Journal of Affective Disorders 191 (2016) 274–279

Contents lists available at ScienceDirect

Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad

Research paper

Polysomnographic characteristics of bipolar hypomanic patients: Comparison with unipolar depressed patients

Tarek Asaad, Walaa Sabry, Menan Rabie, Hanan El-Rassas

Institute of Psychiatry, Faculty of Medicine, Ain Shams University, Egypt

ARTICLE INFO

Article history:
Received 28 September 2015
Received in revised form
24 November 2015
Accepted 4 December 2015
Available online 7 December 2015

Keywords:
Depression
Hypomania
Bipolar disorder
Polysomnography
Sleep

ABSTRACT

Background: Sleep profile in bipolar disorder has received little attention in comparison to sleep studies in major depressive disorders. Specific sleep abnormalities especially in REM sleep parameters have been detected in depression. The current study aimed at investigating whether bipolar disorder shares the same polysomnographic (PSG) changes or not.

Methods: All night polysomnographic assessments were made for 20 patients diagnosed to have hypomania, in addition to 20 patients with major depression and 20 healthy matched controls. All participants were examined using Standardized Sleep Questionnaire, SCID-I for psychiatric diagnosis, based on DSM-IV criteria, YMRS (for hypomanic patients), HAMD (for major depression patients), and all-night polysomnography (for all subjects).

Results: The two patient groups differed significantly from controls in their sleep profile, especially regarding sleep continuity measures, Short REML (Rapid Eye Movement Latency), with increased REMD (Rapid Eye Movement sleep density). High similarity was found in EEG sleep profile of the two patient groups, though the changes were more robust in patients with depression

Limitations: A relatively small sample size, the absence of follow up assessment, lack of consideration of other variables like body mass index, nicotine and caffeine intake.

Conclusion: Similarity in EEG sleep profile between Bipolar disorder patients and patients with major depression suggests a common biological origin for both conditions, with the difference being "quantitative" rather than "qualitative". This quantitative difference in sleep efficiency and SWS (Slow wave sleep), being higher in hypomania, might explain the rather "refreshing" nature of sleep in hypomanic patients, compared to depression.

1. Introduction

Sleep disturbances are highly prevalent in affective disorders and a complex bidirectional relationship exists between disturbed sleep profile and different affective disorders (Taylor et al., 2005). This strong link between sleep and mood regulation makes polysomnography a useful window to discover the underlying pathophysiology of these disorders (Sculthorpe and Douglass, 2010). Polysomnography is considered to be one of the best objective monitoring system and the only way to assess the constitution of sleep stages, including changes in slow wave sleep and rapid eye movement sleep (Sivertsen et al., 2006).

According to the recently postulated vigilance model of affective disorders (vigilance in the sense of "brain arousal"), where destabilizing vigilance (e.g. by sleep deprivation) could trigger (hypo)mania and improves depression, whereas stabilizing vigilance, e.g. by prolonged sleep, improves mania (Hegerl and Hensch, 2014). These casual relationships between sleep/vigilance and mood suggest the existence of a possibly circumscribed underlying neurobiological mechanism and justify the high need for a better understanding and better treatment of these disorders.

In addition, studying sleep architecture could affect the choice of mode of treatment and illness outcome in both depression and BD. For instance, sleep disturbances in depression may predict poor response to Cognitive Behavioral Therapy (CBT) (Thase et al., 1997) and interpersonal therapy (Lam et al., 2003). Recurrence rate was found to be higher in depressed patients after successful CBT treatment if they have an abnormal sleep profile (Thase et al., 1997). Similarly in BD, sleep disturbances have important clinical and therapeutic implications that resulted in introduction of new pharmacotherapy agents (Plante and Winkelman, 2008) and modifications of various psychotherapeutic interventions that are directed to treat the sleep disturbances like Cognitive behavioral therapy for insomnia (CBT-I) (Frank et al., 2000) and interpersonal and social rhythm therapy (Harvey et al., 2009).

Numerous sleep abnormalities have been reported in MDD

http://dx.doi.org/10.1016/j.jad.2015.12.001
0165-0327/

(Major Depressive Disorder) measured with polysomnography especially during REM sleep periods (Sjostrom et al., 2007; Steiger and Kimura, 2010; Pillai et al., 2011). On the contrary less attention has been paid to sleep profile abnormalities in bipolar disorder than in unipolar depression, with only few studies conducted on bipolar patients (Mendels and Hawkins., 1971; Harvey et al., 2009). Sleep disturbance in BP disorders is considered as one of the diagnostic features, being mentioned in DSM-IV diagnostic criteria (American Psychiatric Association, 2000). It is also frequently reported during the euthymic periods of the illness, which proves that sleep disturbance is not only an epiphenomena of acute illness episode (Harvey et al., 2009), but it is a core part of the underlying etiology and maintenance of BD (Plante and Winkelman, 2008).

