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# Bruxism triggered by Escitalopram persists even after discontinuing the drug: a case report

# Bruxismo desencadeado pelo Escitalopram persiste mesmo após descontinuidade do fármaco: um relato de caso

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## ABSTRACT

Bruxism is a motor disorder of multifactorial etiology characterized by an exacerbation of muscular activity associated with cleaning or removing teeth, with sound or wakefulness. Bruxism associated with SSRIs, described in the literature, has been documented by a rare adverse reaction and by the pathophysiology associated with the drug's central system distribution. After the use of Escitalopram for the treatment of depression, a patient presented a functional oral movement disorder caused by repetitive grinding of the teeth unconsciously, characterizing a picture of sleep bruxism, which resulted in hypertrophy of the masticatory muscles and severe headaches. The condition was alleviated by withdrawal of medication and the use of non-pharmacological treatments, however, there was never complete remission. Since depression is a disease that affects 15.5% of the Brazilian population throughout life and that the use of antidepressant drugs is essential for

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its treatment, it is possible to discuss its common and not so common adverse effects, such as the bruxism.

**Indexing terms**: Bruxism. Escitalopram. Selective Serotonin Reuptake Inhibitors. Sleep bruxism.

## RESUMO

O bruxismo é uma desordem motora de etiologia multifatorial caracterizada por uma exacerbação da atividade muscular associada ao ranger ou apertar os dentes, em sono ou vigília. O bruxismo associado aos ISRS, pouco descrito na literatura, é documentado com uma reação adversa rara e de fisiopatologia associada a distúrbios do sistema dopaminérgico central. Após o uso de Escitalopram para tratamento da depressão, a paciente apresentou um distúrbio do movimento funcional oral provocado pelo apertar repetitivo dos dentes de modo inconsciente caracterizando um quadro de bruxismo do sono, que teve por consequência a hipertrofia dos músculos mastigatórios e fortes dores de cabeça. O quadro foi amenizado pela retirada da medicação e pelo uso de tratamentos não farmacológicos, no entanto, jamais houve remissão completa. A depressão é uma doença que abarca 15,5% da população brasileira ao longo da vida e que o uso dos fármacos antidepressivos é fundamental para o seu tratamento, é imprescindível discutir seus efeitos adversos comuns e não tão comuns, como o bruxismo.

**Termos de indexação**: Bruxismo. Escitalopram. Inibidores seletivos de recaptação de serotonina. Bruxismo do sono.

## INTRODUCTION

Bruxism is a parafunction characterized by the non-functional contact of the teeth, which can occur consciously or unconsciously, mainly during sleep, manifested by grinding or clenching them. It is not a disease, but when exacerbated it can lead to a pathophysiological imbalance in the stomatognathic system [1].

In general, patients with bruxism frequently complain of tooth wear, fatigue and muscle pain causing limited mouth opening [1]. Furthermore, they complain of rough tooth surfaces and thermal hypersensitivity and report having been alerted by people around them about the noise of teeth grinding during their sleep [1]. In relation to the muscles, bruxism can cause an increase in tone and dysfunctions in muscular activity such as hypertonism of the masticatory muscles, especially the masseter, contractures and muscle spasms, which can trigger myositis [1].

Headache is also a frequently reported symptom, it is believed that the hypertonic state of the muscles, when the mandible and maxilla are pressed, produces ischemia and accumulation of metabolic products that stimulate nerve endings, causing pain [2]. This symptom is often described as discrete pressure in the forehead region, behind the eyes and along the origin of the masseter in the zygomatic arch, and can also occur unilaterally, just like a migraine without its associated neurological disorders [2].

The etiology of bruxism is not well understood, being a complex and multifactorial disorder and often difficult to identify [3]. Psychosocial factors, sleep disorders, chronic use of centrally acting drugs, occlusal disharmonies and disturbances in the dopaminergic neuronal pathway are commonly considered in its genesis [3]. However, what currently prevails in research on bruxism are central factors, resulting from the neurophysiological and neurochemical mechanisms involved in rhythmic mandibular movements related to chewing, swallowing and breathing [4].

Bruxism can be caused by changes in the activity or availability of several neurotransmitters in the central nervous system (CNS). The studies that present the highest levels of evidence point mainly to disorders in the central dopaminergic system [4].

Dopamine represents more than half of the catecholamine content of the CNS and has, among others, the function of inhibiting spontaneous movements. Thus, movements characteristic of bruxism can be observed if there is a change in the level of this neurotransmitter [4].

