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# Paroxetine-Induced Bruxism: A Rare Adverse Effect in a Patient Treated for Generalized Anxiety Disorder

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**Abstract** | **Case Report**

Bruxism is defined as an involuntary motor activity characterised by teeth grinding or clenching. It can occur during wakefulness or sleep. Although often multifactorial, certain psychotropic medications – particularly selective serotonin reuptake inhibitors (SSRIs) – have been implicated in its onset, especially paroxetine. We present a case study of a 53-year-old female patient who developed bruxism six weeks after commencing paroxetine treatment for generalized anxiety disorder. The diagnosis was confirmed through clinical and dental examinations. Following a gradual discontinuation of paroxetine, sertraline was introduced as a replacement. This resulted in a complete resolution of bruxism within ten days, without any worsening of anxiety. This adverse effect is thought to result from dopaminergic inhibition secondary to excessive stimulation of 5-HT serotonergic receptors. Although rare, cases of bruxism induced by antidepressant medication must be recognised early. It is essential to closely monitor patients, particularly during the initiation of paroxetine treatment. An individualised approach and therapeutic adjustment generally allow for complete symptom resolution without compromising the patient's psychiatric stability.

**Keywords:** Paroxetine, iatrogenic bruxism, generalized anxiety disorder, side effect, case report.



## INTRODUCTION

Bruxism is defined as the abnormal movement of grinding or clenching the teeth, which may occur during sleep or wakefulness. Daytime bruxism has been demonstrated to be associated with the individual's emotional state, while nocturnal bruxism is recognised as a component of abnormal movement disorders [1,2]. Although frequently of multifactorial origin, bruxism can also be induced by certain psychotropic medications, particularly selective serotonin reuptake inhibitor (SSRI) antidepressants [3].

Paroxetine, in particular, has been associated with rare but well-documented cases of bruxism, suggesting an iatrogenic extrapyramidal effect. This phenomenon is hypothesised to be associated with a functional imbalance between the serotonergic and dopaminergic systems, with excessive stimulation of 5-HT receptors potentially inhibiting nigrostriatal dopaminergic transmission, which is responsible for fine motor control [4,5].

We report here a case of bruxism that developed under paroxetine in a patient with no prior neurological or dental history, with rapid resolution after discontinuation of the treatment. We also discuss possible pathophysiological mechanisms and appropriate management strategies for this type of adverse effect.

## CASE REPORT

The patient is a 53-year-old married woman of urban origin, followed since 2018 for chronic lymphocytic leukemia, currently under hematological monitoring without specific treatment. She had no psychiatric, neurological, or dental history. She was referred to the psychiatry clinic by her oncologist for the management of persistent anxiety symptoms that had been evolving for more than six months. Psychiatric evaluation revealed constant anxious ruminations, diffuse muscle tension, sleep disturbances with difficulty falling asleep, marked irritability, and persistent hypervigilance. Her mood was neutral, with no pathological sadness, suicidal ideation, or delusional symptoms. There was no disturbance in thought

processes, no dissociative symptoms, and no self-image disorders. These findings led to a diagnosis of generalized anxiety disorder (GAD) according to DSM-5 criteria. Treatment with paroxetine was initiated at an initial dose of 10 mg/day, then increased to 20 mg/day after 10 days. Observation after three weeks showed partial improvement in anxiety symptoms, notably better stress tolerance and subjective improvement in sleep quality. However, after six weeks of treatment, the patient reported the onset of morning jaw pain accompanied by nocturnal teeth grinding, as noted by her spouse. She also began to unconsciously and repeatedly clench her jaw during the day. These symptoms were new and had not been present before the introduction of paroxetine. Physical examination revealed bilateral tenderness of the masseter muscles on palpation, without temporomandibular joint abnormality. Dental examination showed early wear of the occlusal surfaces of the molars. The findings were consistent with mixed bruxism (both nocturnal and daytime). Comprehensive laboratory testing and a basic neurological evaluation revealed no abnormalities. The patient was not taking any other medications. There was no personal or family history of bruxism or extrapyramidal/neurological disorders. Her score on the Clinical Bruxism Index (CBI) was 11/21, indicating moderate to severe bruxism induced by paroxetine. A decision was made to gradually discontinue the drug. Paroxetine was stopped over the course of a week and replaced with low-dose sertraline (25 mg/day). Within 10 days after stopping paroxetine, there was complete resolution of bruxism (CBI = 2/21), both nocturnal and daytime. No anxiety relapse was noted during follow-up. Sertraline was well tolerated, with maintenance of the anxiolytic response (GAD-7 = 6/21) and stable clinical status over three months, without recurrence of the motor disorder.

