

Zaleplon-induced Amnesic Somnambulism with Eating Behaviors Under Once Dose

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Abstract-

Purpose: Zaleplon is a newly-developed rapid-acting non-benzodiazepine hypnotic. Few reports discuss zaleplon-induced somnambulism. This report describes a patient without history of somnambulism, who developed amnesic somnambulism after taking low-dose of zaleplon.

Case Report: A 43-year-old schizophrenic male without history of physical illness, seizure, or somnambulism developed amnesic somnambulism after taking once low-dose of zaleplon from the outpatient-department. Somnambulism did not reoccur after discontinuing zaleplon. However, similar behaviors recurred after accidentally prescribing zaleplon on the first-day of hospitalization.

Conclusion: This patient is the first case without history of somnambulism, who developed zaleplon-induced somnambulism after taking low-dose of zaleplon. This study underscores the importance of monitoring for zaleplon-induced somnambulism, even when administering low-dosage to those without risk factors or history of somnambulism.

Key Words: zaleplon, hypnotics, somnambulism, adverse effect, schizophrenia

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PURPOSE

Amnesic somnambulism (AS) is an amnesic sleep disorder, which etiology is unclear. Hypnotosedatives-induced AS has recently received considerable attention⁽¹⁾. Zaleplon is characterized by short-acting hypnotosedatives, which is believed to have fewer side effects than other hypnotosedatives. Unlike zolpidem, AS after taking low-dose zaleplon has not been reported^(2,3). Here, we report a

patient without history of somnambulism, who developed AS after taking low-dose of zaleplon.

CASE REPORT

Mr. A is a 43-year-old schizophrenic male without history of systematic physical disease, seizures, or somnambulism. He lost follow-up for 4 years without any psychotropics. Recurred psychosis with auditory

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hallucinations attacked for 2 months. He resumed 3mg/day risperidone, and 10mg/day zaleplon. However, AS with eating happened at home after taking 10mg zaleplon and subsided after discontinuing zaleplon. He was later hospitalized for deteriorated psychosis. Zaleplon was accidentally prescribed and similar AS recurred at midnight only few hours after sleep. He walked around and looked for things eatable without any accident or injury. The polysomnography could not be performed because of the unstable psychiatric disease. The laboratory data, neurologic/physical examination and electroencephalography revealed no significant abnormality. He was discharged under 3.5mg/day risperidone, and 1mg/day estazolam without recurred AS.

DISCUSSION

To the best of our knowledge, this is the first case without history of somnambulism, which developed AS after taking once low-dose zaleplon. Although somnambulism can have adverse consequences, its etiology remains unclear. In one recent review article, one of the theoretical etiology of somnambulism was the disturbance of alertness, rapid-eye movement (REM) sleep, and non-REM sleep⁽⁴⁾. The psychotropic drugs, such as hypnotosedatives, could lead to somnambulism through the modulation of sleep-alertness⁽⁴⁾. Actually, some other authors suggests that hypnotosedative-induced somnambulism be related to enhanced γ -aminobutyric acid (GABA) activities at $\alpha 1$ subunit of GABA_A receptors; the activation of $\alpha 1$ subunit are believed to induce the sedative, amnesic, and motor-impairing effects^(1, 5-7). The serotonergic hypothesis is based on increased serotonergic activity during tonic muscle activities and dissociation between activation of serotonergic neurons and arousal^(8,9). Both hypotheses, however, cannot explain the mechanism of hypnotosedative-induced somnambulism. Although another new hypothesis, focusing on the desensitization of GABAergic receptors, is raised, it lacks direct evidence⁽¹⁰⁾.

The non-benzodiazepine hypnotics, including zaleplon and zolpidem, are distinct medications with similar effects on GABA_A receptors⁽¹¹⁾. Dolder et al. summarized risk factors related to hypnotosedative-induced somnambulism by reviewing articles and found that dosage of those medications and the binding affinity for $\alpha 1$ -subunit of

GABA_A receptors are consistently risk factors⁽¹⁾.

In terms of managing zaleplon-induced somnambulism, these studies suggest avoiding further zaleplon usage, or titrating down the zaleplon dosage^(2,3). According to Dolder and Nelson, some strategies may work for managing hypnotosedative-induced somnambulism, such as lowering the hypnotosedatives dosage or discontinuing use⁽¹⁾. In this case, the patient was free from somnambulism after shifting zaleplon to estazolam as the primary management strategy.

CONCLUSION

This case highlights the importance of monitoring somnambulism in subjects taking zaleplon, even under a low dosage.

AUTHORS CONTRIBUTIONS

The first author, Yen-Wen, Chen, is responsible for the first paragraph (introduction) and case presentation.

The second author, Ping-Tao, Tseng, completes the remaining paragraph, including the discussion section.

The third author, Ching-Kuan, Wu, provides the explanation of mechanism of somnambulism of zaleplon.

The corresponding author, Chien-Chih, Chen, takes the responsibility of concluding all the literature and related reference and articles. Besides, he is also responsible for the corresponding to submission.

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