Intensive Sleep Deprivation and Cognitive Behavioral Therapy for Pharmacotherapy Refractory Insomnia in a Hospitalized Patient

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CASE REPORTS

The case of a 59-year-old woman psychiatrically hospitalized with comorbid insomnia, suicidal ideation, and generalized anxiety disorder is presented. Pharmacologic therapies were unsuccessful for treating insomnia prior to and during hospitalization. Intensive sleep deprivation was initiated for 40 consecutive hours followed by a recovery sleep period of 8 hours. Traditional components of cognitive behavioral therapy for insomnia (CBTi), sleep restriction, and stimulus control therapies, were initiated on the ward. After two consecutive nights with improved sleep, anxiety, and absence of suicidal ideation, the patient was discharged. She was followed in the sleep clinic for two months engaging in CBTi. Treatment resulted

in substantial improvement in her insomnia, daytime sleepiness, and anxiety about sleep. Sleep deprivation regimens followed by a restricted sleep recovery period have shown antidepressant effects in depressed patients. Similar treatment protocols have not been investigated in patients with pharmacotherapy refractory insomnia and generalized anxiety disorder.

Keywords: insomnia; anxiety; depression; sleep deprivation; sleep restriction therapy; hypnotics

Citation: Breitstein J, Penix B, Roth BJ, Baxter T, Mysliwiec V. Intensive sleep deprivation and cognitive behavioral therapy for pharmacotherapy refractory insomnia in a hospitalized patient. *J Clin Sleep Med* 2014;10(6):689-690.

Approximately 9% to 12% of the population is affected by chronic insomnia with research exhibiting a strong correlation between insomnia, depression, and anxiety.¹ Treatment of insomnia often involves multicomponent cognitive behavioral therapy for insomnia (CBTi) or sedative-hypnotic medications.² CBTi has greater efficacy in the long-term treatment of insomnia compared to medical therapy, which often loses effectiveness over time.³ Sleep restriction, a component of CBTi, has been successful for insomnia patients failing chronic sedative hypnotic therapy.² Although sleep deprivation may increase affective symptoms and anxiety in healthy adults, sleep deprivation followed by sleep restriction can have antidepressant effects.⁴ Previous studies have demonstrated a single night of monitored sleep deprivation can markedly improve depressive symptoms.⁵⁻⁷ Notably, there is a paucity of literature examining sleep deprivation combined with CBTi in patients with psychiatric disorders who have pharmacotherapy refractory insomnia. This case demonstrates the efficacy of intensive sleep deprivation followed by sleep restriction and stimulus control in an inpatient. The patient had improved sleep quality, decreased anxiety, and resolution of suicidal ideation, all of which had deteriorated on medical therapy alone.

REPORT OF CASE

A 59-year-old married woman was admitted to a psychiatric ward for suicidal ideation due to pharmacotherapy refractory insomnia. She developed difficulties falling and staying asleep after undergoing an uncomplicated hysterectomy 10 weeks prior to admission. As an outpatient her generalized anxiety disorder (GAD) and insomnia was treated with paroxetine 15 mg in

the morning and lorazepam 0.5-2 mg at bedtime. Quetiapine 150 mg at bedtime was added 4 weeks prior to admission. She tapered lorazepam out of fear of addiction. Although she initially improved, her difficulties sleeping worsened despite medical therapy resulting in a persistent fear of never being able to sleep again. Fireworks on the Fourth of July resulted in two consecutive days of no sleep. She presented to our emergency room with a plan to either overdose or walk in front of a train due to her inability to sleep. Her past medical history was notable for a previous episode of severe insomnia and major depression that required an inpatient psychiatric hospitalization to stabilize. She did not present with any depressive symptoms, hallucinations, or delusional thinking at the time of admission. Admission diagnoses were DSM-IV-TR 304.72 Insomnia and 300.02 Generalized Anxiety Disorder (GAD). During the initial portion of her hospitalization, her insomnia did not respond to pharmacologic therapy (eszopiclone 3 mg and zolpidem 5 mg). The patient and ward staff reported she slept less than 3 hours. Clinical evaluation by the sleep medicine service revealed the patient had difficulty initiating and maintaining sleep due to her anxiety. No additional dyssomnias or parasomnias were present. Medical therapies for her GAD consisted of mirtazapine 15 mg at bedtime and clonazepam 0.5 mg three times daily.

