Letters to the Editor

Suicide After Bright Light Treatment in Seasonal Affective Disorder: A Case Report

Sir: In a recent article, Praschak-Rieder et al.[1] described 3 cases of suicide in patients with seasonal affective disorder (SAD) who were treated with bright light therapy. The authors emphasized the importance of collecting data on suicide and SAD. Seasonal affective disorder is a syndrome characterized by the recurrence of depressive episodes, which in most cases begin in fall or winter and remit in the following spring or summer.[2] SAD is a variant of bipolar disorder or recurrent major depressive disorder.[3] Moreover, most patients suffer from atypical symptoms such as hypersomnia, hyperphagia, weight gain, fatigue, and carbohydrate craving. Bright light therapy is a widely accepted and effective treatment for SAD.[4] We present 1 patient who committed suicide after 1 week of treatment with bright light therapy.

Case report. Ms. A, a 65-year-old administrator, sought treatment in 1993 as an outpatient after 5 years of suffering from depressive moods. Just before, and against recommendation, she had left a clinic for affective disorders, where she was admitted. She was diagnosed with bipolar disorder in a depressive episode, after having undergone several hypomanic periods. Ms. A was described as compulsive, with problems in dealing with changes. At that time, Ms. A had 4 adult children and lived a moderately active social and religious life with her husband. Family history revealed that Ms. A's sister committed suicide in 1982 after similar complaints. In the first visit at the outpatient clinic, the rigidity of Ms. A was remarkable: she "just had to go on." After an initial recovery at the end of 1993, Ms. A relapsed into a severe depressive episode. Fluoxetine treatment was started. Because of nausea, dizziness, and an antipathy toward medication, she stopped taking the tablets. Meanwhile, her cataract deteriorated. Quite suddenly, her depressive mood turned into a hypomanic episode. Again she refused medication (lithium). After a depressive episode that started in mid-1994, Ms. A decided in July 1995 to end the treatment in our clinic and returned to her general practitioner. Her situation seemed to be stabilized.

In December 1995, Ms. A presented again with depression. She was gloomy, spiritless, tired, shrank from everything, slept very badly, had no appetite, and had lost 5 kg of weight. She was strongly inclined to stay in bed all day and felt worthless, insufficient, and guilty. Suicidal ideas or gestures were denied; those thoughts were inconceivable to her as a religious person. Nevertheless, Ms. A was rated on the SAD version of the Structured Interview Guide for the Hamilton Rating Scale for Depression (SIGH-SAD)[5] on the "suicide" item (range, 0 to 4), with a score of 3, indicating that the patient had thoughts of taking her own life, possibly including working out a plan, having rehearsed a plan, or having made minor gestures.

Because of presumable seasonality of her depressive complaints and her aversion to taking medication, bright light therapy was started. Ms. A was, for 5 consecutive days, exposed for 30 minutes to 10,000 lux of bright light beginning at 8:00 a.m. In case of severe insomnia, temazepam, 20 mg, in the evening, was allowed. After 3 days of bright light therapy, a small improvement was found, especially an increased drive. Ms. A's score on the SIGH-SAD rating decreased (score before treatment = 37, score after treatment = 21). The item concerning suicidal symptoms decreased to 1: the patient has felt life not worth living—a persistent thought, which recurs. In the morning 5 days after bright light therapy began, Ms. A committed suicide.

Primary, bright light therapy seems to be an activating and resynchronizing treatment. As patients become activated while still dysphoric, suicide attempts are likely to occur. As was the case in the earlier report on suicide in bright light therapy,[1] no suicidal thoughts or severe alterations in mood were found before the treatment started. Also in this case, no suicidal behavior was reported during previous depressive episodes.

In conclusion, in accordance with the report by Praschak-Rieder et al.,[1] suicidal features in SAD patients treated with bright light therapy are rare, but not negligible. Bright light therapy is a powerful treatment that can, in some cases, bear the risk of suicidal ideation. Patients who have had hypomania especially seem to run the risk of developing suicidal features, but further research is recommended.

