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Key Words: sleep apnea–hypopnea syndrome, bipolar disorder, continuous positive airways pressure

Continuous Positive Airways Pressure Treatment in a Patient with Sleep Apnea—Hypopnea Syndrome and Coexisting Bipolar Disorder

By Georgia Trakada, MD, Pashalis Steiropoulos, MD, and Demosthenes Bouros, MD, FCCP

ABSTRACT *~ We present a case of a 43-year-old woman with bipolar disorder who was diagnosed with sleep apnea–hypopnea syndrome (SAHS). After 10 days of treatment with continuous positive airways pressure (CPAP) during sleep, the patient presented a mood switch, from depression to mania and was admitted to the hospital. Withdrawal of CPAP and appropriate therapy controlled the episode.* Psychopharmacology Bulletin. *2008;41(2):89-92.*

CASE HISTORY

Sleep apnea–hypopnea syndrome (SAHS) is a chronic condition characterized by repetitive collapse of the upper airway leading to pathophysiologic changes that affect the neuropsychological and cardiovascular systems.[1] The neuropsychological deficits are mainly related to the severity of hypoxaemia or to sleep deprivation and chronic sleep fragmentation. Several reports have demonstrated that SAHS is associated with neuropsychological and functional deficits, including excessive daytime sleepiness (EDS), depression, and decreased quality of life (QOL). The first line of therapy is continuous positive airways pressure (CPAP), which improves neuropsychological functions and QOL by alleviating depression. Switch from depression to mania by CPAP treatment in a patient with SAHS and coexisting bipolar disorder, to our knowledge, has never previously been reported.

SYMPTOMATOLOGY

A 43-year-old woman was referred to our outpatient Sleep Unit because of daytime sleepiness and snoring. The patient was diagnosed with bipolar disorder 15 years ago and was currently under treatment with quetiapine, fluvoxamine maleate, and alprazolam for a depressive episode.

Dr. Trakada, Dr. Steiropoulos, and Dr. Bouros are affiliated with Department of Pneumonology, Democritus University of Thrace Medical School, Alexandroupolis, Greece.

To whom correspondence should be addressed: Georgia Trakada, MD, Department of Pneumonology, Democritus University of Thrace Medical School, Alexandroupolis 68100, Greece; E-mail: gtrakada@hotmail.com

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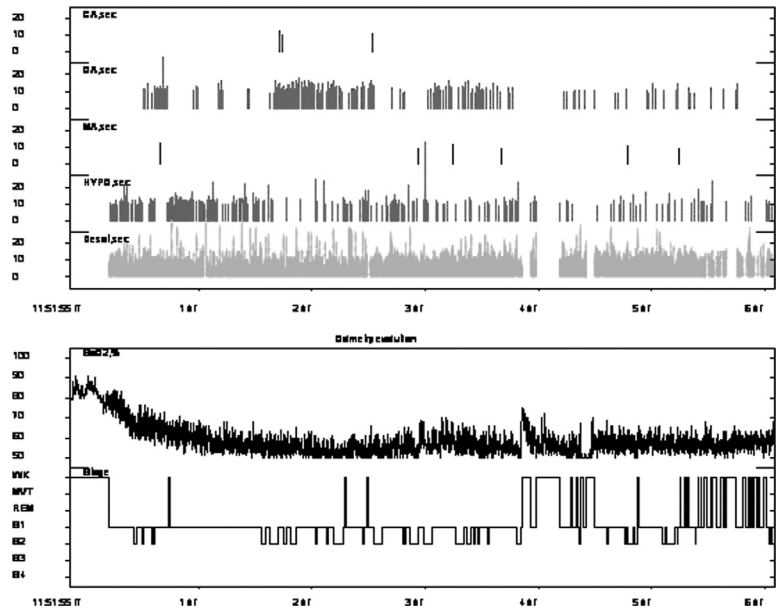
On physical examination, the patient was a drowsy obese woman. Her weight was 120 kg with a height of 156 cm and a body mass index (BMI) of 47.7 kg/m^2. Lung auscultation revealed mild wheezing, whereas heart auscultation was normal. Her Epworth Sleepiness Scale was 14.

DIAGNOSIS

The patient underwent standard full-night polysomnography performed in a quite, partially soundproof room, with stable humidity and temperature. All parameters were recorded simultaneously on polygraphic records (Alice-4, Respironics, Murrysville, PA). Polysomnographic findings were as follows: time in bed 373 min, sleep latency 20.5 min, total sleep time (TST) 292 min, sleep efficiency (SE) 78.28%; stage 1, 78.1% (241 min), and stage 2, 21.9% (64 min) (Figure 1). No slow wave sleep (SWS) or rapid eye movement sleep (REM) were detected. The absolute number of respiratory events during sleep was 496 (3 central, 242 obstructive and 6 mixed apneas, and 245 hypopneas) and the apnea–hypopnea index (AHI) was 101.9, with a mean SaO_2 59% and a minimum SaO_2 44%. CPAP titration was carried out on a second night, normalizing the AHI at a pressure of 8 cm H_2O. Additional oxygen supply (3 L/min) was adjusted to CPAP because of profound hypoxemia.

FIGURE 1

POLYSOMNOGRAPHIC DATA OF THE PATIENT



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TREATMENT

CPAP treatment was prescribed at home. Good adaptation and compliance were reported by her relatives, with progressive disappearance of daytime and nighttime symptoms. However, after 10 days of treatment, the patient went into an aggressive mood with incoherence and agitation, and attempted to hit her relatives. She was finally taken to hospital by the police and admitted with an acute maniac episode. Withdrawal of CPAP for a week and appropriate treatment controlled the episode. The patient started a new CPAP treatment trial with fewer hours of use, 1 to 4, every night and close monitoring.

DISCUSSION

The prevalence of breathing-related sleep disorders in psychiatric population has scarcely been studied. Ohayon[2] reported that the odds of having a DSM-IV breathing-related sleep disorders diagnosis was 5.26 for individuals with a major depressive disorder diagnosis, even after controlling obesity and hypertension. Furthermore, possible complications of CPAP treatment in this population have scarcely been reported.[3]

During depression, bipolar patients suffer insomnia or hypersomnia, and during mania, there is often a reduced need for sleep. Sleep disturbance persists even when patients with bipolar disorder are between episodes. Prolonged sleep latency, reduced sleep efficiency, increased number and duration of awakenings, reduced delta sleep, shortened REM sleep latency, and increased REM density are common polysomnographic features in patients with bipolar disorder.[4] SAHS causes chronic sleep deprivation, and especially REM sleep deprivation, due to repeated arousals because of obstructed breathing.[1] When treated with CPAP, a REM rebound occurs during the first days of treatment. Furthermore, fluvoxamine suppresses REM sleep, possibly by an inhibitory effect on melatonin degradation.[5]

Our patient had the sleep disturbances of bipolar disorder and the pharmacologic effect of fluvoxamine coexisting with chronic sleep deprivation induced by SAHS. Treatment with CPAP possibly "disorganized" the equilibrium between the patient's cholinergic and dopaminergic systems by increasing dopaminergic activity or stimulating GABAergic pathways, which modulate atonia versus cortical activation and favoured the episode of mania. Also, SAHS possibly contributed to the psychomotor slowing, daytime drowsiness, and memory and concentration difficulties first observed in this patient and nonresponding to the treatment with fluvoxamine.

Patients with bipolar disorders should be evaluated for breathing-related sleep disorders because SAHS may complicate the severity of

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the psychiatric disease. Careful follow-up is needed during treatment with CPAP to prevent the possible relapse of manic symptoms. ✤

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