Effects of a Course of Right Unilateral Ultrabrief Pulse Electroconvulsive Therapy Combined With Venlafaxine on Insomnia Symptoms in Elderly Depressed Patients

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ABSTRACT

Objective: Antidepressant medications have a variety of effects on sleep, apart from their antidepressant effects. It is unknown whether electroconvulsive therapy (ECT) has effects on perceived sleep in depressed patients. This secondary analysis examines the effects of ECT on perceived sleep, separate from its antidepressant effects.

Methods: Elderly patients with major depressive disorder, as defined by *DSM-IV*, received open-label high-dose, right unilateral ultrabrief pulse ECT, combined with venlafaxine, as part of participating in phase 1 of the National Institute of Mental Health–supported study Prolonging Remission in Depressed Elderly (PRIDE). Phase 1 of PRIDE participant enrollment period extended from February 2009 to August 2014. Depression severity was measured with the Hamilton Depression Rating Scale-24 item ($HDRS_{24}$), and measures of insomnia severity were extracted from the $HDRS_{24}$. Participants were characterized at baseline as either "high-insomnia" or "low-insomnia" subtypes, based upon the sum of the 3 $HDRS_{24}$ sleep items as either 4–6 or 0–3, respectively. Insomnia scores were followed during ECT and were adjusted for the sum of all the $HDRS_{24}$ non-sleep items. Generalized linear models were used for longitudinal analysis of insomnia scores.

Results: Two hundred forty patients participated, with 48.3% in the high-insomnia and 51.7% in the low-insomnia group. Although there was a reduction in the insomnia scores in the high-insomnia group, only 12.4% of them experienced remission of insomnia after a course of ECT, despite an increase in utilization of sleep aids across the course of ECT, from 8.6% to 23.2%. The degree of improvement in insomnia symptoms paralleled the degree of improvement in non-insomnia symptoms. A "low" amount of improvement on the sum of the HDRS non-insomnia items (HDRS-sleep) was accompanied by a "low" amount of improvement in insomnia scores (change of -1.6 ± 1.2, $P < .0001$), while a "high" amount of improvement on the sum of the HDRS non-insomnia items was accompanied by a "higher" amount of improvement in insomnia scores (change of -3.1 ± 1.6, $P < .0001$). After adjustment for non-insomnia symptoms, there was no change in insomnia in the low-insomnia group.

Conclusions: We found that ECT, combined with venlafaxine, has a modest anti-insomnia effect that is linked to its antidepressant effect. Most patients will have some degree of residual insomnia after ECT, and will require some consideration of whether additional, targeted assessment and treatment of insomnia is warranted.

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Sleep disturbance is common among older people, with more than 50% of people older than 65 years reporting sleep difficulties.[1] Insomnia is among the symptoms of major depressive disorder (MDD),[2] and older MDD patients more often state insomnia is a focus of concern, while younger patients do not.[3,4] The presence of insomnia complicates the presentation of MDD. It was an independent risk factor for poorer quality of life in a mixed-age sample of patients with MDD,[5] and treatment of insomnia has been shown to improve the quality of life in middle aged patients with MDD.[6] Further, the presence of sleep problems was found to be an independent risk factor for greater intensity of suicidal ideation in a mixed-age sample of MDD[7] and is associated with greater rates of suicide death in the elderly.[8,9] The persistence of insomnia in older MDD patients, after otherwise successful resolution of an MDD episode, forebodes increased risk of MDD relapse, at least in older MDD patients who achieved remission through interpersonal psychotherapy.[10] Thus, optimum treatment of MDD ought to include relief from insomnia symptoms.

Most treatments of MDD produce improvement in insomnia symptoms. Early comparisons of psychotherapy versus tricyclic antidepressants (TCAs) reported that insomnia symptoms were among the first symptoms to improve with TCAs, before general improvement in mood was seen. This pattern of change was not seen with psychotherapy.[11] Some sedating antidepressants have benefits for insomnia symptoms that occur independent of a diagnosis of MDD or any other psychiatric disorder. For example, low doses of either trazodone or the TCA doxepin are associated with improvement in sleep in nondepressed

insomniacs, and these benefits are evident both in patient self-report and in polysomnography.[12,13] This pattern is not seen with selective serotonin reuptake inhibitors (SSRIs), as these medicines may improve sleep complaints in patients with MDD, without necessarily improving objective sleep as reflected in polysomnography.[14]