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Progression of Abnormal Involuntary Movements During Risperidone Treatment

Sir: The propensity for risperidone to cause tardive dyskinesia is unknown at this time. A high D_2 receptor affinity,[1] dose-related EPS,[2] and potential to mask tardive dyskinesia are properties that risperidone shares with typical antipsychotics. This similarity to typical antipsychotics suggests a potential to cause tardive dyskinesia. The effect of a high degree of seroto-

nin blockade by risperidone may reduce net dopamine blockade and may imply a reduced risk of tardive dyskinesia. However, accumulating evidence supports the development of tardive dyskinesia in susceptible risperidone-treated individuals. We report 2 cases of risperidone-influenced tardive dyskinesia.

Case 1. Ms. A, a 52-year-old divorced white woman with a diagnosis of psychotic disorder NOS which began when she was 49 years of age, received thiothixene for 5 months and then stopped taking her medication. Psychotic symptoms returned after 1 month, at which time she presented with a dystonic torticollis. Ms. A was placed on treatment with risperidone, 6 mg/day, and has remained medication compliant. She was first seen in our research clinic 7 months later with stable and mild involuntary movements of the lips, jaw, knees, and fingers and persistence of a severe 70°-left-facing dystonic neck posture. After 18 months on risperidone treatment, she suddenly developed severe choreic tongue movements, and her lip and jaw movements suddenly worsened and persisted without further worsening for a year. Results of laboratory tests, CT scan, and genetic testing for triplet repeat disorders including Huntington's disease were within the normal range. Psychiatric status required maintenance of risperidone at 6 mg/day.

Case 2. Ms. B, a 42-year-old white woman with a diagnosis of schizoaffective disorder, was diagnosed with tardive dyskinesia when she was 36 years of age. She was treated briefly with neuroleptic medications when she was 17 years of age and has had a total neuroleptic exposure of approximately 8 years. Her involuntary muscle movements included head bobbing, anterograde dystonic neck postures, and mild tongue, lip, and finger chorea. She discontinued all medications on her own for over a year, and the head and neck dystonia disappeared. Approximately 18 months ago, she was placed on risperidone. The dosage was titrated to 6 mg/day across several days. After 7 days, the head and neck dystonia returned and persisted during a 6-week trial of risperidone. Subsequent treatment with thioridazine, 800 mg/day (the only medication the patient would agree to take), for 1 year has resulted in persistent dyskinetic movements.

Associations between risperidone and tardive dyskinesia, both new and reemergent, have been reported.[3–9] All of these cases, including those in this report, have a history of previous exposure to typical antipsychotic agents. Current literature includes case reports of dyskinetic movements that appeared after 1 to 12 months of risperidone treatment. The cases of the 2 women reported here represent a different phenomenon. In these 2 cases, there is a diagnosis of tardive dystonia. While the dystonic syndromes first appeared during typical neuroleptic treatment, they did not improve, but worsened, with risperidone therapy. In the first case, a dramatic worsening of the choreic component occurred after 18 months of risperidone treatment. In the second case, an obvious and debilitating dystonia that had previously resolved while the patient was medication-free reappeared within 1 week of risperidone exposure. The worsening of these dystonic syndromes with risperidone cannot be attributed to withdrawal phenomena since in both cases risperidone was started after the patients had been off neuroleptic medication for a month or more. Further, close clinical monitoring, reports by live-in family members, and stable psychiatric symptoms all strongly indicate medication compliance.

Risperidone can mask tardive dyskinetic movements.[10–12] Whether masking by risperidone will persist without subsequent worsening of movements as seen with typical antipsychotics is not known. Not only were abnormal involuntary movements not masked, but our 2 patients with neck dystonia experienced a worsening of the dystonic syndromes over time with exposure to risperidone, which indirectly implicates risperidone as a causal agent in tardive dyskinesia. Patients with tardive dystonia may have an increased sensitivity for risperidone to exacerbate abnormal involuntary movements.

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Recurrent Mood Shifts of Premenstrual Dysphoric Disorder Can Be Mistaken for Rapid-Cycling Bipolar II Disorder

Sir: Some authors have postulated that the diagnosis of bipolar II disorder is underdiagnosed among outpatients attending mood disorder clinics.[1,2] This diagnosis has been observed to occur more frequently in women in some,[3] although not all,[4] reports. Rapid-cycling variants of bipolar disorder occur approximately 3 to 5 times more frequently in women than in men.[5] We present a case in which the diagnosis of rapid-cycling bipolar II disorder was misapplied for a patient whose recurrent mood cycling was found to result from premenstrual dysphoric disorder (PMDD).

