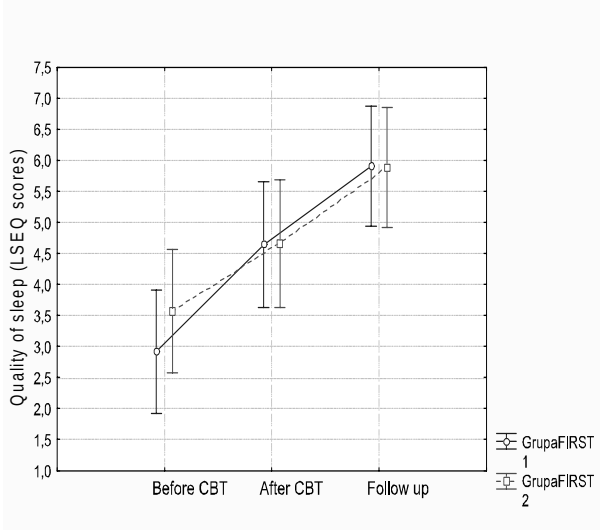

Figure 5. Quality of sleep. (0.95 confidence interval)

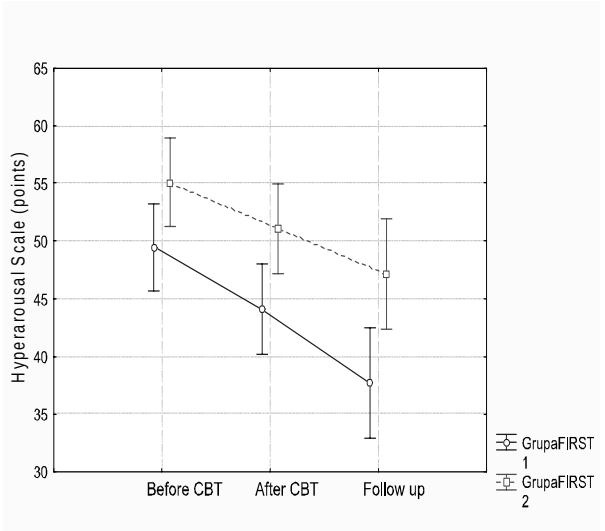

Figure 6. Hyperarousal Scale. (0.95 confidence interval)

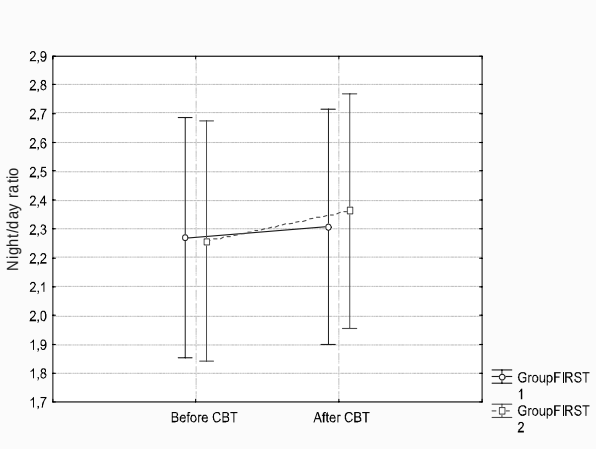

Figure 7. Actigraphic Night/Day ratio (0.95 confidence interval)

DISCUSSION

The study results support the efficacy of cognitive-behavioural therapy in the treatment of primary insomnia. This observation corresponds with other studies (Edinger et al. 2001, Edingeret al. 2005, Morin et al. 1999). Cognitive-behavioural therapy improves the spectrum of sleep parameters with shortened sleep latency, longer total sleep time, improved sleep efficacy and lessens the number of awakenings during night. Also the level of hyperarousal diminished in accordance with Harvey's model of PI (Harvey 2002).

The observed sleep latency shortening is corresponds with the one observed in other surveys indicating the efficacy of CBT treatment in shortening the time by 25-33min. (Morin et al. 1994, Murtagh&Greenwood 1995, Espie et al. 2001, Jacobs et al. 2004). The effect is attributed to the decrease in the level of hyperarousal associated with relaxation, cognitive reconstruction etc.

Higher quality of sleep observed in the increased sleep efficiency and the reduction in the number of awakenings during the night correspond with the results from Morin et al. (Morin et al. 1994).

Our study failed in confirming the hypothesis of a better treatment response in patients with higher predisposition for the developing insomnia as an effect of the vulnerability to stress. Drake et al. found out that individuals with high FIRST scores exposed to stressful situation exhibit longer sleep latency also after ingestion of caffeine before sleep (Drake et al. 2006). Further studies indicate a positive correlation between the incidence of stressor and the development of insomnia within 12 months in individuals with high FIRST scores. Thus, it was hypothesised that high FIRST scores may predict the development of short term and chronic insomnia (Drake et al. 2004). However, on the contrary to that hypothesis no correlations between FIRST scores and other stress related parameters (objective and subjective) and CBT efficacy were found. Our study suggests that low hyperarousal level is crucial for the CBT efficacy in the PI patient. As CBT influences hyperarousal level it cannot affect other factors important in the etiology of insomnia (eg. environmental or genetic). Thus, it seems to be the major point for the CBT intervention for the modifiable factors associated with PI.

A serious limitation of our study is the lack of polysomnographic recordings. However, a number of surveys indicate there are no substantial differences between polysomnographic recordings typical for insomniac patients and those who sleep well. Insomniac patients often exaggerate their complaints about sleep as related to cognitive hyperarousal with patients' distorted expectations and beliefs about sleep, lack of sleep and its effects during day (Frankel et al. 1976). Another study also confirmed discrepancies between objective (actygraphy) and subjective (sleep diary) sleep assessment measures (Tang et al. 2007). In our study there was also no correlation between actygraphic record and other data. Thus, there is weak evidence for the use of polysomnographic recordings in CBT efficacy studies.

CONCLUSIONS

CBT is an effective treatment in primary insomnia. It substantially improves quantity and quality parameters of sleep and diminish the level of hyperarousal in patients with PI. No relationship between CBT efficacy and predisposition to insomnia as determined by higher vulnerability to stress seems to exist.

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Conflict of interest: None to declare.

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Correspondence:

Maria Gałuszko-Węgielnik
Department of Psychiatry, Medical University of Gdańsk
Dębinki 7 st. build. 25, 80-952 Gdańsk, Poland
E-mail: mgaluszko@gumed.edu.pl