Information regarding the impact of electroconvulsive therapy (ECT) and repetitive transcranial magnetic stimulation (rTMS) on insomnia symptoms in MDD is limited. More is known about the impact of ECT on polysomnography sleep than is known regarding ECT and self-reported sleep. The methodology is inconsistent across studies of ECT's effects on polysomnography sleep, but the general finding is that ECT is associated with increases in polysomnography total sleep time and suppression of rapid eye movement sleep.[15] rTMS is associated with reduction in insomnia symptoms, but the improvement in insomnia symptoms is linked with improvement in the overall MDD syndrome, that is, rTMS does not seem to have an anti-insomnia effect apart from its antidepressant effect.[16]

We have recently completed a study of ECT in the elderly, entitled Prolonging Remission in Depressed Elderly (PRIDE). Phase 1 of PRIDE was an open-label study of the effects of high-dose, right unilateral ultrabrief pulse (RUL-UBP) ECT augmented with venlafaxine in a large sample of elderly MDD patients, while phase 2 was a randomized comparison of outpatient continuation ECT plus pharmacotherapy versus pharmacotherapy alone in prolonging remission for those participants who had remitted in phase 1. The primary aim of phase 1 was to describe the efficacy and safety of RUL UBP ECT, and the methods and efficacy results of phase 1 are reported elsewhere.[17] We conducted a secondary analysis of phase 1 PRIDE data for the purpose of (1) describing the impact of RUL UBP ECT on insomnia complaints in elderly MDD patients and (2) assessing whether ECT has benefits for sleep, apart from ECT's antidepressant effect.

Clinical Points

- Complaints of insomnia are common in elderly patients, especially those with major depressive disorder (MDD). As insomnia is associated with adverse outcomes such as suicide, the aim of this study was to examine whether electroconvulsive therapy (ECT) had an anti-insomnia effect, apart from its effect on MDD, in a sample of elderly MDD patients receiving ECT and venlafaxine.
- Results showed that ECT, combined with venlafaxine, has a modest anti-insomnia effect that is linked to its antidepressant effect. Further, residual insomnia was common in this sample, despite otherwise successful ECT. Providers should consider targeted assessment and treatment of insomnia in elderly MDD patients receiving ECT.

METHODS

Patients

Full details of the methods of phase 1 of the PRIDE study can be found elsewhere.[17] Recruitment occurred at multiple sites from February 2009 to August 2014, and approval for the study was obtained from the institutional review board at each site. Participants provided their own, written, informed consent. The study is registered with ClinicalTrials.gov (identifier: NCT01028508). Briefly, participants were inpatients or outpatients, age ≥ 60 years, with a diagnosis of unipolar major depressive episode, as defined by *DSM-IV*,[18] and Hamilton Depression Rating Scale-24 item ($HDRS_{24}$) total score ≥ 21.[19] Exclusion criteria were bipolar disorder, schizoaffective disorder, dementia, substance abuse/dependence in last 6 months, active general medical or neurologic conditions that would have affected cognition or treatment response, contraindications to lithium or venlafaxine, or failure to respond to an adequate trial of lithium plus venlafaxine, or any ECT in the current episode.

Procedures

Medication washout and concomitant medications. Psychotropic medications were discontinued within 1 week of starting phase 1. Rescue medication for agitation, anxiety, or insomnia was limited to lorazepam up to 3 mg/d, or equivalent.

ECT. ECT procedures were standardized as follows: 3 times per week, standard RUL placement (D'Elia placement).[20] A dose titration procedure to determine seizure threshold was carried out at the initial session, and subsequent sessions were administered at 6 × seizure threshold. Seizure adequacy criterion was a motor seizure ≥ 15 s, and restimulation, if needed. If the $HDRS_{24}$ demonstrated < 25% drop from baseline by treatment 6, the stimulus dose was increased by 50%. If the $HDRS_{24}$ demonstrated < 25% drop from baseline by treatment 9, the stimulus dose was increased again by 50%.

Medication. Venlafaxine was started 1–5 days prior to ECT or up to 2 days after the first treatment at an initial dosage of 37.5 mg po, increased by 37.5 mg every 3 days or as tolerated, with a target dose of 225 mg/d.