Movement disorders are clinical syndromes related to the increase or decrease in voluntary or involuntary actions related to movement and not associated with spasticity [5].

Among these symptoms, extrapyramidal symptoms such as akathisia, dyskinesia, parkinsonism and bruxism stand out spasticity [5]. It is estimated that this varied range of motor changes is related to the variability and involvement of different subtypes of serotonergic 5-HT receptors spasticity [5,6].

Some studies suggest the involvement of serotonergic receptors in the pathophysiology of bruxism, since serotonergic neurons act on dopaminergic neurons and act in the genesis of involuntary movements. The articles propose that increasing the availability of extrapyramidal serotonin levels could indirectly inhibit the release of dopamine in the striatum, increasing the stimulation of 5-HT1A receptors spasticity [5]. This information is corroborated by the effective relief of bruxism through the use of Buspirone, a 5-HT1A spasticity [5] receptor antagonist.

Although there appears to be general agreement that selective serotonin reuptake inhibitors (SSRIs) exacerbate bruxism, either as a direct result of the drug or as an exacerbation of an underlying condition, information about a possible association between SSRIs and bruxism is emerging mainly from sporadic clinical cases and observational studies spasticity [7].

Given that bruxism is centrally modulated, it is essential to discuss its relationship with the use of psychotropic medications, such as antidepressants, which modify some brain processes that affect mood, behavior and cognition, and are among the most prescribed drugs in the world, even more so in developed countries spasticity [4].

Antidepressants are preferably classified based on their pharmacological action, because new generation medications do not share common structures spasticity [8]. They can be divided according to the proposed mechanism of action, increasing the synaptic efficiency of monoaminergic transmission (particularly of noradrenergic and/or serotonergic neurons) spasticity [8]. Antidepressant medications produce an increase in the concentration of neurotransmitters in the synaptic cleft by inhibiting metabolism, blocking neuronal reuptake or acting on presynaptic autoreceptors spasticity [8].

Considering their pharmacological properties, it is conceivable that antidepressants have some adverse effects spasticity [9]. Serotonin syndrome can be mentioned as an acute side reaction to the use of antidepressants, in which there is an excess of serotonin, leading to hyperstimulation of its receptors, and consequently, to its adverse effects spasticity [9]. This syndrome can be characterized by disorders of rapid and involuntary muscle contractions, rigidity and hyperactivity spasticity [9]. These dysfunctions point to the hypothesis that bruxism could be a potential adverse effect of antidepressants, which have a therapeutic effect by influencing the activity of neurotransmitters, such as norepinephrine, dopamine and serotonin spasticity [9].

As an example of some classes of antidepressants associated with bruxism are Selective Serotonin Reuptake Inhibitors (SSRIs), Selective Serotonin and Noradrenaline Reuptake Inhibitors (SNRIs) and Selective Dopamine Reuptake Inhibitors (SSRIs) spasticity [10]. SSRIs (Citalopram, Fluoxetine, Fluvoxamine, Paroxetine and Sertraline) potently and selectively inhibit serotonin reuptake, resulting in increased

serotonergic neurotransmission. Although they share the main mechanism of action, it is worth highlighting that medications in the SSRI class are structurally distinct from each other, with marked differences in the pharmacodynamic and pharmacokinetic profile spasticity [10].

As a rule, case reports relating antidepressants and bruxism highlight the drug that caused or exacerbated bruxism and the methods for resolving the problem, as well as few symptoms associated with bruxism. The reports lack some important aspects, such as better detailing of the clinical parameters of bruxism triggered and the time taken to diagnose that bruxism was caused by the use of antidepressants. This report seeks to expose in detail the treatments carried out throughout the period of the condition, the clinical aspects of bruxism that emerged, as well as the time elapsed for the association between medication and bruxism to be made, seeking to consolidate the importance of prescription correctness of antidepressant drugs and the relevance of the adverse effects of these treatments.

## CASE REPORT

Woman, 43 years old, nurse, in 2010, was diagnosed by a psychiatrist with anxiety and depression disorder, with prescription of atypical antidepressant medication, Mirtazapine (30 mg, 1 tablet at night). She used the medication for a period of 12 months, with a significant improvement in her depressive symptoms during the initial 8 months, however, in the last 4 months the psychiatric disorder worsened, requiring a new evaluation and change of medication. In 2011, following professional reassessment, she suspended the use of Mirtazapine and introduced the SSRI antidepressant, Escitalopram (10 mg, 1 tablet in the morning).

