CASE REPORT

A Case of Extreme Paradoxical Insomnia Responding Selectively To Electroconvulsive Therapy

Kristen Case, M.D.¹; Thomas D. Hurwitz, M.D.^{1,3}; S.W. Kim, M.D.¹; Michel Cramer-Bornemann, M.D.^{2,4}; Carlos H. Schenck, M.D.^{1,4}

Departments of ¹Psychiatry and ²Neurology, University of Minnesota Medical School, Minneapolis, MN; ³Minneapolis Veterans Affairs Medical Center, Minneapolis, MN; and ⁴Minnesota Regional Sleep Disorders Center, Minneapolis, MN

Severe paradoxical insomnia, documented by actigraphy, was the predominant presenting complaint of a 48-year-old woman subsequently diagnosed with major depression. Both disorders remitted following a course of 5 electroconvulsive therapy treatments in spite of being previously refractory to hypnotic and antidepressant pharmacotherapy. **Keywords:** Actigraphy, insomnia, sleep state misperception, paradox-

E limination of insomnia, a precursor and often concurrent symptom of clinical depression, is an important component of treatment undertaken to achieve complete remission of depression.¹ Electroconvulsive therapy (ECT) is a safe, effective, generally well-tolerated treatment for major depression, including depression with atypical features. The following is a case of major depression presenting as severe insomnia that was selectively responsive to ECT.

REPORT OF CASE

A 48-year-old married, employed female with no prior psychiatric history was admitted to hospital with a suicidal plan "because no one can figure out what is wrong with me." She complained of a total of "35 hours sleep during the previous month," unresponsive to clonazepam 1mg, temazepam 30 mg, zolpidem 20 mg, or quetiapine 25 mg at bedtime. She attributed this to a 3-day course of prednisone 40 mg for an allergic reaction to radiopaque venogram dye prior to emergency surgery for superficial phlebitis, and gastritis from subsequent nonsteroidal antiinflammatory use.

In hospital, mirtazapine 30 mg, and sequentially prescribed temazepam 30 mg, zolpidem 10 mg, zaleplon 10 mg, trazodone 150 mg at bedtime, and oxazepam 15 mg twice daily did not help her sleep. Mirtazapine 30 mg nightly (as antidepressant)

Disclosure Statement

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

Submitted for publication March, 2007 Accepted for publication October, 2007

Address correspondence to: Dr. Thomas D. Hurwitz, MD. Department of Psychiatry, 116A, Minneapolis VAMC, Minneapolis, MN 55417. Tel: (612) 725-2000; Fax: (612) 725-2292; E-mail: hurwi001@tc.umn.edu.

ical insomnia, major depression, electroconvulsive therapy, ECT **Citation:** Case K; Hurwitz TD; Kim SW; Cramer-Bornemann M; Schenck CH. A case of extreme paradoxical insomnia responding selectively to electroconvulsive therapy. *J Clin Sleep Med 2008*;4(1):62-63.

was continued, and after 10 days she reported minimally improved sleep and mood. Venlafaxine 37.5 mg daily was added, and chloral hydrate 2 gm was ordered at bedtime. Despite relentless subjective insomnia, she appeared to remain in bed throughout the night and described minimal daytime sequelae. There were no clinical features of other sleep disorders. She had become hopeless with fear of sleeplessness, and a diagnosis of major depression was established.

Mirtazapine was changed to paroxetine 40 mg daily, associated with reports of 4-5 hours of subjective sleep during each of her last 2 nights in hospital. Four days after discharge from hospital, she was readmitted with suicidal ideation "worse than ever." She described no subjective sleep and auditory hallucinations. Wrist activity monitoring (actigraphy) was started.² Chloral hydrate, zolpidem, oxazepam, and topiramate were tapered and discontinued, while quetiapine was titrated to 500 mg at bedtime with no change in her subjective report of insomnia over 2 weeks before electroconvulsive therapy was begun. She declined to complete a sleep-wake diary. In spite of her perception to the contrary, actigraphy clearly indicated consolidated periods of apparent sleep (Fig. 1). This supported a diagnosis of paradoxical insomnia, defined as "a complaint of severe insomnia that occurs without evidence of objective sleep disturbance and without the level of daytime impairment commensurate with the degree of sleep deficits reported."3

With increasing hopelessness, helplessness, and conviction that intractable insomnia would cause permanent harm, she underwent 5 standard bilateral electroencephalographically monitored ECT treatments at a rate of 3/week.

Following the first treatment, in stark contrast to prior reports, she described 7 hours of nocturnal sleep. This improvement persisted and, when discharged from hospital, insomnia, depressed mood, hopelessness, psychomotor slowing, and suicidal ideation were absent. Using quetiapine at bedtime and venlafaxine by day, she refused further ECT as an outpatient, because she "no longer needed help with my sleep." For the



Figure 1—This tracing represents accumulated counts of wrist movements in 1-minute time bins across lines of 24-hours duration. Data collected over 3 weeks indicate clearly demarcated periods consistent with intact nocturnal sleep during one week before, 9 nights during, and 4 nights immediately following administration of 5 ECT treatments. Apparent sleep duration before ECT, when she complained of severe insomnia, is approximately at least 7-8 hours.

next 12 months, she continued venlafaxine, 225 mg, and quetiapine 200 mg at night. By two years after initial presentation, she had successfully discontinued quetiapine and diminished venlafaxine to 37.5 mg daily without further relapse of depressive symptoms or subjective insomnia.

DISCUSSION

Various unconventional therapies for severe insomnia with or without associated depressive symptoms have appeared in the literature. Treatment of lifelong insomnia (not associated with depression) with chronic nightly opioid medication has been reported, including a review of the literature on atypical treatments of extreme insomnia.4 Based upon Medline, PsycInfo, and Embase literature searches, this appears to be the first published case of paradoxical insomnia (formerly called "sleep state misperception") responsive to ECT. Unlike patients with primary, and certainly paradoxical insomnia, those with depressive disorders are typically more accurate in their subjective perceptions of sleep, making this case a unique subtype for whom complete remissions of major depression and paradoxical insomnia were maintained at a 24-month follow-up visit. Before ECT, her insomnia complaint had been refractory to pharmacotherapy, including quetiapine, and this subjective misperception of sleep normalized with the treatment. Even without sleep diary information, her consistent self-reports before and after ECT were clearly different. ECT is well established as safe, effective, and generally well-tolerated treatment for major depression. Insomnia commonly precedes major depression.^{5,6} This case establishes that paradoxical insomnia can also herald major depression of suicidal severity, and documents the impact of ECT on the symptoms common to both.

REFERENCES

- 1. Fava M. Daytime Sleepiness and Insomnia as Correlates of Depression. J Clin Psychiatry 2004;65:27-32.
- Littner M, Kushida C, Bailey D, et al. Practice parameters for the role of actigraphy in the study of sleep and circadian rhythms: an update for 2002. An American Academy of Sleep Medicine Report. Sleep 2003;26:337-41.
- American Academy of Sleep Medicine. International classification of sleep disorders, 2nd ed.: Diagnostic and coding manual. Westchester, IL: American Academy of Sleep Medicine, 2005.
- Schenck CH, Mahowald MW. Severe, childhood-onset, idiopathic, life-long insomnia responding selectively to opiate therapy: case report with 19 year follow-up. Sleep Med 2001;2:531-6.
- Breslau N, Roth T, Rosenthal L, et al. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. Biol Psychiatry 1996;39:411-18.
- Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention. JAMA 1989;262:1479-84.