CASE REPORT

Paroxetine-induced severe sleep bruxism successfully treated with buspirone

Aysel Milanlıoglu

Van Medical Park Hospital, Clinic of Neurology, Van, Turkey.

Email: ayselmilanlioglu@yahoo.com
Tel.: 90-530-8263565 / 90-432-2223222

INTRODUCTION

Sleep bruxism is characterized by the involuntary clenching or grinding of the teeth during sleep and can cause severe health problems, including the destruction of tooth structure, temporo-mandibular joint dysfunction, myofascial pain, and severe sleep disturbances (1). Iatrogenic sleep bruxism may be common during treatment with pyschotropic medications, such as antipsychotics and antidepressants, especially selective serotonin reuptake inhibitors (SSRIs) (2). This article reports the case of a depressive woman with paroxetine-induced sleep bruxism who was successfully treated with buspirone.

CASE DESCRIPTION

A 38-year-old married woman who was suffering from severe headaches, sadness, anhedonia, and feelings of worthlessness and decreased energy and concentration visited our outpatient clinic. This patient was diagnosed with a depressive mood and muscle contraction headaches, and a regimen of paroxetine was initiated with a bedtime dose of 10 mg/day. The patient had no previous personal or family history of any movement disorder, substance abuse or mental illness. After 7 days at this dosage without adverse effects, the dose was increased to 20 mg/day. At the 3-week follow-up visit, the patient showed marked improvement in her headaches and depressive symptoms, and she was pleased with this treatment. However, her husband noted his wife's severe teeth clenching and associated loud grinding noises during sleep. All laboratory results were within the normal ranges, and all potential organic contributors to depression and bruxism were excluded. The dental examination also did not reveal any abnormalities.

A few days later, the patient started to complain of jaw pain and stiffness in the morning. As this problem caused her distress, the dosage was reduced to 10 mg/day. Due to the patient's lack of improvement over the next week, paroxetine treatment was discontinued, although the bruxism did not resolve after the therapy had been discontinued.

As buspirone has been shown to be useful for the treatment of bruxism, we began to administer buspirone to the patient at a dose of 5 mg/nightly. This dose was eventually increased to 10 mg/nightly. Within 2 weeks of beginning buspirone treatment, the patient and her husband reported the disappearance of the jaw pain and stiffness, as well as the bruxism symptoms.

DISCUSSION

Bruxism is a common movement disorder that affects 8-21% of the population. The majority of bruxism symptoms are mild or moderate, although rare but severe cases may lead to serious periodontal damage, temporo-mandibular dysfunction, sleep disturbances, jaw pain, and stiffness. As a result, such cases must be treated with medication (3).

The neurochemical mechanism underlying the development of bruxism remains unclear. However, recent investigations have suggested that the central dopaminergic system (especially within the meso-cortical tract), which controls muscular or motor activity, may be involved in the pathophysiology of bruxism (4). It has been hypothesized that the mechanism for SSRI-induced bruxism may involve excessive serotonergic action on the meso-cortical neurons arising from the ventral tegmental area. This action leads to a dopaminergic deficit, which causes a specific form of akathisia and akathisia-like movement of the jaw muscles, thereby leading to bruxism (5).

Kishi described an old, depressive Japanese man who displayed paroxetine-induced bruxism symptoms that were effectively treated with the 5-HT1A receptor tandospirone (6). To the best of our knowledge, the case described here is the first reported case of paroxetine-induced sleep bruxism to be treated successfully with buspirone.

Drug-induced movement disorders typically respond to a reduction in drug dosage, whereas our patient exhibited no improvement following the dose reduction or discontinuation of the drug in question. As a result, these symptoms may have been prevented through the use of buspirone alone.

Buspirone is an agonist of the 5-HT1A receptor that increases dopaminergic neuron firing in the ventral tegmental area and increases the synaptic release of dopamine in the prefrontal cortex. These effects ameliorate drug-induced bruxism. Buspirone can also ameliorate extrapyramidal side effects, such as akathisia and tardive dyskinesia, and this property may be an additional explanation for the bruxism-related effects of the drug (5).

Thus, the present case describes rare but important side effects associated with the use of SSRIs. In light of our findings, we emphasize that all clinicians should be aware that antidepressants may cause bruxism. Furthermore, buspirone may be an effective treatment for the bruxism associated with the use of these medications.

No potential conflict of interest was reported.

REFERENCES

1. Kuloglu M, Ekinci O, Caykoylu A. Venlafaxine-associated nocturnal bruxism in a depressive patient successfully treated with buspirone. J Psychopharmacol. 2010;24:627-8, http://dx.doi.org/10.1177/0269881109102612.
2. Winocur E, Gavish A, Voikovitch M, Emodi-Perlman A, Eli I. Drugs and bruxism: a critical review. J Orofac Pain. 2003;17:99-111.
3. Attansio R. An overview of bruxism and its management. Dent Clin North Am. 1997;41:229-41.
4. Lavigne GJ, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. Crit Rev Oral Biol Med. 2003;14:30-46, http://dx.doi.org/10.1177/154411130301400104.
5. Jaffee MS, Bostwick JM. Buspirone as an antidote to venlefaxine-induced bruxism. Psychosomatics. 2000;41:535-36, http://dx.doi.org/10.1176/appi.psy.41.6.535.
6. Kishi Y. Paroxetine-induced bruxism effectively treated with tandospirone. J Neuropsychiatry Clin Neurosci. 2007;19:90-1, http://dx.doi.org/10.1176/appi.neuropsych.19.1.90.