Case report. Ms. A, a 37-year-old woman, presented with recurrent episodes of depressed mood accompanied by crying spells, anhedonia, lethargy, hypersomnia, and increased appetite. These episodes typically lasted 10 to 14 days and were followed by an elevation of mood, an increase in alertness, self-esteem, activity, and productivity, and decreased need for sleep relative to the previous 2 weeks. These expansive phases tended to last approximately 1 week.

Ms. A had experienced these mood swings since her early 20s, but they had worsened in her early to mid-30s. Her family history was notable for bipolar disorder in her paternal uncle, responsive to lithium monotherapy. At 35 years of age, Ms. A experienced a major depressive episode for which she was hospitalized. At that time, the diagnosis of rapid-cycling bipolar II disorder was given, and lithium, 600 mg/day, was initiated. As this treatment produced intolerable gastrointestinal side effects, Ms. A was switched to valproate within 2 weeks. The valproate dose reached 1000 mg/day with no improvement of her mood symptoms. Bupropion, 225 mg daily, was added 3 weeks later and led to an unsustained improvement in her mood. As she continued to suffer frequent severe exacerbations over the course of the following year, she presented to our clinic for a consultation.

A careful assessment of the relationship of Ms. A's symptoms to her menstrual cycle revealed an association between the depressions and the luteal phase of her cycle (i.e., the 2 weeks preceding the onset of menses). The improvement in mood and functioning occurred with the onset of menstruation. Daily charting over the following 2 months confirmed these observations. Initiation of sertraline, 100 mg/day, accompanied by a tapering and discontinuation of valproate and bupropion, produced an excellent resolution of Ms. A's symptoms within 4 weeks, with no escalation into hypomania over an 18-month follow-up.

Following the onset of menses in women with PMDD, the distinct improvement in mood, energy level, and productivity and the resolution of hypersomnia and lethargy may be mistaken for a hypomanic episode. Patients may indeed present with symptoms that appear to meet the criteria for hypomania, including enhanced self-esteem, reduced need for sleep (relative to the premenstrual phase), and increased goal-directed activity. The mood switch that follows the onset of menses may be very sudden, occurring, in our experience, in as few as 2 to 4 hours. Dramatic mood changes over similar time frames have also been observed in patients with bipolar disorder,[6] and insights into the etiology of premenstrual dysphoric disorder may help elucidate the "switch" mechanism of bipolar disorder.[5] Ms. A's major depressive episode at 35 years of age contributed to her diagnosis of bipolar II disorder. It is important to keep in mind that the lifetime prevalence of major depressive episodes is substantially higher in women with PMDD[7] compared with the general population of women of reproductive age. Given Ms. A's positive family history for bipolar disorder, she would benefit from close monitoring for a switch to mania or hypomania while taking antidepressant medication. However, during our 18-month follow-up, what appeared to be hypomania instead seems to reflect increased well-being following the resolution of premenstrual dysphoric symptoms.

This case illustrates the importance of assessing the relation of symptoms across the menstrual cycle for menstruating women who present with recurrent episodes of affective instability.[8] Preferably, patients should chart their symptoms prospectively across at least 2 cycles. Women in their late 20s through mid-30s appear to be at highest risk for distressing symptoms of PMDD.[9] Effective treatment strategies are now readily available,[10] and particular success has been noted with standard doses of serotonin reuptake inhibitors.[11–14] While bupropion has been observed to produce mood-stabilizing effects in rapid-cycling bipolar II patients,[15] no systematic data exist on its use for PMDD. In our patient, it was not an effective intervention. Low-dose valproate also has been reported as a treatment for patients with rapid-cycling bipolar II disorder, but is of little benefit for PMDD.[16] Similarly, lithium does not appear to be helpful for this condition.[17] Our patient's case suggests that care should be taken in making the diagnosis of bipolar II disorder in women of reproductive age who have monthly mood cycling.