The medication was effective for treating depression, however, after 40 days some adverse symptoms appeared, such as persistent pain on both sides of the face, worsening in the morning. Throughout the treatment, which lasted for 3 years and 6 months, the pain that was previously sporadic and morning pain evolved into frequent pain not limited to just the morning. Furthermore, there was a process of hypertrophy in the region of the masseter and temporal muscles, coupled with limited mandibular movements; tooth wear; headaches that worsened over time and; pain radiating to the neck and trapezius region. Several visits to the psychiatrist were made, however, without mentioning the signs and symptoms, due to the lack of clear correlation between them.

As a result of the severe pain, self-medication was carried out, initially in acute cases, progressing to chronic use due to the worsening of symptoms. Non-steroidal anti-inflammatory drugs were used, Diclofenac sodium (100 mg, 1 tablet every 12 hours) alternating with Ibuprofen (600 mg, 1 tablet every 12 hours) and Naproxen (500 mg, 1 tablet daily). Associated with anti-inflammatories, she used the muscle relaxants Cyclobenzaprine (10 mg, 1 tablet at night), interspersed with Lysine Clonixinate + Cyclobenzapine Hydrochloride (125 mg + 5 mg, 1 tablet at night). Naratriptan Hydrochloride (2.5 mg, 1 tablet per day) was used to treat headaches.

Due to the irradiation of pain to the neck and shoulder region, with no signs of improvement due to medication, she underwent follow-up with a professional physiotherapist for 3 months, using pulsed ultrasound associated with laser, seeking to promote relaxation of the muscles, however there was no favorable result and treatment was stopped.

At the beginning of 2013, due to dental wear, she sought dental care. During evaluation, the clinical presentation of signs and symptoms associated with the physical examination clinically diagnosed sleep bruxism (Figure 1A). To alleviate the symptoms, the professional prescribed the use of a semi-rigid

mouthguard for a period of 4 months, but there was no improvement, and replacement with a rigid mouthguard was recommended, which reduced the symptoms.

Due to the ineffectiveness of non-invasive therapies, as an adjuvant to the rigid protector, Botox (onabotulinum toxin A) applications were performed on both sides of the masseter and temporal muscles, respectively 30 international units (IU) and 10 IU, promoting partial relaxation of the muscles. Two months later, the signs and symptoms returned. After 8 months, a new Botox application was performed, this time inoculating 75 UI into the masseter muscle and 15 UI into the temporal muscle on both sides. There was greater relaxation of the muscles in relation to the previous application, however, along with the signs of relief, a decline in the muscles of the face and tongue was observed with a decrease in chewing strength over the 4 months that passed.

In a new consultation with the dental professional, the hypothesis was raised that the bruxism was related to some medication used by the patient, but it was not investigated. The patient, who only used medication to control depression, through reading articles and literature reviews, sought a correlation between her diagnosis of bruxism and the medication she used and found an association between them.

In mid-2014, when the woman returned to the psychiatrist, she reported all the adverse signs and symptoms caused by the treatment with Escitalopram and addressed the association between