Assessments

Diagnostic, demographic, and baseline clinical assessments. Baseline assessments were obtained for eligible patients who provided informed consent. Diagnosis was established using either the Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID-I)[21] (study years 1–2) or the Mini-International Neuropsychiatric Interview (MINI)[22] (years 3–6). The change to the MINI as a diagnostic instrument was made to minimize the baseline assessment time burden on patients.

Depression outcome measures. The $HDRS_{24}$ was administered 3 times per week, before each ECT. The primary outcomes in phase 1 were remission status and the longitudinal trajectory of $HDRS_{24}$ total scores. Patients were classified as remitters on the basis of (*a*) $HDRS_{24} \leq 10$ on 2 consecutive ratings and (*b*) $HDRS_{24}$ did not increase > 3 points on the second consecutive $HDRS_{24}$ or remained ≤ 6. The minimum number of ECT required for remission was 2, but there was no maximum. Nonremitters (*a*) did not reach

above remission criteria, (*b*) had at least 12 treatments, and (*c*) reached a plateau defined as no clinical improvement (< 3-point decrease in $HDRS_{24}$ after last 2 consecutive treatments). Participants were classified as dropouts if they left the study after fewer than 12 sessions of ECT and had not yet met criteria for remission.

Standardization and quality control for HDRS$_{24}$. Raters at all sites were trained to criteria using a set of videotaped clinical interviews. Ongoing consistency across sites and clinical raters was insured by having each rater rate a new set of videotapes annually for comparison with ratings of a "gold standard" rater.

Insomnia severity. Insomnia severity was measured by the sum of the 3 insomnia items of the $HDRS_{24}$, measuring initial, middle, and terminal insomnia, respectively. Each of the 3 items is scored 0–2, creating a range for their sum of 0–6. Notably, the use of any dose of any hypnotic medication resulted in a score of at least "1" on the middle insomnia item. The psychometric properties of the sum of the 3 insomnia items have been previously reported, showing that the sum loads on to a single factor when the $HDRS_{17}$ is subjected to a factor analysis,[23] and this measure is sensitive to treatment effects in samples of patients with MDD.[24] For the purpose of the present investigation, baseline insomnia sum scores of 0–3 were characterized as the "low-insomnia" group, while baseline sum scores 4–6 were characterized as the "high-insomnia" group. At the end of treatment, "remission of insomnia" was declared when the insomnia sum score became 0 and remained 0 through the end of the course of ECT. "Partial remission" was declared when the insomnia sum score became ≤ 2 and remained ≤ 2 for the remainder of the ECT course.

Suicidal ideation. The intensity of suicidal ideation was measured with the Beck Scale for Suicide Ideation (SSI)[25] and the $HDRS_{24}$ (item 3).

Non-insomnia depression symptoms, cognition, and medical comorbidity. Non-insomnia depressive symptoms were calculated by subtracting the insomnia sum scores from the $HDRS_{24}$, to create the HDRS-sleep. Global cognitive function was measured with the Mini Mental State Examination,[26] and severity of general medical conditions was measured with the Cumulative Illness Rating Scale—Geriatric (CIRS-G).[27]

Table 1. Demographic and Clinical Characteristics of Entire Sample at Baseline[a]

Characteristic	n	Value
Demographics		
Age, y	240	69.9 ± 7.6
Education, y	239	14.5 ± 3.3
Female	240	138 (57.5)
White	240	228 (95.0)
Hispanic	240	9 (3.8)
Baseline clinical characteristics		
Recurrent episode	240	210 (87.5)
Psychosis	240	28 (11.7)
Melancholia	239	141 (59.0)
Atypical	239	5 (2.1)
Psychiatric hospitalizations	229	2.4 ± 3.4
$HDRS_{24}$	240	31.2 ± 7.3
MMSE (raw score)	239	27.5 ± 2.4
CIRS-G total categories	240	4.6 ± 2.3
CIRS-G total score	237	8.6 ± 4.2
CIRS-G Severity Index	237	1.9 ± 0.5
CGI Severity	239	6.3 ± 0.9
Benzodiazepine use	240	153 (63.8)
SSRI antidepressant use	240	55 (22.9)
Family history of mental disorders		
Psychiatric illness	233	160 (68.7)
Mood disorder	231	143 (61.9)
Major depressive disorder	230	136 (59.1)
Bipolar disorder	231	33 (14.3)
Baseline insomnia and suicidality measures		
HDRS insomnia score[b]	240	3.4 ± 1.8
0–3 (low)	124	51.7
4–6 (high)	116	48.3
$HDRS_{24}$ item 3	240	1.6 ± 1.1
Beck SSI total score	202	4.6 ± 7.3
Beck SSI Index		
0	105	52.0
1–2	29	14.4
3–15	39	19.3
≥ 16	29	14.4
Adjusted $HDRS_{24}$ total score[c]	240	27.9 ± 6.6

