

Cognitive Behavioral Therapy for Insomnia as Treatment for Post-Concussive Symptoms

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ABSTRACT

Postconcussive symptoms (PCS) are frequently reported in those who have sustained traumatic brain injury and there are few evidence-based treatments available following the acute recovery phase. PCS includes a range of symptoms, and therefore, rehabilitation has focused more broadly on cognitive, emotional, and/or physical complaints. A common presentation with PCS is sleep concerns or insomnia, which may represent a more definitive cluster of symptoms for treatment interventions to target and enhance recovery. At present, little is known about the impact of treating sleep concerns on the expression of PCS. Therefore, the current study examined the degree to which PCS improved with treatment focused on insomnia, specifically Cognitive Behavioral Therapy for Insomnia (CBTI). In a primary care setting, thirty Veterans seeking treatment for insomnia completed measures of PCS, mood, and sleep-related variables both before and after CBTI treatment. Results suggested statistically significant and clinically meaningful improvements in PCS symptoms, depression and anxiety, and sleep-related variables in the whole sample and among those with a history of TBI. Important reductions with sleep medication were observed as well. Given these findings, targeting insomnia may represent an important focus for interventions to enhance longer term recovery in TBI populations.

Keywords: Insomnia; Military; Traumatic Brain Injury; Veterans.

Abbreviations: CBTI: Cognitive Behavioral Treatment for Insomnia; DSM-5: Diagnostic and Statistical Manual of Mental Disorders – Fifth Edition; EMA: Early Morning Awakening; HADS: Hospital Anxiety and Depression Scale; ISI: Insomnia Severity Index; mTBI: Mild Traumatic Brain Injury; NSI: Neurobehavioral Symptom Inventory; PCS: Postconcussive Symptoms; SE: Sleep Efficiency; SL: Sleep Latency; SNQ: Sleep Need Questionnaire; TBI: Traumatic Brain Injury; TST: Total Sleep Time; VA: Veterans Affairs; WASO: Wake After Sleep Onset.

INTRODUCTION

Traumatic brain injuries (TBI) of all severity levels are common among military service members returning from recent conflicts in the Middle East [1]. Correspondingly, subjective complaints of postconcussive symptoms (PCS), including of cognitive, emotional, and physical problems, have increasingly demanded clinical attention. Despite the need, few evidence-based treatments are available for PCS following the acute recovery phase [2]. This study aims to examine the effect of one potential evidence-based intervention, cognitive behavioral therapy for insomnia (CBTI) on PCS.

Mild TBI (mTBI), which comprises the bulk of all TBI cases, does not typically lead to long-term impairments beyond what would be expected given premorbid factors and comorbidities [2]. PCS is a cluster of relatively non-specific symptoms that are common among individuals who have sustained head injuries, but are equally common among individuals who present with depression or chronic pain. Research suggests that in the acute and post-acute phase of recovery, PCS can be prevented or reduced with proper rest and/or information on symptom expectation; however, there are little to no empirically supported interventions for PCS in the chronic phase [2].

Sleep problems, a common PCS, are frequent among Veterans, particularly those evaluated for head injuries [3-5] with effects lasting for years following the injury [6]. Recent estimates have estimated that approximately ninety percent of Veterans in VA polytrauma clinics have at least mild insomnia and half to two-thirds have at least moderate insomnia [7,8]. Lending support for the notion for the wide-ranging impact of poor sleep, Cantor and colleagues [9] found that sleep problems among those with remote histories of TBI was related to reduced satisfaction with life. Often, in clinical practice, these sleep concerns are overlooked and not identified as an area of clinical intervention among those with head injuries.