This case also underscores the importance of detailed questioning using objective standards to assess whether a patient's symptoms (e.g., elevated mood, decreased need for sleep, increased productivity) may be within the normal range. If, after careful questioning, the patient continues to meet criteria for hypomania, her diagnosis may be rapid-cycling bipolar disorder entrained to the menstrual cycle.

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Gabapentin Augmentation for Fluoxetine-Treated Patients With Obsessive-Compulsive Disorder

Sir: Serotonin reuptake inhibitors (SRIs) are the most effective pharmacologic treatment for obsessive-compulsive disor-

der (OCD). However, few patients have a complete response to SRIs. Because augmentation strategies are needed, we initiated a pilot study of gabapentin in patients with OCD who had a partial or incomplete response to SRI treatment. Gabapentin is a neutral gamma-aminobutyric acid (GABA) analog, which, in general, has few side effects, and, overall, appears to be a safe, easy to titrate, well-tolerated drug in patients with seizure disorders.[1] To our knowledge, there have been no prior studies of the possible efficacy of adding gabapentin to ongoing SRI treatment in patients with OCD who have a partial response to SRI treatment.

Five OCD patients, all partial responders to fluoxetine monotherapy, were treated openly with the addition of gabapentin for 6 weeks. All met DSM-IV criteria for OCD, and 4 met criteria for lifetime comorbid mood and/or anxiety disorders. Fluoxetine had been administered at a stabilized mean dose of 68 mg/day (range, 30–100 mg/day) for more than 12 weeks. Gabapentin was initiated at 900 mg/day (given 3 times per day) and titrated upward to a maximum of 3600 mg/day in divided doses. The mean dose of adjunctive gabapentin was 1260 mg/day by week 2 (range, 900–1800 mg t.i.d.), and 2520 mg/day by week 6 (range, 900–3600 mg t.i.d.). Response to gabapentin augmentation after 6 weeks was determined by clinical evaluations.

All patients completed the 6-week trial. The fluoxetine dose was not changed for any patient during the study. Overall, all of the patients in our series reported marked subjective improvement in their anxiety, obsessive-compulsive symptoms, sleep, and mood within 2 weeks of initiating gabapentin. These observations were evaluated by our clinical staff and supported by rating data when available. The gabapentin/fluoxetine combination was generally well tolerated during the 6-week period, although 1 patient experienced transient gastrointestinal side effects (we maintained the gabapentin at 900 mg/day and decided against increasing the dose). Gabapentin augmentation also markedly reduced the frequency of migraine headaches in 1 other patient (from 3 per week to 2 in 6 weeks).

This pilot study has a number of limitations, including the following: (1) all patients received gabapentin openly; (2) gabapentin augmentation was evaluated in a small number of OCD patients who were partial responders to fluoxetine treatment; (3) patients did not receive a standard dose of gabapentin; (4) patients had comorbid anxiety and/or depressive psychiatric diagnoses, and it is unclear how these conditions might have affected treatment response. For example, gabapentin may have improved sleep via a direct hypnotic effect, or it may have reversed SRI-related sleep disturbance. All patients reported decreased anxiety and mood improvement by the end of the trial. A larger OCD patient sample with and without comorbid symptoms is required to adequately evaluate the specific effect of gabapentin. Although there are reports of gabapentin-induced hypomania in some patients with epilepsy,[2] we observed no evidence of hypomanic symptoms in our patients. It is unclear if the antimigraine effect noted by 1 patient was a direct effect of gabapentin on mood stabilization, a direct analgesic effect, a direct anxiolytic effect, or an effect on sleep.[3–5] Very preliminary clinical data suggest that gabapentin may be effective in psychotic and bipolar patients with comorbid anxiety disorders, including comorbid OCD (L. Beauclair, M.D., oral communication, September 1997). This is consistent with preclinical data suggesting that treatments that augment 5-HT and GABAergic neurotransmission may have additive effects in the cerebral cortex.[6]

To our knowledge, this is the first report of the possible efficacy of gabapentin augmentation in OCD patients with incomplete treatment response to SRI monotherapy. Gabapentin augmentation was well tolerated in this group of OCD patients. On the basis of these encouraging preliminary results, we are currently conducting a randomized, placebo-controlled trial to evaluate the possible efficacy of gabapentin augmentation in OCD patients partially responsive to SRI monotherapy.

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