CASE REPORT

Olanzapine-Induced Somnambulism: A Case Report

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Abstract

Parasomnia is a disorder of arousal that can be classified as either non-rapid eye movement (NREM) or rapid eye movement (REM) parasomnia. Somnambulism is an example of NREM parasomnia. There are observations that suggest a complex association between parasomnia, co-morbid mental illness and use of antipsychotics. In this case report, we observe the possibility of Olanzapine-induced somnambulism in a patient with schizophrenia and the resolution of somnambulism after Olanzapine was withdrawn.

Keyword: Olanzapine, Somnambulism, Sleep Walking

Introduction

Parasomnia is a disorder of arousal, partial arousal and sleep-stage transition which leads to undesirable physical or experiential events during sleep. It can be broadly categorised to REM and NREM parasomnia. Likely associations exist between psychiatric disorders and REM parasomnias, especially nightmare disorder and sleep paralysis [1]. On the other hand, somnambulism (sleepwalking), a type of NREM parasomnia, is strongly associated with the use of psychotropic medications [1]. By blocking 5-HT2C receptors, antipsychotics enhance slow wave sleep which may give rise to parasomnias [8]. Behaviours reported with antipsychotic-induced somnambulism were head banging, throttling other members, talking loudly, and urinating at bedside [6]. This case report illustrates somnambulism seen in a patient with schizophrenia during an acute relapse while being treated with rapidly titrated doses of Olanzapine.

Case Report

SP is a 48-year-old Chinese female with a 30-year history of schizophrenia. She had been in remission on a monthly dosing of long-acting injectable (LAI) Fluphenazine 25mg, in combination with oral Quetiapine IR 100mg ON, Fluvoxamine 150mg ON and Benzhexol 2mg OD. She had no comorbidities, no family history of somnambulism and no history of using alcohol, nicotine, or illicit substances.

She presented to the Emergency Department (ED) with a 2-week history of mood lability and persecutory delusions. SP developed a single episode of generalised tonic-clonic seizure at home prior to presentation. In ED,

the high levels of agitation and excessive talkativeness required rapid chemical tranquillisation to control her behaviour. Physical examination, biochemical markers and CT brain showed no significant abnormalities. SP was admitted to the psychiatry ward for further stabilisation.

During the first 5 days of admission, SP continued to display irritability, logorrhoea, reduced need for sleep and increased goal-directed activity. She was started on a combination of Olanzapine, Sodium Valproate, and Clonazepam. Physical restraint was not enforced. Medication dosages were optimised to T Olanzapine 20 mg ON, T Sodium Valproate 400 mg Om/600mg ON and T Clonazepam 2mg TDS on day 4 of admission as symptoms were still not well controlled. On day 6 of admission, the acute mania ceased, and SP returned to her premorbid self, a quiet & well-mannered lady.

SP then began to display somnambulism in the ward during the night. She would awake from sleep, speaking in Mandarin to Malay staff nurses despite being able to converse in fluent Malay during the daytime. She also became disruptive and had to be restrained physically and chemically. The following morning SP had no recollection of the night's incident.

For the next 6 days, SP continued to display similar cyclical/circadian behaviours. She would imitate the act of defecating in the hallway, waking other patients to shower, speak in Mandarin and react aggressively whenever she was put on physical restraints. Such behaviours were exclusive to the night, which did not correlate with her euthymic presentation during the day. SP would only have vague recollection of the events that transpired the night before.

Our initial diagnosis was nocturnal frontal lobe epilepsy (NFLE) but electroencephalogram (EEG) results ruled out any evidence of seizures hence the diagnosis was changed to parasomnia secondary to antipsychotic use. SP was switched from Olanzapine to Risperidone and her symptoms gradually resolved. She no longer displayed nocturnal abnormal behaviour 3 days after Olanzapine was removed and remained well after she was being discharged home.

Discussion

Somnambulism is a series of complex behaviours arising from sudden arousals from slow-wave sleep (SWS) resulting in abnormal behaviour while in a state of altered consciousness [5]. The exact mechanisms of the disorder are unknown, but it is suspected to be related to changes in SWS during stage 3 to 4 of NREM sleep [7]. There also have been studies that found sleep deprivation to be a trigger for sleepwalking episodes [2]. This is consistent with our case report of SP who developed sleep deprivation from her acute phase and experienced somnambulism after being treated with rapidly titrated antipsychotics in the ward.

Differentiating parasomnia from NFLE can be challenging. First, a detailed clinical history including descriptions of the content of sleep-related behaviours and its time course must be obtained. However, this information may not be readily available if the patient lives alone or the other tenants in the house are unaware of its occurrences. Second, the behavioural patterns in parasomnia and NFLE have a lot of similarities [5]. Third, there is no gold standard test to differentiate parasomnia from NFLE. Polysomnography can be used to aid in identifying parasomnia episodes,

but that is subjected to the emergence of behavioural events during recording and availability of service in the hospital [5]. We were unable to obtain the necessary history for SP as she lives with her elderly mother who is a poor historian. Despite having no prior history of epilepsy/seizures and a normal EEG, NFLE could still be a differential diagnosis in view of SP's initial presentation of tonic clonic seizure to the hospital.

Lastly, it has been postulated that certain medication may affect SWS resulting in parasomnia disorders. Stallman et al. identified 29 drugs associated with sleepwalking in their systematic review. Among the atypical antipsychotics, Quetiapine and Olanzapine stands out with the greatest number of case reports of antipsychotic-induced sleepwalking [2]. Chopra et al. also discovered similar findings in their systematic review whereby Quetiapine had the most case reports of antipsychotic-induced sleepwalking followed by Olanzapine [6].

SP displayed somnambulism after initially being treated with high doses of Olanzapine for acute management of her manic symptoms. This was a stark contrast to her gentle demeanour during the day as her manic symptoms began to subside. She no longer displayed nocturnal abnormal behaviour 3 days after Olanzapine was removed and remained well after she was discharged. Therefore, we infer that the sleepwalking episodes were precipitated by Olanzapine use.

Conclusion

Sleepwalking can be traumatic and potentially harmful to the patient and other inhabitants within the vicinity; hence it is imperative that it be promptly recognised when sleepwalking is precipitated by antipsychotics and the causative agent be discontinued for the safety of the patient and the other inhabitants.

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