CBTI is an empirically validated treatment that uses behavioral conditioning, cognitive restructuring, and sleep hygiene principles to treat insomnia [10,11]. CBTI is gaining a broad evidence base in primary care as well as specialty clinics. With this evidence base, data also suggest that the benefits of CBTI spread to other aspects of functioning, to include anxiety, depression, and symptoms of post-traumatic stress disorder [12,13]. Among Veterans in a polytrauma clinic, the quantity and quality of sleep has been found to affect emotional and cognitive functioning [8]. Moreover, CBTI has augmented other

treatment interventions and suggested enhanced outcomes. For example, adding behavioral therapy for insomnia to usual care for patients with depression and insomnia produced added benefits on sleep and mood measures [14]. Additionally, adding CBTI to a cognitive behavioral intervention for patients managing chronic pain and depression demonstrated improvements in sleep and fatigue as well as with pain related disability and depression [15].

Therefore, it is reasonable to suspect that improving sleep can improve some of the cardinal symptoms of PCS. Limited research has examined therapy for sleep disorders among those with TBI. Ouellet and colleagues [16] found that CBTI was beneficial in a sample of 11 individuals with histories of TBI. In their sample, CBTI produced important benefits in insomnia, as well as physical and emotional functioning. Instead of employing a multimodal intervention for the myriad of symptoms associated with PCS, this study will examine the degree to which PCS improves with treatment focused on insomnia. The sample includes Veterans with and without a history of head injury. This decision was made because the component symptoms of PCS are not specific to TBI, and because in many cases there are no objective correlates of PCS, only subjective distress. Specific hypotheses include [1] CBTI will result in improved subjective sleep quality, reduced sleepiness, greater sleep efficiency, reduced depression/anxiety, and a reduction in symptoms that are commonly attributed to concussions [2]. These treatment effects will be observed regardless of whether individuals have sustained concussions in the past.

METHODS

Participants

Thirty-five patients seen within a VA Healthcare System primary care setting for complaints of insomnia were recruited for this study. Thirty Veterans completed the requirements of the study including undergoing CBTI for an overall completion rate of 85.7%. Average age of participants was 51.23 ± 13.48 and 23 (76.7%) were male. All participants completed at least five sessions of CBI with an overall average of 7.03 ± 2.04 sessions. Twenty-eight (93.3%) had psychiatric and/or substance use disorder comorbidities, 13 (43.3%) reported chronic pain, 21 (70%) were taking sleep medications at intake, 8 (26.7%) had history of or current diagnosis of obstructive sleep apnea, 5 (16.7%) had history of or current diagnosis of restless leg syndrome, and 11 (36.7%) met American Congress of Rehabilitation Medicine criteria for mTBI [17]. Psychiatric

and chronic pain disorders were generally diagnosed prior to treatment, but CBTI providers confirmed these diagnoses at intake. In every case, prescriptions for sleep medications were made prior to the referral for CBTI, but there were no data available regarding when these medications were initiated. TBI diagnoses were made by the first author based on self-reported injury characteristics. By patient estimates, the onset of insomnia occurred 12.97 to 12.99 years ago, more specifically patients estimated onset within the past 10 years for 18 Veterans, 11 to 25 years for 8 Veterans, and more than 25 years ago for 4 Veterans. All participants met DSM5 criteria for Insomnia Disorder [18] and reported some type of daytime impairment related to sleeping concerns including daytime fatigue (93.3%), concentration problems (76.7%), and mood difficulties (76.7%).

MATERIALS AND MEASURES

Participants were given the Hospital Anxiety and Depression Scale (HADS) [19], which is a 14-item measure of anxiety and depression at both intake and end of treatment. For both the Anxiety and Depression subscales, scores of 8 to 10 are considered mild, 11 to 14 moderate, and 15 to 21 severe. Participants were also given the Insomnia Severity Index (ISI) [20], which is a seven-item measure of subjective difficulties with sleep at each session throughout the study. Scores of 8 to 14 are considered mild, 15 to 21 moderate, and 22 to 28 severe. To assess need for sleep, participants were given the Sleep Need Questionnaire [21] at each session throughout the study. Ranges of scores are 4-20 with higher scores indicating subjective desire for more sleep. Participants were also given the Neurobehavioral Symptom Inventory (NSI) [22], which is a self-rating of 22 commonly reported postconcussive symptoms. These symptoms have been parsed into four factors (affective [six items], cognitive [four items], somatic [seven items], and vestibular [three items]) by Meterko, et al. [23]. Two items do not load unambiguously on any single factor, and are therefore only included in the total score and not on any of the factors. The total score and affective factor score includes one item pertaining to sleep quality. Therefore, this item will be omitted from some analyses below. There are no set standards for quantifying levels of impairment with the NSI. Each item is scored from zero (no problems) to four points (very severe problems).