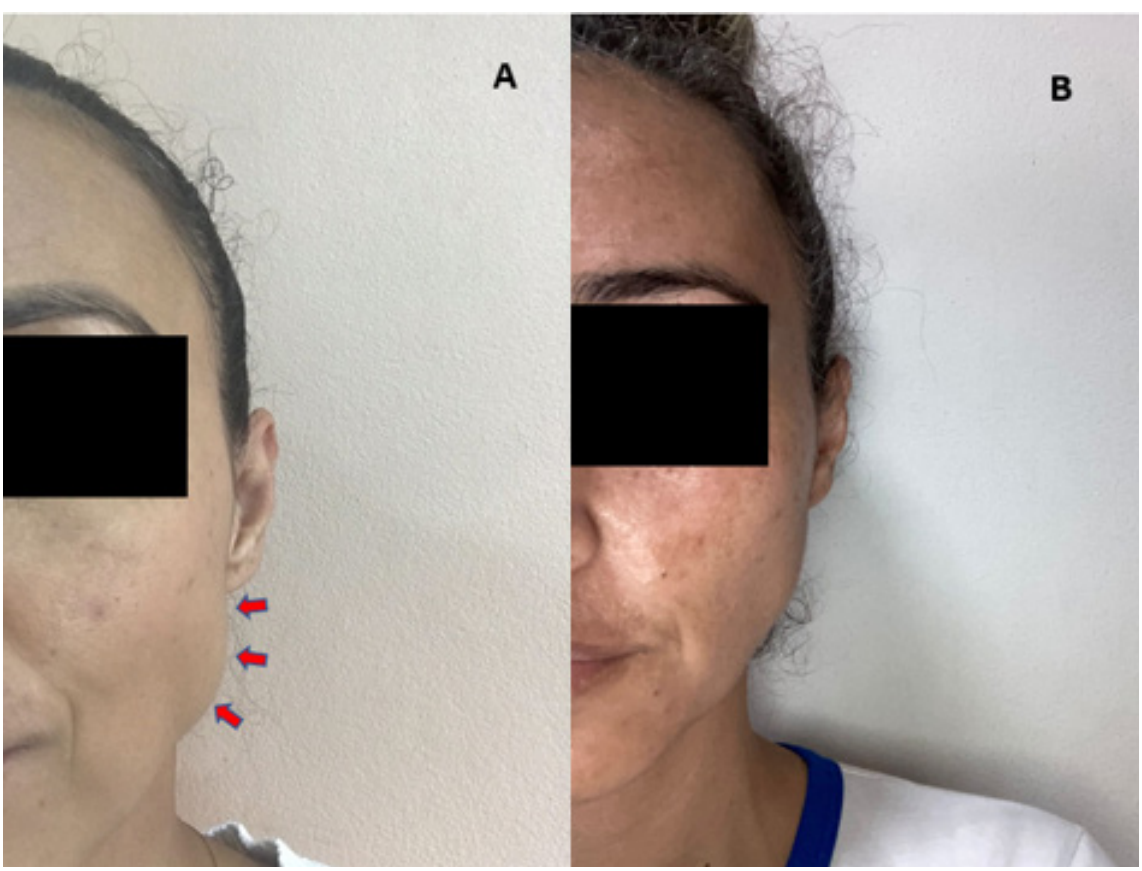


**Figure 1**. A) taken at the first dentist appointment: hypertrophy of the masseter muscle during the period of worsening bruxism, indicated by the red arrows. B) taken during consultation by the psychiatrist, after 1 year of using Lurasidone.

antidepressants and bruxism. After the professional's analysis of the condition, it was found that the emergence of bruxism was compatible with the period of replacement of the antidepressant, Mirtazapine and Escitalopram.

The use of Escitalopram was suspended, and the antipsychotic Lurasidone (20 mg, 1 tablet at night) was prescribed. After 30 days of the new treatment, there was a reduction in pain in the face, in the masseter and trapezius muscles, in addition to a considerable reduction in headaches, alleviating the bruxism that had been spreading.

Two months after starting drug therapy, the symptoms became mild, without the need for medication to control muscle pain and headaches, however the patient continued to use a rigid mouth plate daily to sleep. In 2015, one year after starting treatment with Lurasidone 20 mg (Figure 1B), the patient remained with controlled depressive disorder and a partial reduction in bruxism symptoms.

Over the subsequent years, she used several medications to control her depression, which were sometimes changed, to the detriment of loss of effectiveness. The medications used, however, did not significantly impact the symptoms of bruxism.

The bruxism condition, initially triggered by the use of Escitalopram, never completely resolved, even after discontinuation of the drug.

## DISCUSSION

The condition of bruxism was evidenced after the introduction of Escitalopram, from the SSRI class, for the treatment of depression. The literature suggests that there is a relationship between bruxism and the use of antidepressants, especially SSRIs. The study by Gerber and Lynd [11] reinforces the relationship between this adverse effect and this class of medications. Of the 16 cases of bruxism identified in their literature searches, all were caused by SSRIs, nine cases by the use of Fluoxetine 10-60 mg/ d, four by Sertraline 25-50 mg/d, two by Fluvoxamine 100 mg/d, one by Paroxetine 20 mg/d. Furthermore, they also reported 60 cases of bruxism, reported to Pfizer (Canada), which were associated with sertraline, 7 of which occurred in patients with a previous history of bruxism, and 3 cases involved patients who were taking a neuroleptic concomitantly [11].