[a]Data are represented as mean ± SD for continuous variables and n (%) for categorical.
[b]Insomnia score = sum of $HDRS_{24}$ items 4–6; range, 0–6.
[c]Adjusted $HDRS_{24}$ total score = $HDRS_{24}$ total score – insomnia score (sum items 4–6); range, 0–54.
Abbreviations: CGI = Clinical Global Impressions, CIRS-G = Cumulative Illness Rating Scale—Geriatric, $HDRS_{24}$ = Hamilton Depression Rating Scale-24 item, MMSE = Mini Mental State Examination, SSI = Scale for Suicide Ideation, SSRI = selective serotonin reuptake inhibitor.

Statistical Analysis

Frequency distributions for categorical variables and measures of central tendency (mean) and variability (standard deviation) were used to describe the baseline characteristics for the phase 1 study sample. For group comparisons, *t* test and χ^2 test were used for continuous and categorical variables, respectively. Generalized linear models were used for longitudinal analysis of insomnia scores with subjects as the unit of analysis to account for within-subject variability. To investigate the effect of non-insomnia symptoms on insomnia scores over time, observed (unadjusted) insomnia scores were compared with scores adjusted for non-insomnia symptoms ($HDRS_{24}$ total score minus insomnia sum score). For multiple comparisons of insomnia scores between observed and predicted means at each time point, *P* values were adjusted using Bonferroni correction. Change from baseline insomnia scores to the last observed insomnia score adjusted for change in non-insomnia symptoms were compared between high- and low-insomnia groups.

In order to detect whether ECT had an impact on sleep, apart from its antidepressant effect, we used percent change in HDRS-sleep from baseline to last measurement to determine patients for whom ECT worked (high percent change) and those for whom it did not work (no or minimal change). From the 2 upper and the 2 lower quintiles of HDRS-sleep, we included only patients with high insomnia scores at baseline to explore whether insomnia scores changed differently for those for whom ECT was effective compared to those for whom ECT was not effective. In addition, we compared the means for change from baseline to last

Table 2. Demographic and Clinical Characteristics by Insomnia Score (High/Low) at Baseline[a]

	Baseline Insomnia Score				
Characteristic	n	Low (0–3)	n	High (4–6)	*P* Value
Demographics					
Age, y	124	70.9 ± 7.9	116	68.8 ± 7.1	.027
Education, y	124	14.7 ± 3.4	115	14.4 ± 3.1	.412
Female	124	68 (54.8)	116	70 (60.3)	.389
White	124	121 (97.6)	116	107 (92.2)	.058
Hispanic	124	3 (2.4)	116	6 (5.2)	…
Baseline clinical characteristics					
Recurrent episode	124	103 (83.1)	116	107 (92.2)	.032
Psychosis	124	11 (8.9)	116	17 (14.7)	.163
Melancholia	123	64 (52.0)	116	77 (66.4)	.024
Atypical	123	3 (2.4)	116	2 (1.7)	…
Psychiatric hospitalizations	117	1.8 ± 1.7	112	3.0 ± 4.4	.006
MMSE (raw score)	123	27.5 ± 2.3	116	27.5 ± 2.4	.836
CIRS-G total categories	124	4.6 ± 2.3	116	4.6 ± 2.3	.797
CIRS-G total score	122	8.4 ± 4.3	115	8.8 ± 4.2	.415
CIRS-G Severity Index	122	1.9 ± 0.4	115	2.0 ± 0.5	.010
CGI Severity	123	6.1 ± 0.8	116	6.4 ± 0.9	.011
Benzodiazepine use	124	76 (61.3)	116	77 (66.4)	.412
SSRI antidepressant use	124	27 (21.8)	116	28 (24.1)	.663
Family history of mental disorders					
Psychiatric illness	120	82 (68.3)	113	78 (69.0)	.909
Mood disorder	119	77 (64.7)	112	66 (58.9)	.366
Major depressive disorder	119	74 (62.2)	111	62 (55.9)	.329
Bipolar disorder	119	17 (14.3)	112	16 (14.3)	1.000
Baseline insomnia, suicidality, and depression measures					
Insomnia score[b]	124	1.9 ± 1.0	116	5.0 ± 0.8	< .0001
$HDRS_{24}$ item 3	124	1.5 ± 1.1	116	1.7 ± 1.2	.165
Beck SSI total score	105	3.2 ± 5.6	97	6.2 ± 8.4	.004
Beck SSI Index					.012
0	61	58.1	44	45.4	
1–2	15	14.3	14	14.4	
3–15	22	21.0	17	17.5	
≥ 16	7	6.7	22	22.7	
Adjusted $HDRS_{24}$ total score[c]	124	26.2 ± 5.4	116	29.7 ± 7.3	< .0001