Participants were instructed to complete a sleep diary daily upon awakening based on their recall of the previous night sleep. Weekly sleep diaries were completed prior to each visit following the intake. From the diaries, the following variables

were calculated as weekly averages: sleep latency (SL) referring to number of minutes from trying to go to sleep and falling asleep, wake time after sleep onset (WASO) referring to number of minutes awake after sleep onset, early morning awakening (EMA) indicating number of minutes waking earlier than desired, sleep efficiency (SE; percentage of time the participant slept between entering and exiting the bed), and total sleep time (TST) in minutes.

Procedures

Prospective participants were invited to participate and underwent informed consent. The study was approved by the Institutional Review Board and Research and Development Committees. All participants underwent an intake evaluation to assess for insomnia and potential comorbid problems. All prospective participants completed intake measures, including the HADS, ISI, SNQ, and NSI at the intake appointment. Those prospective participants meeting criteria for insomnia were offered CBTI and instructed on how to complete the sleep diary. At session two, baseline sleep diary data were obtained. Participants kept a sleep diary throughout the study and completed the ISI and SNQ at each visit. At the final session, participants completed the HADS, ISI, SNQ, and NSI and submitted their final sleep diary.

Broadly speaking, CBTI is a family of treatments that employ behavioral conditioning, cognitive restructuring, and sleep hygiene principles to treat insomnia [11,12]. The procedures used in the current study were part of the National VA training on insomnia [12] and the clinicians providing services had completed this training program and consultation.

RESULTS

Participants who completed treatment were compared to those who prematurely dropped out of treatment on demographic variables, initial sleep estimation variables, and initial questionnaire variables. There were no significant differences between completers and non-completers on any variable except for baseline sleep medication. Completers were more 9.33 times more likely to be on sleep medication at baseline than non-completers, $\chi^2(1)=4.52, p<0.05$. Therefore, non-completers were excluded for the remainder of the analyses.

Analyses of PCS

To examine the effects of CBTI on PCS, repeated measures analyses were conducted on NSI variables for all participants.

There was a significant decrease in total NSI symptoms (excluding the sleep item) pretreatment to posttreatment, $t(29)=7.09$, $p<0.001$, $d=1.54$, a significant decrease in the affective subscale of the NSI, $t(29)=5.94$, $p<0.001$, $d=1.33$, a significant decrease in the somatic subscale of the NSI, $t(29)=4.06$, $p<0.001$, $d=0.47$, a significant decrease in the cognitive subscale of the NSI, $t(29)=5.25$, $p<0.001$, $d=1.03$, and a significant decrease in the vestibular subscale of the NSI, $t(29)=3.5$, $p<0.01$, $d=0.62$. As noted, effects sizes were moderate to large with larger effects seen for total symptoms, affective symptoms, and cognitive symptoms.

Effects of CBTI on PCS among those with identified histories of TBI were also calculated. There was a significant decrease in total NSI symptoms (excluding the sleep item) pretreatment to posttreatment, $t(10)=5.23$, $p<0.001$, $d=1.68$, a significant decrease in the affective subscale of the NSI, $t(10)=3.79$, $p<0.001$, $d=1.46$, a significant decrease in the cognitive subscale of the NSI, $t(10)=4.16$, $p<0.01$, $d=1.38$, and the vestibular subscale of the NSI, $t(10)=2.67$, $p<0.05$, $d=0.81$. As noted these are all large effect sizes. There were no significant differences pre-post on the somatic subscale of the NSI for those with history of TBI. Mean differences among variables including effect sizes are listed in **Table 1**.