Although the SSRI class is highlighted in relation to this adverse effect, other antidepressant drugs can also predispose it, as demonstrated in the analysis by Garrett and Hawley [12] who compiled case reports of 46 patients with bruxism associated with antidepressants. In this study, 10 pharmacological agents were identified: 6 SSRIs were reported (Citalopram, Escitalopram, Fluoxetine, Fluvoxamine, Paroxetine and Sertraline), along with 3 SNRIs (Atomoxetine, Duloxetine and Venlafaxine) and 1 SSRI (Bupropion). The majority of agents reported were SSRIs (74%), followed by SNRIs (24%). The most commonly reported agents were Fluoxetine (12 cases), followed by Venlafaxine and Sertraline (7 cases each) [12]. Another study that corroborates this perspective is research by Uca et al. [13] who, through a cross-sectional case-control trial with 807 participants, concluded that despite the association between bruxism and SSRIs, other medications, which do not include Serotonergic antidepressants, such as Duloxetine and Mirtazapine, are also frequently associated with this disorder.

In fact, there are two hypotheses that permeate the symptoms of bruxism in relation to the use of drugs to control depression, the first of which states that bruxism is a disorder only exacerbated by the medications in question, while the second hypothesis states that the condition can be triggered by the use

of drugs in patients who did not previously have the disorder. These theories can be based respectively on the studies of Stein et al. [14] and Elison and Stanziani [15]. In the work of Stein et al. [14] it is stated that movement disorders can respond to SSRIs, such as the exacerbation of bruxism, since the serotonergic system has been related to preclinical models of repetitive behaviors, including repetitive chewing. In the publication by Elison and Stanziani [15], sleep bruxism was triggered between 2 and 4 weeks after the start of treatment with fluoxetine or Sertraline in four depressed patients selected in a psychiatric clinic with a diagnosis made according to the criteria DSM-III-R. Given that, until the moment of using the medication, the patient had never presented any sign or symptom of bruxism noticed by her or others, it is a clinical condition triggered by the medication.

Another point in the review by Gerber and Lynd [11], which corroborates the theory that the bruxism exposed in the report may have some relationship with the use of the drug Escitalopram, was the period for the signs and symptoms to appear (40 days). The authors noted that the onset of adverse reactions such as the appearance of symptoms related to bruxism varied between 1 day and 11 months after starting monoaminergic drug therapy. The study demonstrated that bruxism was managed by suspending the offending agent, reducing the dose or adding other drugs such as Benztropine, Procyclidine and Buspirone, promoting improvement or complete resolution taking a few days or up to 12 weeks. In the patient's case, the SSRI drug was discontinued, however the signs and symptoms did not completely disappear, they were only alleviated after 8 weeks of using Lurasidone, which belongs to the antipsychotic class [11].

It is worth highlighting that unlike the report by Raja [16], the first in the literature to make a causal link between Escitalopram and sleep bruxism, the patient in this report suffered irreversible damage after using the medication, given that despite the reduction in symptoms after changing the drug to control depression, these symptoms, although milder, are present and are managed with non-drug therapies using a rigid plate for nighttime use.

Therefore, the diagnosis of bruxism associated with antidepressants can be complex, as in the case presented, in which, after the appearance of signs and symptoms, it took around 3 years and 6 months until a correlation was made between the medication and the condition of bruxism. According to Possidente et al. [17], the lack of investigations into the incidence and prevalence of the association between the use of antidepressant medications and bruxism promotes the idea that this correlation is uncommon, providing a series of incorrect diagnoses that make correct treatment for this condition difficult, which is more common than one might imagine.

## CONCLUSION

Depression is a serious medical problem that affects a large number of the population and given its high incidence rates in Brazil and around the world, it is essential to discuss the use of antidepressants for its treatment, as well as their adverse effects. Among the unfavorable effects, bruxism stands out, which is centrally modulated, and although rare, can lead the patient to comorbidities. Therefore, cautious administration and frequent monitoring of each patient are crucial.

There are some hypotheses capable of justifying the persistence of symptoms in a milder form after stopping the use of Escitalopram, however, these need to be further investigated. The most theoretically based thesis states that, after discontinuation of the SSRI drug, the alternative use of other antidepressant drugs, capable of associating with less avidity with different serotonergic receptors, generated mild sensitization

and mild symptoms of bruxism. This theory is justified, as the different drug classes of antidepressant drugs are capable of acting heterogeneously on the subtypes of 5-HT receptors, justifying the discrepancy in the frequency and intensity of extrapyramidal symptoms.

## Collaborators

GB Resende, IS Souza e Silva and LB Oliveira, conceptualization (equal), data curation (equal), formal analysis (equal), funding acquisition (equal), investigation (equal), methodology (equal), project administration (equal), writing- original draft (equal) and writing- review & editing (equal).

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