[a]Data are represented as mean ± SD for continuous variables and n (%) for categorical.
[b]Insomnia score = sum of $HDRS_{24}$ items 4–6; range, 0–6.
[c]Adjusted $HDRS_{24}$ total score = $HDRS_{24}$ total score – insomnia score (sum items 4–6); range, 0–54.
Abbreviations: CGI = Clinical Global Impressions, CIRS-G = Cumulative Illness Rating Scale—Geriatric, $HDRS_{24}$ = Hamilton Depression Rating Scale-24 item, MMSE = Mini Mental State Examination, SSI = Scale for Suicide Ideation, SSRI = selective serotonin reuptake inhibitor.

observed insomnia score in the high (≥ 50% improvement) versus low (< 25% improvement groups based on change in non-insomnia symptoms (HDRS-sleep). Significance level α was maintained at 0.05. All analyses were conducted using SAS 9.4 (Cary, NC).

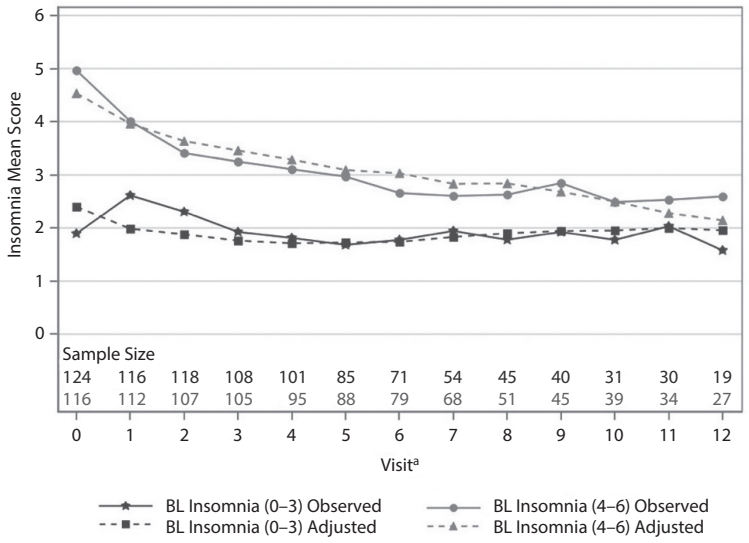
RESULTS

Two hundred forty patients entered phase 1 and contributed to the present report, including 148 remitters, 24 nonremitters, and 68 dropouts. The treatment response of the dropouts was very similar to the nonremitters.[17] The sample was mostly female and white, with a high baseline degree of depressive symptom severity, well-preserved global cognitive function, and a moderate degree of overall insomnia severity (Table 1). Most patients were taking a benzodiazepine at the time of being evaluated for participation (63.8%), but summed-insomnia scores were not significantly different in those who did versus those who did not take a benzodiazepine at baseline. Only 22.9% were taking an SSRI. Patients were nearly evenly split between the high-insomnia (48.3%) and low-insomnia groups (51.7%). In the low-insomnia group at study entry, 3 of 124 patients were taking a sleep aid, but by the end of phase 1, 24 patients (19.4%) were taking a sleep aid. Similarly, in the high-insomnia group at study entry, 10 of 116 patients were taking a sleep aid, but by the end of phase 1, 27 patients (23.2%) were taking a sleep aid. The high-insomnia group was distinguished by younger age, history of greater severity of general medical illness, more psychiatric hospitalizations, greater severity of suicidal ideation intensity, and greater severity of non-insomnia symptoms of depression (Table 2).