Table 1: Pre- and post-treatment test scores.

Variable	Sample Total=30 mTBI=11	Pre-Treatment	Post-Treatment	Significance	Effect size (d or r)
ISI	Total	19.47 ± 4.0	6.6 ± 5.0	$t(29)=12.0$, $p<0.001$	2.84
	mTBI	19.64 ± 4.23	5.18 ± 3.57	$t(10)=8.77$, $p<0.001$	3.69
SNQ	Total	14.53 ± 1.81	9.8 ± 2.12	$t(29)=9.05$, $p<0.001$	2.4
	mTBI	14.82 ± 1.94	9.64 ± 2.01	$t(10)=5.01$, $p<0.01$	2.62
NSI Sleep Item (used variable R)	Total	3.0 ± 0.95	1.0 ± 0.95	$t(29)=9.57$, $p<0.001$	2.11
	mTBI	3.18 ± 0.75	0.91 ± 0.7	$t(10)=8.33$, $p<0.001$	3.13
*NSI Total	Total	30.0 ± 9.6	16.93 ± 7.26	$t(29)=7.09$, $p<0.001$	1.54
	mTBI	29.73 ± 9.89	14.91 ± 7.66	$t(10)=5.23$, $p<0.001$	1.68
*NSI Affective	Total	10.3 ± 4.13	5.5 ± 2.97	$t(29)=5.94$, $p<0.001$	1.33
	mTBI	11.82 ± 4.53	5.91 ± 3.48	$t(10)=3.79$, $p<0.01$	1.46
NSI Somatic	Total	6.23 ± 4.13	4.43 ± 3.54	$t(29)=4.06$, $p<0.001$	0.47
	mTBI	5.36 ± 4.86	4.27 ± 4.9	$t(10)=1.64$, ns	0.22
NSI Cognitive	Total	6.37 ± 2.7	3.7 ± 2.47	$t(29)=5.25$, $p<0.001$	1.03
	mTBI	6.1 ± 3.33	2.36 ± 1.91	$t(10)=4.16$, $p<0.01$	1.38
NSI Vestibular	Total	2.37 ± 2.2	1.23 ± 1.36	$t(29)=3.5$, $p<0.01$	0.62
	mTBI	1.73 ± 1.79	0.55 ± 1.04	$t(10)=2.67$, $p<0.05$	0.81
HADS Anxiety	Total	10.27 ± 3.71	7.6 ± 3.85	$t(29)=4.55$, $p<0.001$	0.71
	mTBI	10.46 ± 3.24	7.09 ± 3.39	$t(10)=3.67$, $p<0.01$	1.02
HADS Depression	Total	8.2 ± 3.14	5.87 ± 3.77	$t(29)=4.02$, $p<0.001$	0.67
	mTBI	8.18 ± 3.16	7.0 ± 3.29	$t(10)=1.28$, ns	0.37
Taking Meds	Total	20(66.7%)	10(33.3%)	$X^2(1)=7.5$, $p<0.01$	$r=0.5$
	mTBI	7(63.6%)	3(27.3%)	$X^2(1)=2.36$, ns	$r=0.46$
SL	Total	47.79 ± 50.12	12.08 ± 7.64	$t(29)=4.0$, $p<0.001$	1.0
	mTBI	51.18 ± 48.51	13.52 ± 9.03	$t(10)=2.65$, $p<0.05$	1.08
WASO	Total	49.83 ± 46.56	15.5 ± 17.13	$t(29)=4.37$, $p<0.001$	0.98
	mTBI	56.02 ± 43.41	11.12 ± 6.68	$t(10)=3.57$, $p<0.01$	1.45
EMA	Total	39.89 ± 44.8	5.95 ± 10.39	$t(29)=4.47$, $p<0.001$	1.04
	mTBI	10.46 ± 3.24	7.09 ± 3.39	$t(10)=3.67$, $p<0.01$	1.02
SE	Total	73.29 ± 15.21	91.44 ± 5.07	$t(29)=-7.69$, $p<0.001$	-1.6
	mTBI	73.63 ± 12.91	92.62 ± 3.14	$t(10)=-5.59$, $p<0.001$	-2.02
TST	Total	5.82 ± 1.46	6.32 ± 0.95	$t(29)=-3.18$, $p<0.01$	-0.41
	mTBI	6.1 ± 1.45	6.45 ± 1.08	$t(10)=-1.5$, ns	-0.27

