Journal of the
Bahrain Medical Society

CASE REPORT

Insomnia is a Prodromal Symptom for Psychosis

Mohamed A. Serageldin[1*], Waiel Hikmet Salman Alani[2]

[1]Senior Resident, Psychiatric Unit, Bahrain Defence Force Hospital, Bahrain; Specialist-Neuropsychiatrist, Ain Shams University, Cairo, Egypt; Specialist-Psychiatrist, Al Abbaseya Psychiatric Hospital, MOH, Cairo, Egypt.

[2]Consultant Psychiatrist, Bahrain Defence Force Hospital, Kingdom of Bahrain.

***Corresponding author:**

Mohamed A. Serageldin, Senior Resident, Psychiatric Unit, Bahrain Defence Force Hospital, Bahrain; Specialist-Neuropsychiatrist, Ain Shams University, Cairo, Egypt; Specialist-Psychiatrist, Al Abbaseya Psychiatric Hospital, MOH, Cairo, Egypt, Tel: (+973) 34135982, Email: mserag5414@gmail.com

Received date: January 31, 2019; **Accepted date:** May 16, 2019; **Published date:** June 30, 2019

Abstract

A 23-year-old male patient, complaining of insomnia for the last two years, has been unresponsive to several trials of sedatives and hypnotics. Moreover, he has experienced multiple fluctuating symptoms of dysphoric mood, isolation, anhedonia, and easy provocation; accordingly, he was prescribed a dosage of escitalopram (20 mg/day per day) and quetiapine (50 mg/day), at night. Later, the symptoms were resolved, except for insomnia. Consequently, other trials of psychotropic medication were received, without any improvement till he presented with delusion of persecution, auditory hallucinations (second person with ordering type), television broadcasting, and depersonalization. At that time, olanzapine 20 md/day, in addition to depakine chrono (500 mg, BID) and risperidone (2 mg/day), were prescribed; but, he showed minimal improvement. This case indicates that a possibility of insomnia, to be the prodromal symptom for psychosis, should be given considerable attention in young patients, with resistant insomnia.

Keywords: Insomnia; Schizophrenia; Psychosis; Prodromal symptom; Polysomnogram.

Introduction

Insomnia is one of the prevalent sleep disorders. Approximately, one-third of the adult population is reported to experience sleep disturbance, and around 6-10% meet the diagnostic criteria for insomnia disorder.[1] Experience of chronic insomnia is also reported by about 5-12% of the general population. It is defined as an experience of an inability to obtain an adequate amount or quality of sleep, for minimum of three nights per week, for at least a month. There are wide variations in the prevalence of insomnia, depending on the definition of insomnia considered, populations studied, and research methodologies used; its prevalence ranging from 5-40%, in Western and Asian countries.[2] Sleep disruption is considered to be an important trans-diagnostic factor involved in the development and maintenance of many psychiatric disorders.[3] It was hypothesized that the link between sleep and psychiatric symptoms is often bi-directional and co-occurring. Sleep or psychiatric disorder usually affects the severity of other comorbid disorders.[4]

Psychotic disorders are relatively common and are considered some of the most debilitating psychopathologies, with onset in adults.[5] A variety of sleep problems, ranging from insomnia to parasomnias, which were associated with psychosis in adults, has been indicated by both objective and subjective studies. In adults with schizophrenia, certain sleep disorders such as insomnia, are frequently observed, and is characterized by sleep-onset and maintenance insomnia, obstructive sleep

apnea, restless leg syndrome, and periodic limb movement disorder. The significant presence of insomnia has often been observed as a preceding symptom before a psychotic episode, with demonstrated effect on the prognosis, when it continues during the illness.[5]

