



CORRESPONDENCE

Propranolol-Induced Somnambulism: An Ignored Adverse Effect?

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We present a 24-year-old male patient with a history of episodic migraine without aura since age 12. He was initially medicated with topiramate, but due to adverse effects, a switch was made to propranolol with a gradual increase to 40 mg twice a day. Despite an improvement in the frequency, duration, and intensity of the headache crisis, 2 weeks after starting this therapy, the patient started having nocturnal episodes, every week, characterized by wandering around the house and carrying out purposeless everyday tasks, with minimal interaction, lasting several minutes. The patient reported occasional soliloquies, but without a previous history of somnambulism or other sleep parasomnias, particularly in childhood, but with a positive family history of sleepwalking (brother). A diagnosis of somnambulism induced by propranolol was made. The patient stopped gradually the propranolol and started amitriptyline until 25 mg/day, with the resolution of the sleepwalking and good control of his migraine.

In this report, the patient had no history of somnambulism, although positive familiar history. He experienced episodes of sleepwalking 15 days after starting propranolol until 40 mg bid. The symptoms completely disappeared after the withdrawal of propranolol. In the literature, there are 5 more cases of somnambulism related to propranolol [1-3]. All patients were female (age range: 24–61 years old), with propranolol doses between 20–120 mg/day. In one of them, propranolol was added with another drug (olanzapine). In 3 of the patients, there was a history of somnambulism. The onset of sleepwalking ranged from 1–33 days after

drug introduction. The resolution of sleepwalking in all cases involved discontinuing the medication [1-3]. Apart from propranolol, there are 2 more cases of sleepwalking related to other beta-blockers (metoprolol) [4,5].

The proposed mechanism for somnambulism induced by some beta-blockers, such as propranolol and metoprolol, but not all is still unclear, but it appears to involve reduced melatonin synthesis, via central beta-1 adrenergic receptors [6]. These actions could result in increased arousal during sleep and abnormal behaviors [7]. Another theory is that beta-blockers bind to 5-hydroxytryptamine receptors and precipitate abnormal rapid eye movement and non-rapid sleep behavior [7].

Withdrawal of the drug appears to be the best choice [1-3], but the use of melatonin can improve sleep quality in patients taking beta-blockers, decreasing sleep latency and increasing sleep maintenance [6,8].

In conclusion, sleepwalking is a rare adverse effect caused by propranolol in patients with or without a previous history of sleepwalking or another sleep disorder. The minimum dose to induce sleepwalking is unknown. Since propranolol is a drug widely used in the clinical practice of neurology, physicians need to be aware of this adverse effect.

Ethics Statement

Informed consent was obtained from the patient.

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Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

Availability of Data and Material

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generated or analyzed during the study.

Author Contributions

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