*Omitting sleep item

Other outcomes of CBTI

Results demonstrate significant reduction in ISI scores pre-post, $t(29)=12.0$, $p<0.001$, $d=2.84$, a significant reduction in sleep need, $t(29)=9.05$, $p<0.001$, $d=2.4$, a significant reduction in anxiety symptoms, $t(29)=4.55$, $p<0.001$, $d=0.71$, and a significant reduction in depressive symptoms, $t(29)=4.02$, $p<0.001$, $d=0.67$, consistent with previous research. Furthermore, when examining CBTI effects on sleep diary outcome data, significant improvements were found in SL, $t(29)=4.0$, $p<0.001$, $d=1.0$, WASO, $t(29)=4.37$, $p<0.001$, $d=0.98$, EMA, $t(29)=4.47$, $p<0.001$, $d=1.04$, SE, $t(29)=-7.69$, $p<0.001$, $d=-1.6$, and TST, $t(29)=-3.08$, $p<0.01$, $d=-0.41$.

Similar outcomes were found in those reporting histories of TBI. There was a significant reduction in ISI, $t(10)=8.77$, $p<0.001$, $d=3.69$, sleep need, $t(10)=5.01$, $p<0.01$, $d=2.62$, anxiety, $t(10)=3.67$, $p<0.01$, $d=1.02$. No significant differences were found on depression, $t(10)=1.28$, *ns*, $d=0.37$, yet the raw means note a moderate effect. There also were significant improvement in SL $t(10)=2.65$, $p<0.05$, $d=1.08$, WASO, $t(10)=3.57$, $p<0.01$, $d=1.45$, EMA, $t(10)=3.69$, $p<0.01$, $d=1.02$, and SE, $t(10)=-5.59$, $p<0.001$, $d=-2.02$. There were no significant differences on TST, $t(10)=-1.5$, *ns*, $d=-0.27$.

To further examine if TBI status affected outcomes, change scores on the pertinent variables were calculated by subtracting post scores from baseline scores. There were no significant differences between those with a history of TBI and those with no history of TBI on any sleep variable (SL, WASO, EMA, TST, SE), any post concussive symptom variable (NSI total, NSI sleep, NSI affective, NSI cognitive, NSI somatic, NSI vestibular), and any other outcome variable (depression, anxiety, ISI, sleep need).

Chronic pain status was examined to determine if it influenced change score outcomes. There were no significant differences between those with a reported history of chronic pain and those with no history of chronic pain on any sleep variable (SL, WASO, EMA, TST, SE) and on any psychiatric or sleep related variable (depression, anxiety, ISI, sleep need). In terms of post concussive symptoms, those with chronic pain reported significant more change in total NSI score ($M=17.23 \pm 9.69$) than those without chronic pain ($M=9.88 \pm 9.44$), $t(28)=-2.09$, $p<0.05$, $d=0.77$ as well as significant more change on the affective subscale of the NSI ($M=6.69 \pm 4.39$ vs. 3.35 ± 4.0) $t(28)=-2.17$, $p<0.05$, $d=0.81$. There were no significant differences on other subscales of the NSI according to chronic pain status.

Given some shared symptoms or underlying constructs of the outcomes that were assessed (i.e., sleep disturbance as a symptom of depression), further correlations between measures were evaluated. There was no significant correlation with length of time with insomnia and change in ISI, $r(30)=0.09$, *ns*. Furthermore, there were no significant correlations with length of time with insomnia and change in depression, anxiety, or any sleep-related variable (SL, WASO, EMA, TST). However, there was a significant positive correlation with length of time with insomnia and change in NSI vestibular, $r(30)=0.38$, $p<0.05$, and NSI total, $r(30)=0.40$, $p<0.05$. No correlations on other NSI subscales.