Among the patients in the high-insomnia group, only 12.4% met our definition of full remission from insomnia, but 59.3% reached partial remission. The average number of ECT sessions required to reach full remission of insomnia in the high-insomnia group was 6.1 ± 2.2, while the number of sessions required to reach partial remission was 5.1 ± 3.4. The mean change in the insomnia score for the high-insomnia group was 2.7 ($P < .0001$). Insomnia scores showed very little change across the course of ECT in the low-insomnia group (Figure 1). When insomnia scores were adjusted for non-insomnia depression symptoms, comparison with the observed scores showed little change; all *P* values were > .05 except at baseline and visit 1 in the low-insomnia group. Mean insomnia scores, adjusted for change in non-insomnia depression symptoms, decreased by 0.50 for the low-insomnia group and by 2.56 for the high-insomnia group ($P = .002$ and $P \leq .0001$, respectively). The change in scores for the high-insomnia group was statistically significantly greater than for the low-insomnia group ($P < .0001$). Of the 124 patients in the low-insomnia group, 112 remained in the low-insomnia group, but 12 (9.6%) transitioned from the low-insomnia group to the high-insomnia group by the end of the ECT course.

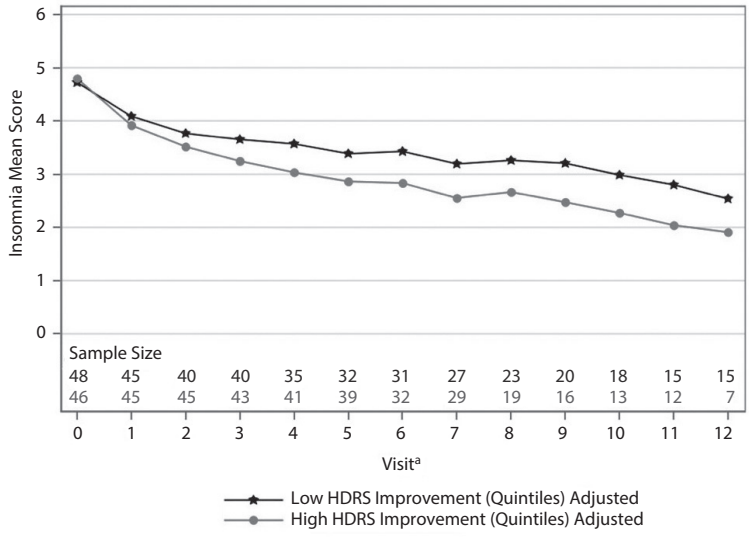
We found that insomnia scores decreased regardless of whether ECT had a strong versus weak antidepressant effect ($P > .5$), and the change was statistically significant over time ($P < .05$) (Figure 2). Further, we found that a "low" amount of improvement on the sum of the HDRS non-insomnia items (HDRS-sleep) was accompanied by a "low" amount of improvement in insomnia scores (mean ± SD change of −1.6 ± 1.2, $P < .0001$), while a "high" amount of improvement on the sum of the HDRS non-insomnia items was accompanied by a "higher" amount of improvement in insomnia scores (change of −3.1 ± 1.6, $P < .0001$). After further adjusting for age, recurrent episode, melancholia, psychiatric hospitalizations, CIRS-G severity, and CGI Severity, no statistically significant relationship with insomnia scores was observed (all $P > .1$).

Figure 1. Changes in Insomnia Scores According to High Versus Low Baseline Insomnia Score



[a]Visits 13–17 were excluded due to small sample sizes.
Abbreviation: BL = baseline.

Figure 2. Changes in Insomnia Scores According to High Versus Low Change in Overall Depression Severity



[a]Visits 13–17 were excluded due to small sample sizes.
Abbreviation: HDRS = Hamilton Depression Rating Scale.