Growing evidence indicated the importance of sleep disorders as potential markers for development of psychotic symptoms as well as prognosis of psychotic disorders in adults.[6] Studies have provided the evidence that chronic insomnia may precede the presence of psychosis, and all of these results imply that sleep disturbances may be inherent in the pathophysiology of schizophrenia.[2] Although few studies have examined sleep in the prodromal period, retrospective data from schizophrenia samples suggest that impairments in sleep duration and continuity, precede onset of psychosis.[7] Abnormalities in sleep dysfunction, preceding schizophrenia onset, was suggested after results from an online database search targeting genetic high risk, clinical high risk, and retrospective studies of patients with schizophrenia, prior to onset.[8] Relationship between sleep deficits and psychosis symptoms at a single time, examined throughout several studies highlighting the link, and investigating the self-reported sleep disturbance in the prodrome, have revealed increased sleep latency and nocturnal awakenings among studied samples compared to healthy controls. This also indicated significant associations between poor sleep quality and precipitation of the negative symptoms.[7] All in all, these results suggest that sleep difficulty occurs in the prodromal period along with pointing to its possible role in the etiology and pathophysiology of psychosis.

Case presentation

At this stage, we report a 23-year-old nonsmoker, Bahraini male, working at the Bahrain Defence Force, who presented with insomnia in March 2017, and has been unresponsive to several trials of sedatives and hypnotics. At first, an Ethical approval was obtained from the Research and Research Ethics Committee, Bahrain Defence Force Royal Medical Services. The written and informed consent was obtained from patient, before starting the study. During initial observation, previous reports of premorbid symptoms, a family history of psychosis, or any other mental disorders, were not observed. In addition, he had no previous history of psychotropic drug use, including antidepressants and antipsychotics, or alcohol and substance abuse. Additionally, no abnormal developmental or neurodevelopmental disorders, were found. Consequently, Standardized Sleep Questionnaire (Arabic version)[9] was considered. Neurological examinations and laboratory tests, including thyroid function tests, hemoglobin level, and hematocrit, arterial blood gases, drug and alcohol toxicology screening, and electroencephalography, detected no abnormalities.

After four months, he presented with a dysphoric mood, isolation, and anhedonia, with being easily provoked. For this presentation, he was prescribed mirtazapine (30 mg/day) as a sedative antidepressant medication, but no improvement was noticed. Subsequently, he was diagnosed with a major depressive disorder, for which his medication was changed to escitalopram (10 mg/day, titrated to 20 mg/day) and quetiapine (50 mg/day) at night, as a sedative medication. Post one week, he showed improvement of the depressive symptoms, but still complained of insomnia. After three months, zopiclone (7.5 mg) was prescribed instead of quetiapine, as a sedative medication. Nevertheless, he had not been compliant with the antidepressant, since his depressive symptoms improved without any relapse.

A month later, he reported irritability, restlessness, being easily provoked, and feeling energetic. At this point, he was a patient with bipolar affective disorder, which was considered as the shifting effect of the escitalopram or was one of the depressive symptoms, which was the index episode of the bipolar affective disorder. Hence, depakine chrono (500 mg, BID) was added to his current medications. A week later, his symptoms improved, and yet he was experiencing from insomnia. So, quetiapine was re-prescribed, combined with zopiclone and titrated to 125 mg/(HS), and continued for one month. During follow-up, he complained from lower limb pain with a sense of electric shocks in his head, which he attributed to depakine that was

changed to tegretol CR and titrated to 400 CR mg/day.

Upon this complaint, a sleep study was initiated when the polysomnogram reflected prolonged sleep latency, a decreased stage (four durations), a reduced rapid eye movement (REM), disruption in sleep continuity, shortened REM latency, and a normal-to-increased REM density, after which zopiclone was ceased and valium (5 mg), was commenced. Post that, he quit the follow-up visits.

