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Management of antidepressant-induced bruxism

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Introduction

Ms. C is a 22-year-old female with a past medical history of gastrointestinal reflux disorder (GERD), major depressive disorder, and generalized anxiety disorder. She is currently receiving psychotherapy virtually, but recently, her mood and sleep have worsened, requiring her to increase her coffee intake to 5 cups per day. Because her symptoms have become exceedingly disruptive to her personal life, she is now motivated to discuss pharmacotherapy. She is started on sertraline 50 mg by mouth once daily, hydroxyzine hydrochloride 10 mg by mouth 4 times daily as needed for anxiety, and trazodone 25 mg by mouth as needed for insomnia. Approximately 4 weeks after starting the medications, she notices prolonged periods during which her "jaw feels clenched down." She realizes that she has been waking up with jaw pain in the morning and is now noticing the same tightness throughout the day, most recently yesterday while at work. She reports that she has only been taking the sertraline and questions if it could be causing her jaw clenching.

Background

Approximately 1 in 8 Americans aged 12 and over are currently prescribed an antidepressant.[1] While nausea, headaches, drowsiness, and dry mouth are appreciated as common adverse effects of these medications, bruxism is underrecognized. Bruxism is a condition associated with unconscious repetitive movements of the mouth, such as jaw clenching or teeth grinding. These abnormal movements can occur during sleep or in a wakeful state and, in severe cases, can lead to jaw pain, fatigue, headaches, and damaged teeth. Risk factors for bruxism include stress, consumption of alcohol, tobacco, or coffee, and other medical conditions such as anxiety, sleep apnea, and GERD.[2] The incidence of antidepressant-associated bruxism is currently unknown; however, a recent multicenter study from 2015 found an incidence of 14% among all patients taking antidepressants.[3] Symptoms of bruxism can be seen as early as 4 weeks after starting or increasing the dose of an antidepressant, with an average onset of 2 to 4 months.[3,4]

The exact mechanism by which antidepressants cause bruxism is not fully understood. Dopaminergic and serotonergic neurons primarily regulate motor activity of the jaw in the mesocortical tract. Specifically, dopamine inhibits spontaneous jaw movement, while serotonin blocks dopaminergic signaling and allows for such jaw movement. Thus, as many antidepressants are serotonergic, the reduction in dopaminergic tone is thought to be responsible for the abnormal jaw movements that are characteristic of this condition.[5,6] However, an alternative hypothesis for the pathophysiology of bruxism involving norepinephrine has also been proposed and will be further discussed in the next section.

Antidepressants Associated With Bruxism

Antidepressant-associated bruxism appears to occur more frequently with selective serotonin reuptake inhibitors (SSRIs) and serotonin and norepinephrine reuptake inhibitors (SNRIs). In a meta-analysis from 2018, authors described 46 unique cases of antidepressant-induced bruxism, with 74% being due to SSRIs.[4] Of the SSRIs, fluoxetine (26%),

Practice Points:

1. Risk factors associated with bruxism include stress, substance use (nicotine, alcohol, caffeine), and medical conditions, such as anxiety or sleep apnea. Antidepressants may also induce bruxism, though the exact mechanism is not well understood.
2. Symptoms of bruxism may emerge as soon as 4 weeks after starting or increasing the dose of an antidepressant, with a typical onset of 2 to 4 months. After adequate treatment, symptoms usually resolve within a few weeks.
3. There are currently no guidelines for the management of antidepressant-induced bruxism and no FDA-approved medications for this indication. Suggested management strategies include lowering the dose, discontinuing the offending antidepressant, or adding on pharmacotherapy that antagonizes serotonin and enhances dopamine transmission. This includes partial agonists of 5-HT_{1A}, such as buspirone or aripiprazole.

sertraline (15%), and paroxetine (13%) were the most common offending agents. Of the SNRIs, which accounted for 24% of cases, venlafaxine was most frequently responsible (15%), with duloxetine and atomoxetine constituting a smaller number of cases (4% each, respectively).[4] Additionally, a 2023 study investigating the associations between drug exposure and bruxism reported sertraline as the most frequent causative antidepressant (51 cases), followed by venlafaxine (50 cases), escitalopram (39 cases), citalopram (35 cases), and duloxetine (22 cases).[7] This study also sought to determine the potential mechanism of bruxism by using the pKi values of each antidepressant at the relevant monoamine transporters (NET and SERT). Of note, an association was found between the reported odds ratios for bruxism and NET, but not SERT. In contrast to the prior study, these findings suggest that bruxism may be due to norepinephrine activity rather than serotonin. Thus, more studies are needed to elucidate if bruxism is related to only serotonergic properties or if norepinephrine may also play a role. A complete list of antidepressants that have been associated with bruxism can be found in Table 1.