DISCUSSION

The main findings of the study were that only 12.4% of the high-insomnia sample experienced full remission of insomnia symptoms by the end of ECT, but 59.3% met our definition of partial remission for insomnia symptoms by the end of the ECT course. Remission and/or partial remission for insomnia symptoms occurred relatively early—after 5 or 6 ECT sessions. The adjusted mean improvement in insomnia symptoms across both the low-insomnia group and the high-insomnia group could be partially explained by the increased use of sleep aids across the course of ECT in both groups. The increased prescription of sleep aids during the course of ECT is noteworthy and may be related to the fact that venlafaxine is known to disrupt polysomnographic sleep.[28]

The high-insomnia group showed a greater degree of improvement in insomnia symptoms compared with the low-insomnia group. However, when changes in the insomnia scores were contrasted between those with strong antidepressant response to ECT versus those with weaker antidepressant response to ECT, we found that the degree of change in insomnia scores was related to overall change in non-insomnia symptoms, suggesting that improvement in insomnia symptoms is part of the antidepressant effect of ECT. This finding is similar to what has been reported regarding the impact of rTMS and tDCS on insomnia symptoms in MDD,[16,29] suggesting that therapeutic brain stimulation for MDD exerts its effect differently from antidepressant medication.

The overall sample exhibited a high degree of depressive symptoms at baseline, with 48% reporting some degree of suicidal ideation and 48% reporting a high degree of insomnia symptoms. The high-insomnia group had a greater proportion of recurrent episodes of MDD, more psychiatric hospitalizations, greater suicidal ideation, and a greater intensity of non-insomnia MDD symptoms. The association between insomnia and suicide has been reported many times,[30] including in samples of elderly MDD patients, but our report is the first to confirm this association in an ECT sample. The use of sleep aids increased over the course of study participation, with similar rates of use in the low-insomnia and high-insomnia groups.

This report is subject to a number of limitations. First, the findings are limited to elderly adults with MDD who are taking concomitant venlafaxine and low dose, as-needed lorazepam in the context of RUL UBP ECT. Other forms of ECT, higher doses of benzodiazepines, or different concomitant antidepressants could have produced different results. As mentioned, venlafaxine has been reported to have some sleep-disturbing effects, and this could have affected the results of this study.[28] However, the combination of antidepressant medications with ECT is more effective than ECT alone in the treatment of MDD, and hence the results of this study are likely to be of relevance as more practitioners adopt the combination of antidepressants and ECT.[31] Second, as this was an open-label study with no comparison group, the improvement in the high-insomnia group could be related

to regression to the mean, and the lack of change in the low-insomnia group may be explained by a floor effect with the $HDRS_{24}$ insomnia sum score. Third, using only the $HDRS_{24}$ insomnia sum score may have produced different results from other insomnia assessment methods, including patient-report sleep diaries, the Insomnia Severity Index,[32] actigraphy, or polysomnography. Fourth, as polysomnography was not conducted, it is unknown what portion of the sample was experiencing primary sleep disorders, such as sleep apnea or periodic limb movement disorder, both of which show increasing prevalence with age. In earlier work, we have reported a rate of 16% of unsuspected primary sleep disorders in a sample of young and middle-aged MDD patients.[33] It is likely that the rate of primary sleep disorders was higher in the present sample, and it is unknown whether the presence of primary sleep disorders might affect clinical outcomes in ECT.

The findings of this report provide several clinical lessons: first, that the intensity of insomnia symptoms is positively associated with the intensity of suicidal ideation, implying that ECT practitioners should view the progression or remission of insomnia as a potential indirect indicator of suicidal ideation. Second, most elderly MDD patients can expect improvement in insomnia during ECT, especially those with the most extreme symptoms, but most will also be left with some residual insomnia symptoms. Indeed, in this study, the rate of prescription of sleep aids actually increased over the course of ECT, and ECT practitioners should be alert to the need to assess and treat incompletely resolved insomnia complaints in elderly depressed patients. Third, improvement in insomnia can usually be expected early, and it is unclear that lengthening the course of ECT will lead to further improvement in residual insomnia.

The persistence of insomnia symptoms in otherwise successfully treated patients could result in residual deficits in quality of life or greater risk of relapse; these questions will be examined in future reports. If residual insomnia symptoms represent regression to population means (since insomnia complaints are common in population studies of the elderly) and are benign, they might require no special treatment; alternatively, if they confer some ongoing risk of relapse or poor quality of life, they may require unique treatment strategies. Disrupted sleep is a major concern for elderly patients with MDD,[4] and these data provide evidence that RUL-UBP ECT, combined with venlafaxine, can result in some relief from insomnia symptoms.

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