Despite the accumulating evidence for high biological vulnerability in bipolar patients (Jones et al., 2002), the exact etiologies are still not fully understood. A biological overlap between BD (Bipolar Disorder) and MDD which is mainly represented in substantial genetic and non shared environmental correlations between the two disorders are being a study focus in many researches (Thase et al., 1997; McGuffin et al., 2003; Bauer et al., 2006).

Many speculations about the mechanism for sleep changes in such disorders and its correlations with other biologic abnormalities have been mainly identified in depression (Peterson and Benca, 2006). So studying sleep profile in BD in comparison to a well established features reported for MDD, would be helpful in understanding more etiological aspects for BD and whether any similar sleep profile is shared between both disorders.

Considering the fact that sleep architecture can be affected by increased daytime activity in normal subjects, some studies claimed that it is still unclear whether polysomnographic abnormalities seen in manic episode of BD; are caused by the manic state per se or are secondary to other features of mania, such as increased levels of physical or mental activity (Plante and Winkelman, 2008). Another factor contributing to the contamination of sleep studies in BD is psychotropic medications which are likely to impact sleep and, thus, may present as a confounding variable in research (First et al., 1995).

Taken together the previous studies' assumptions, we decided to conduct this study on hypomanic patients, first because of the scarcity of sleep studies for this group of patients and secondly for the assumption that the low severity of bipolar features in hypomania would allow for better polysomnography results interpretation. Thus we could further investigate the sleep profile in bipolar disorder in comparison to the well-established features reported for major depression.

In the present study sleep disturbances in patients with hypomanic bipolar disorder II, Major depressive disorder as well as in healthy controls were investigated with polysomnographic measurements. *The aim was to assess if there are any similarities in polysomnography evaluated sleep profile characteristics between patients with MDD and hypomanic episode of BD-II (bipolar Disorder Type II).*

1.1. ***Hypothesis***

The polysomnograms by patients with hypomanic episodes of BD-II and those with MDD were expected to show significant differences in sleep structure and architecture than normal subjects. If similarities exist between BD-II and MDD, they might be indicative of some etiological overlap and common biological factors shared between both disorders.

2. Methodology

2.1. *Site of the study*

This is a cross-sectional, case control observational study; where the patients were recruited from the outpatient clinics of the Institute of Psychiatry, Ain Shams University, Cairo, Egypt. It is located in Eastern Cairo, serving a catchment area of eastern greater Cairo, together with the nearby provinces.

2.2. *Ethical considerations*

The study was conducted in accordance with the Helsinki Declaration for medical research of 1975 and in compliance with the guidelines of the Research and Ethics committee of Institute Of Psychiatry, Ain Shams University. The patients were informed about the nature of the research and the confidentiality of the obtained information. It was stated that the participation in the study is voluntary and the participants have the freedom to withdraw at any time. A printed consent was signed by each participant, including detailed description of the procedure, expected benefits, and possible drawbacks.

2.3. *Participants in the study*

2.3.1. *Patient group*

They were recruited over a two years duration and they all met the following criteria: patients of both sexes were included with an age ranged between 18–45 years, diagnosis of bipolar disorder-II (hypomanic episode), or unipolar depression (Major depressive episode), by DSM-IV criteria; no use of alcohol, drugs, or medications within 1 week before study, participants were also excluded if they reported suicidal ideations or plans. In order to control for confounding factors, patients with extreme age ranges, with history of co-morbid medical, psychiatric disorders, primary sleep disorder, alcohol, smoking and substance abuse were excluded. This was confirmed through conducting a urine toxicological screening for each participant.

2.3.2. *Control group*

For the purpose of comparison, we recruited 20 Egyptian male and female healthy volunteers without a past or family history of psychiatric illness in their first degree relatives. They were matched with the case group for age, gender, social standard and other demographic variables as far as possible. Control subjects with a medical or neurological disorder were excluded.

2.4. *Tools*

The following tools were used in the study:

2.4.1. *Assessment of clinical symptoms in the case group*

2.4.1.1. The structured clinical interview for DSM-IV axis I disorder clinician version (SCID I-CV) (First et al., 1995). A semi structured diagnostic interview based on an efficient but thorough clinical evaluation, to confirm the diagnosis of bipolar disorder, determine its type and exclude other axis-I comorbid psychiatric conditions. The study used the Arabic version of the Structured Clinical Interview for DSM-IV axis I Disorders (SCID-I) (Asaad et al., 2014). The clinical version was used rather than the research version for its relatively easier administration and coverage of the diagnoses most commonly encountered in clinical settings.

2.4.1.2. Hamilton rating scale for depression (HRSD) (Hamilton, 1980). This is a 21- item rating scale for assessing the severity of depressive symptoms, and monitoring treatment. It is the most