## DISCUSSION

Bruxism is defined as an involuntary motor disorder characterised by repetitive jaw-clenching or teeth-grinding movements. It has been observed to occur during both the day and night. It is classified among movement disorders and can be idiopathic or secondary to neurological, psychiatric, or drug-related causes, notably antidepressants [6].

In this case study, the onset of bruxism occurred after a period of six weeks of paroxetine therapy, with no other confounding factors identified, and resolved rapidly after the discontinuation of the drug. This finding provides substantial support for the drug's causality. According to the Naranjo scale, this adverse reaction can be classified as "probable" (score $\geq$ 6) [7]. The observations presented herein are in alignment with extant literature on the subject. Romanelli et al. described a case of nocturnal bruxism in a young adult treated with paroxetine, with complete resolution after buspirone initiation [8]. In a similar vein, Milanlıoğlu et al. reported a severe case in a 38-year-old female patient, which also improved with buspirone, a partial 5-HT1A receptor agonist, suggesting a role in restoring dopaminergic balance [9]. In a similar vein, Kishi *et al.,* established a comparable correlation in an elderly patient suffering from depression, whose paroxetine-induced bruxism exhibited a positive response to tandospirone, a 5-HT1A receptor agonist [10]. In this case study, the gradual discontinuation of paroxetine resulted in the complete dissolution of bruxism within a period of ten days, without any anxiety relapses. This outcome was facilitated by the transition to sertraline, which was found to be more tolerable. This underscores the necessity for a personalized approach to managing such adverse effects, with therapeutic adjustments based on meticulous clinical evaluation.

Antidepressant-associated bruxism (AB) has been observed to occur with greater frequency in conjunction with SSRIs (selective serotonin reuptake inhibitors) and SNRIs (serotonin-norepinephrine reuptake inhibitors). In a 2018 meta-analysis, the authors identified 46 unique cases of antidepressant-induced bruxism, of which 74% were attributable to SSRIs. Among SSRIs, fluoxetine (26%), sertraline (15%), and paroxetine (13%) were the most frequently implicated agents [11].

The pathophysiology of this phenomenon is likely to be associated with an imbalance between serotonergic and dopaminergic systems. Excessive stimulation of 5-HT receptors, particularly 5-HT2, has been demonstrated to inhibit dopamine release in the striatum, thereby disrupting fine motor control. This functional dopaminergic deficit has been shown to be analogous to that which has been observed in certain extrapyramidal syndromes [12]. This finding aligns with the extrapyramidal nature of drug-induced bruxism, as evidenced in the present study.

Consequently, it is imperative to implement a systematic screening procedure for bruxism, particularly during the initial phase of SSRI therapy. This symptom may be under-recognised by clinicians or unreported by patients, particularly when it occurs during sleep.

## CONCLUSION

Paroxetine-induced bruxism is a rare but clinically significant adverse effect. In our case, its onset after six weeks of treatment, in the absence of other causes, and its rapid disappearance after discontinuation strongly support drug causality. This case highlights the importance of systematically screening for this symptom during SSRI treatment and the need to adjust therapy, when necessary, in order to avoid complications and ensure good treatment adherence.

### Informed Consent

The patient provided written informed consent for the publication of this case report, including

anonymized medical data and the results of psychiatric follow-up.

**Conflict of Interest Statement**

The authors declare that they have no conflicts of interest related to this article.

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