TABLE 1: Antidepressants associated with bruxism[3-7]

SSRIs	SNRIs	Others
Fluoxetine	Venlafaxine	Bupropion
Sertraline	Duloxetine	Vortioxetine
Paroxetine	Atomoxetine	Mirtazapine
Escitalopram		
Citalopram		
Fluvoxamine		

SNRI = serotonin-norepinephrine reuptake inhibitors; SSRIs = selective serotonin reuptake inhibitors.

Treatment of Antidepressant Associated Bruxism

Currently, there are no established guidelines for the management of antidepressant-associated bruxism. However, the current literature suggests several strategies, such as discontinuing or lowering the dose of the causative agent or adding medications to treat the symptoms. Though some case reports describe successful intervention by lowering the dose of the antidepressant, this strategy has mixed results in terms of efficacy.[3,4] In a systematic review of published case reports, the most commonly documented successful intervention was the addition of buspirone ($n = 20$), a 5-HT_{1A} partial agonist.[6] Buspirone is thought to increase the release of dopamine in the prefrontal cortex, thus reducing the symptoms of antidepressant-associated bruxism.[4] Other literature suggests that buspirone improves bruxism via adaptive neuronal and receptor mechanisms rather than direct receptor activity, which may better explain the 2- to 3-week delay in resolution of symptoms.[4] The range of possibly effective doses of buspirone used in these studies was 5 to 30 mg in 1 to 3 divided doses.[4]

Additionally, the use of low-dose aripiprazole (10 mg/d), a second-generation antipsychotic, which also has activity as a 5-HT_{1A} partial agonist, has been reported as a successful intervention in a single-case report.[12] Trazodone, a 5-HT_{2A} antagonist, at doses up to 200 mg/d, has also been used with benefit in 1 case report.[8] Other pharmacologic agents that have been reported in the treatment of antidepressant-induced bruxism, however, with inconsistent benefit, include the tricyclic antidepressant, amitriptyline, the first-generation antipsychotic, chlorpromazine, the second-generation antipsychotic, clozapine, and gabapentin.[4] Nonpharmacologic strategies have also been reported to alleviate symptoms, including electroconvulsive therapy and cognitive behavioral therapy.[4,6] There is also documentation of the successful use of dental appliances alone and in combination with pharmacotherapy, including both hard and soft occlusal night guards.[11,12] Risk-factor reduction (managing stress and reducing alcohol, coffee, and tobacco use) should also be recommended in addition to pharmacotherapy.

In the treatment of sleep bruxism in general, the dopamine agonist bromocriptine and dopamine precursor L-dopa have been used with some benefit.[10,14] In a small placebo-controlled trial ($n = 6$), 2 patients with sleep bruxism who were treated with bromocriptine showed an average 25% reduction in the number of bruxism episodes per hour of sleep.[10] L-dopa has been used in a small double-blind trial

TABLE 2: Treatment strategies for bruxism[4,7-10]

Antidepressant-Associated Bruxism		
Pharmacologic	**Nonpharmacologic**	**Sleep Bruxism**
5-HT_{1A} partial agonists	• CBT	Dopaminergic agents
• Buspirone	• ECT	• Bromocriptine
• Aripiprazole	• Dental appliances	• L-dopa
5-HT_{2A} partial agonists/antagonists		• Tryptophan
• Trazodone		Clonidine
• Chlorpromazine		Propranolol
• Clozapine		Hydroxyzine
Amitriptyline		Flurazepam
Gabapentin		

CBT = cognitive behavioral therapy; ECT = electroconvulsive therapy.

in combination with benserazide (n = 10), which also reduced the frequency of bruxism episodes.[14] Other agents that have been studied in sleep bruxism in children with less consistent results include clonidine, propranolol, hydroxyzine, and flurazepam.[9] However, it is unclear at this time if these agents are also beneficial in antidepressant-induced bruxism. A summary of the treatment strategies for both antidepressant-associated bruxism and sleep bruxism can be found in Table 2.

Case Update

To address the bruxism, Ms. C was counseled to reduce her coffee intake and implement stress-management strategies. Along with these nonpharmacologic interventions, her dose of sertraline was lowered from 50 to 25 mg by mouth daily, and buspirone 5 mg by mouth twice daily was added. However, her symptoms of jaw clenching continued to persist after a month with these changes. Given that she continued to experience minimal mood benefit and an intolerable adverse effect while on the combination of sertraline and buspirone, both were discontinued and replaced with bupropion XL 150 mg by mouth daily. After 3 weeks on the bupropion alone, her bruxism had completely resolved. She was titrated to bupropion XL 300 mg by mouth daily, after which she noted improvement in both her depression and anxiety without the return of any bruxism-related symptoms.

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