All sedative hypnotic medications were discontinued. Mirtazapine and clonazepam were continued for anxiety. The patient was treated with sleep deprivation, having 40 hours of visually monitored wakefulness (06:00 day one to 22:00 day two). This was followed by 8 hours of recovery sleep from 22:00 to 06:00; she reported 7 hours of good quality sleep. The patient stated sleep deprivation alleviated her anxiety about not sleeping. Treatment with sleep restriction of 5 hours' time in

Figure 1—Changes in Insomnia Severity Index (ISI) score, Epworth Sleepiness Scale (ESS) score, and self-reported (SR) sleep duration from pre-treatment to post-treatment.

Inpatient stay from July 5 to July 16; outpatient July 18 to September 12.

bed was initiated. Following two nights of consolidated sleep and devoid of suicidal ideation or significant anxiety she was discharged. She continued treatment in the sleep clinic for 2 months engaging in CBTi and gradually increasing time in bed to 7 hours 45 minutes using her sleep diary. Subjective sleep quality markedly improved, as did her scores on the Insomnia Severity Index and Epworth Sleepiness Scale (**Figure 1**). At 2 months follow-up, she no longer met criteria for insomnia. Her anxiety surrounding sleep had resolved. She remained on mirtazapine and clonazepam for her GAD.

DISCUSSION

This case illustrates that a treatment protocol consisting of intensive sleep deprivation followed by sleep restriction and stimulus control therapy rapidly treated severe, pharmacotherapy refractory insomnia in a patient with comorbid anxiety and suicidal ideation in a psychiatric inpatient setting. The treatment response was further augmented with outpatient CBTi. Although sleep deprivation has been used for inpatient depression treatment,⁴⁻⁹ this report is the first to our knowledge documenting the use of intensive sleep deprivation for severe insomnia in a hospitalized patient with GAD and suicidal ideation.

Sleep restriction therapy for insomnia has been reported in the inpatient setting, but the therapy required 2 weeks rather than a few days.⁹ Intensive sleep retraining, which involves 24+ hours of sleep deprivation and multiple EEG monitored naps, has demonstrated efficacy in rapidly treating insomnia, but requires EEG laboratory monitoring.⁸ It is not clear if sleep deprivation or conditioning from frequent naps provides the major benefit of this therapy.

Sleep deprivation therapy was chosen for the patient in combination with traditional CBTi principles for the following reasons: First, sleep deprivation seems to impact natural circadian rhythms that have been disrupted and can reduce arousal levels.⁵ Secondly, sleep deprivation directly challenged the patient's cognitive distortions that she would

never sleep normally again and remain awake forever. Finally, the patient received no therapeutic response from medical therapy. Her treatment response to sleep deprivation occurred in a timeline rivaling that expected from sedative-hypnotic medication. Potential mechanisms for her improvement included an enhanced sleep drive, decreased anxiety about falling and/or staying asleep, and a relearned ability to initiate and maintain sleep. It is conceivable these cognitive and behavioral shifts would not have occurred with medical therapy alone.

The literature reports sleep deprivation can achieve an acute and lasting antidepressant effect.^{6,7} We are unaware of any reports of sleep deprivation ameliorating anxiety symptoms. Our patient reported decreased anxiety during the period of sleep deprivation, noting she no longer had concerns about sleeping.

Limitations of this report are that the patient was not monitored with actigraphy or EEG during her sleep deprivation, and we relied on self-reported symptoms and sleep duration. Additionally, it cannot be ruled out that the medications she received (i.e., mirtazapine and clonazepam) resulted in decreased anxiety and improved sleep over time. Regardless, this outcome warrants further research into the effectiveness of intensive sleep deprivation followed by CBTi in the treatment of pharmacotherapy refractory insomnia in adults with comorbid psychopathology.

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SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication November, 2013

Submitted in final revised form February, 2014

Accepted for publication February, 2014

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DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest. The opinions and assertions in this manuscript are those of the authors and do not necessarily represent those of the Department of the Army, Department of Defense, or U.S. Government.