A year later, he presented with psychotic symptoms fulfilling Diagnostic and Statistical Manual of Mental Disorders diagnostic criteria for schizophrenia, delusion of persecution, auditory hallucinations (second person with ordering type), television broadcasting, and depersonalization. Further to this, easy provocation was being experienced with bouts of agitation and aggression. Consequently, toxicology screening was conducted and the results were negative. He was prescribed an antipsychotic: olanzapine (10 mg/day) with titration (20 mg/day), together with depakine chrono (500 mg, BID) oral tablets, which led minimal improvement of symptoms, with respect to delusions. Hence, risperidone (2 mg/day, oral tablet) was prescribed, which also led to minimal improvement of his psychotic symptoms. On the other hand, he reported partial improvement in his sleep latency (of four hours), as he had recorded. The patient had been admitted into the hospital for four days, but his family signed a leave against medical advice. Then, a psychoeducation about the electric convulsive therapy was illustrated, but they were reluctant about it.

Discussion

In this case report, we noticed the fluctuation in the symptoms and diagnoses related to insomnia, which is confusing?! All through the patient's history, he initially presented with insomnia, and then with depressive symptoms that might be a secondary depression to insomnia. However, it resolved after one week of the antidepressant prescription. After a while, he presented with severely manic symptoms of bipolar affective disorder, which was also controlled within a week from taking a mood stabilizer. Finally, he was attended for his psychotic symptoms that were in concordance with multiple retrospective studies, which reported the importance of insomnia as a vitally potential marker for the development of the psychotic symptoms as well as the prognosis of the psychotic disorders, in adults.

The correlation between sleep disturbances, as a prodromal symptom or a predictor of psychosis, is not thoroughly studied. This is because most of the sleep researches are challenging due to the cognitive abilities that affect the self-awareness during the psychotic episode, along with the reliability of the subjective reports.

Another point to add is that the neurodevelopmental diathesis-stress model is one of the proposed models to provide a possible explanation for the bi-directional nature of psychosis and sleep. It is also proposed that there are shared genetic and environmental factors that have a great impact on the development of sleep dysfunction as well as psychosis.[10] On top of this, the interaction between sleep and cognitive deficits, due to the psychosocial and biological changes, associated with neuro-maturational factors across stress-related developmental stages, has been proven to be associated with psychosis-like symptoms. While the concept is untested, it supports the biological underpinnings of sleep and psychiatric disorders, whereas it contradicts the traditional stress-diathesis model for schizophrenia.[8]

It has been found that insomnia is a predictor of psychotic-like experiences, as conducted in the study by Lee et al.[11] Nevertheless, this finding has not been supported by a longitudinal study conducted by Cannon et al., but he has acknowledged that sleep disturbance is only a general symptom in youth, with a highlighted risk of psychosis.[12] Although the results of these studies are contradictory, sleep disturbance may have indirectly led to higher levels of suspicion/paranoia or higher levels of unusual thought content in the former study, which are considered as predictors of psychosis. [8]

Another evidence has been provided by Chung et al., to shed light on the idea that chronic insomnia may take place before the presence of psychosis.

All of these results considered, imply that sleep disturbances are to an extent inherent in the pathophysiology of schizophrenia.[2] In a longitudinal study, conducted by Jessica et al., some results suggesting a potential role for sleep problems in the etiology of schizophrenia, have been determined; such an outcome throws a strong light on sleep health as a possible target for prevention/intervention efforts.[13] Castro et al., have also observed sleep physiology abnormalities in at-risk samples.[14] Another study for Lunsford-Avery et al., has outlined a comparison between increased sleep latency and nocturnal awakenings among psychotic sample to health restraints, with significant associations between poor sleep quality and increased negative symptoms.[7]

Finally, it was concluded that inspite of the perplexic presentation of the patient since he complained from insomnia, it was obvious from our case that insomnia/sleep disorders could be considered as a significant predictor of development of psychosis in individuals with high risk. So, sleep symptom assessments in clinical practice, as an integral part of assessment and treatment planning for youth presenting with psychosis-like experiences, is extremely needed for better future consequences and a better quality of life.

Conflict of interest

Authors have no conflict of interest to declare.

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