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Brief report

Rate of switch from depression into mania after therapeutic sleep deprivation in bipolar depression

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Abstract

Sleep deprivation is a potentially useful non-pharmacological treatment for depression. A relationship between sleep loss and the onset of mania has been reported, so it is possible that a switch from depression into mania after sleep deprivation might be expected in bipolar depressed patients who are treated with sleep deprivation. In a sample of 206 bipolar depressed treated with three cycles of sleep deprivation, alone or in combination with heterogeneous medications, we observed a 4.85% switch rate into mania and a 5.83% switch rate into hypomania. These percentages are comparable to those observed with antidepressant drug treatments. © 1999 Elsevier Science Ireland Ltd. All rights reserved.

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1. Introduction

The potential usefulness of sleep deprivation in the treatment of bipolar depressed patients has been suggested (American Psychiatric Associa-

tion, 1995). A common risk when treating bipolar depression is the occurrence of a switch from depression into mania, and in bipolar patients a close relationship has been observed between sleep loss and the onset of mania (e.g. Wehr, 1992; Barbini et al., 1996). Few data are available, however, on the rate of switch from depression into mania after therapeutic sleep deprivation in bipolar patients.

In their review on the effects of sleep depriva-

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tion in depression, Wu and Bunney (1990) cited 10 studies that had reported switches from depression into hypomania or mania, with a 30% rate of switch after therapeutic sleep deprivation in bipolar depressed patients. Years of publication of the 10 studies ranged from 1974 to 1982. In more recent studies on the effects of sleep deprivation, switches into mania disappear except for rapid-cycling bipolar patients. Wehr et al. (1982) report the spontaneous occurrence of sleep deprivation before the switch into a manic phase in 13/15 rapid-cycling bipolar patients, while therapeutic sleep deprivation caused 7/9 drug-free depressed rapid-cycling bipolar patients to switch into mania. More recently, the relationship between sleep loss and the onset of mania in patients with rapid-cycling bipolar disorder has been well established (Leibenluft et al., 1996).

A major problem in the literature about sleep deprivation is the diagnostic heterogeneity of the samples, because many of the study groups included patients with endogenous, reactive, unipolar, bipolar, secondary, and schizoaffective depression (Leibenluft and Wehr, 1992). Therefore, on the basis of the available data, the rate of switch from depression into mania that should be expected after sleep deprivation in non-rapid-cycling bipolar patients cannot be reliably estimated. In the present study, we report the rate of switches from depression into mania observed in a sample of 206 bipolar depressed inpatients treated with therapeutic sleep deprivation.

2. Methods

During the years 1994–1998, 206 bipolar depressed inpatients were treated with serial repetition of total sleep deprivation (TSD) at our research center for mood disorders. On the basis of DSM-III-R and DSM-IV criteria, patients were diagnosed as bipolar disorder, depressive episode without psychotic features. Patients were treated according to experimental protocols aimed at sustaining the transient antidepressant effects of TSD (e.g. Benedetti et al., 1996, 1997, 1998; Barbini et al., 1998; Colombo et al., 1998; Smeraldi et al., 1998, 1999). Inclusion criteria were as follows:

absence of other Axis-I diagnoses; absence of mental retardation (Axis II); absence of pregnancy, history of epilepsy, major medical and neurological disorders; no treatment with long-acting neuroleptic drugs in the 6 months preceding admission; no treatment with neuroleptics or irreversible monoamine oxidase inhibitors in the last month before admission; absence of a history of drug or alcohol dependency or abuse within the last 6 months; and, for lithium-treated patients, no period off lithium during the last 6 months. All the experimental protocols were approved by the ethical committee of our hospital, and written informed consent was obtained from each patient.

In each study, patients were randomized to receive three TSD cycles, composed of a night of TSD followed by a recovery night, alone or in combination with several medications (see Table 1). For the purpose of the present work, mania and hypomania were defined according to DSM-IV criteria with the exception of the temporal criterion for mania.

3. Results

During the TSD treatment, we observed 10 (4.85%) switches into a manic phase, and 12 (5.83%) switches into a hypomanic phase. The rate of switches was not associated with current medication status (switches into mania: $\chi^2 = 5.33$, d.f. = 4, $P = 0.255$; switches into hypomania: $\chi^2 = 1.45$, d.f. = 4, $P = 0.836$; all switches together: $\chi^2 = 5.20$, d.f. = 4, $P = 0.268$).

Since manic symptoms appeared during hospitalization, patients were immediately administered an antimanic treatment. Restoration of night-time sleep with i.v. benzodiazepines (diazepam, 10 mg, or lorazepam, 4–8 mg) resulted in a rapid resolution of manic symptomatology in 3/10 patients, who returned to euthymic conditions in 3–5 days; 3/10 patients had a manic phase which resolved within 1 month with mood stabilizers (lithium salts and carbamazepine) and benzodiazepines (flurazepam up to 45 mg/day); 4/10 patients developed a manic episode with psychotic features and needed neuroleptic medi-

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