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Psychotropic and Bruxism: About a Clinical Case

Anouar Kaddaf[1*], Zakaria Hammani[2], Mohamed Kadiri[1]

[1]Department of Psychiatry, Military Hospital of Instruction Mohamed V, Rabat
[2]Department of Psychiatry, Military Hospital of My Ismail, Meknes

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***Corresponding author:** Anouar Kaddaf
Department of Psychiatry, Military Hospital of Instruction Mohamed V, Rabat

Abstract **Case Report**

Bruxism is more prevalent among patients who suffer from mental illness than it is among the general population. There are several endogenous factors that might be associated with bruxism. These include personality problems, heredity, neurotransmitter abnormalities (dopamine and serotonin), anxiety disorders, and sleep disorders. A lot of psychotropic drugs, especially those that mess with the dopaminergic, serotonergic, and noradrenergic systems, can cause or make bruxism worse. The purpose of this study is to evaluate the effects of psychotropic medicines on bruxism in order to improve the management of this disorder of the temporomandibular joint in patients who suffer from mental illness. This will be accomplished via the use of a clinical case and a review of the relevant literature.

Keywords: psychotropic drugs, bruxism, antidepressants, antipsychotics, dopaminergic, serotonergic and noradrenergic systems, temporomandibular joint disorders.

INTRODUCTION

Patients with mental illness suffer from bruxism more than the general population. Indeed, several endogenous factors, such as personality disorders, genetics, neurotransmitter disorders (dopamine, serotonin), anxiety disorders and sleep disorders, can be accompanied by bruxism.

Many psychotropic drugs, mainly those that interfere with the dopaminergic, serotonergic and noradrenergic systems, constitute an important factor in the induction or exacerbation of bruxism.

Through a clinical case a clinical case and a review of the literature we will take stock of the effects of psychotropic drugs on bruxism with the aim of improving the management of this disorder of the temporomandibular joint in patients with mental illness.

CLINICAL CASE

Patient aged 46, married and mother of 5 children. His history is marked by right lumbosciatalgia treated effectively 4 years ago, and a single isolated psychiatric consultation 2 years ago for reactive depressive manifestations. The patient had resumed her life normally until the advent of the current episode.

In fact, the patient came accompanied by her daughter for symptoms that had been evolving for 1 month, consisting of semi-mutism with insomnia, slowness, lack of appetite, and at times hetero aggression. Her daughter attributes the appearance of these symptoms to family conflicts. The examination found a patient older than her age with a wrinkled face, conscious, mute, slow, not very expressive, and sad. Communication was difficult, and an attempt to explore the graphics function was inconclusive.

The diagnosis of a depressive state was made, and the patient was put on venlafaxine 75 mg/day and hydroxyzine 25 mg/day.

The evolution was marked by a marked improvement in mood, appetite and sleep, however, despite her effort to communicate, the patient remained silent. In fact, the patient made her inability understood, tried to communicate, to express that she had improved but that she could not open her mouth, and that she was suffering from mandibular pain. Her daughter had reported that they found it difficult to feed her, with the notion of a difficult, upset and painful opening of the mouth which set in 1 month after the start of treatment.

A maxillofacial opinion had highlighted on the CT image of the facial mass, a secondary centered bruxism at the origin of the mandibular blockage. This

secondary bruxism can be caused by taking psychotropic medications. Venlafaxine was stopped and the evolution was marked by the gradual resolution of bruxism.

DISCUSSION

Bruxism is the abnormal movement of grinding or clenching the teeth that may occur during sleep or wakefulness. Daytime bruxism is related to the emotional state of the person, while nighttime bruxism is considered a part of abnormal movements.

Psychotropic drugs are directly involved in the induction, exacerbation, but also improvement of bruxism. The authors implicate the dopaminergic, serotonergic and noradrenergic systems in the induction of bruxism [1].

As in our patient, the incrimination of venlafaxine in bruxism has been well described in the literature [2]. Indeed, all non-tricyclic antidepressants, especially SSRIs, but also venlafaxine and bupropion, are implicated in the induction or aggravation of bruxism because their serotonergic effect can mimic dopaminergic antagonism. [3].

This effect on bruxism could be managed by the addition of propranolol or a serotonergic partial agonist such as buspirone or tandospirone which are two serotonergic partial agonists [3].

Bruxism induced by fluoxetine and sertraline was first mentioned in 1993. Citalopram was mentioned later in the literature with a dose-dependent relationship (from 40 mg/day) [3, 4].

Tricyclic antidepressants do not seem effective in reducing bruxism, but can improve patients' symptoms because of their analgesic effect, their effectiveness in improving the quality of sleep and their effect on reducing stress perceived by the subjects suffering from bruxism [1-3].

The first cases of bruxism induced by antipsychotics were described in 1993 in eight patients who manifested daytime bruxism, long after the start of their treatment [3].

Classic antipsychotics are involved in the induction of bruxism, because of their dopaminergic antagonism. Bruxism that occurs directly after initiation of treatment with an antipsychotic could be linked to acute dystonia. [3].

Finally, psychotropic drugs that act as gamma-amino-butyric acid (GABA) agonists, such as clonazepam, are effectively used in the treatment of bruxism [3-5].

CONCLUSION

Patients with psychiatric pathologies frequently present temporomandibular joint disorders such as bruxism. When treating these patients, the effect of psychotropic drugs on the induction or exacerbation of bruxism should be considered. Typical antipsychotics and SSRI-type antidepressants are the psychotropic drugs most implicated in the induction or exacerbation of bruxism.

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