Associations of treatment with the use of sleep aid medication were also examined in the sample. Twenty (66.7%) were taking some sleep aid at baseline. Following treatment, of those on meds at baseline, 6 (30%) had no changes in meds, 4 (20%) reduced meds, and 10 (50%) stopped meds, $X^2(1)=7.5$, $p<0.01$, $r=0.5$. Among only those with TBI, 7 (63.6%) were taking some sleep aid at baseline and following treatment only 3 (27.3%) were taking sleep aides, $X^2(1)=2.36$, *ns*.

DISCUSSION

This study found that the component symptoms of PCS, along with aspects of insomnia, depression, and anxiety, abate with CBTI. These treatment effects were observed among those with and without a history of TBI. That is, the symptoms commonly classified under the heading of PCS resolved for individuals with and without a history of head injury. Furthermore, treatment effects were observed among individuals with all levels of insomnia chronicity. These results lend support to the notion that focusing treatment efforts on improving sleep can have benefits for individuals with multiple problems, as is common in the VA TBI population [1,24].

Currently, there are few treatment options in the post-acute phase of TBI. Acutely, TBI is often treated with rest and provision of information relating to the expected trajectory of recovery. In the chronic phase of TBI, however, repeated evaluations and reassurances might have limited impact [25]. These individuals have sustained injuries years prior to VA treatment, and so have accumulated years of apparent evidence of intractable problems from their head injury. CBTI did not specifically address each aspect of PCS, only sleep. Nevertheless, improved sleep appears to be associated with improved functioning in many aspects of subjective emotional, cognitive, and somatic functioning. This approach to treatment has the advantage of diverting focus from the Veteran's diagnostic status, which

can be viewed by many Veterans as immutable. Excessive attention to "TBI" risks feeding into attribution biases of disability, whereas CBTI offers a clear and tangible method for bringing about substantive change.

Given the promising results of this study, additional research should include control interventions. The argument can be made that the symptomatic improvement observed in this study was a function of the high level of distress at the time of initial assessment compared to reevaluation at a later point in time. The Veterans in the current study reported on average approximately 13 years of insomnia, making spontaneous recovery in the present sample unlikely. Improvement could also have been the demand characteristics or non-specific clinical attention. Inclusion of a control group receiving a control intervention would address this issue. To date, control interventions in CBTI studies have produced minimal meaningful changes [13, 26].

Additional research is needed exploring the effects of CBTI in polytrauma clinics. The individuals in the present study were seeking treatment for insomnia, not TBI. TBI status was assessed, but not directly addressed in the application of CBTI. On the other hand, in polytrauma clinics, Veterans tend to be younger, mostly male, and oftentimes have preexisting beliefs related to the prognosis from head injury. For these individuals, CBTI has the advantage of being a brief, flexible, and focused treatment approach that offers the potential for meaningful reduction of chronic PCS.

Although subjective somatic and cognitive improvement was noted, additional research is needed to confirm these findings using more objective methodologies. Research indicates that subjective cognitive functioning is poorly associated with performance on objective neuropsychological testing [27,28]. In a heterogeneous sample of individuals with TBI and sleep-wake disorders, Wiseman-Hakes and colleagues [24] found that treatment of the sleep disorders led to improvements in cognitive performance on objective testing. Their results are regarded as preliminary, however, as the study did not employ a control group to account for practice effects. Additional research is needed to examine the effects of CBTI on objective cognitive testing. Yet, the present study does provide preliminary evidence that CBTI is an effective intervention for PCS.

CONCLUSION

CBTI is a promising intervention for reducing the core symptoms of PCS and reducing symptoms of depression and

anxiety. Confirmation is needed from randomized clinical trials.

Disclosure Statement

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