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BiteStrip analysis of the effect of fluoxetine and paroxetine on sleep bruxism

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ABSTRACT

Objective: The relationship between sleep bruxism (SB) and selective serotonin reuptake inhibitors (SSRIs) is still under debate because of the lack of well-designed objective studies. The current study investigates possible effects of SSRIs, fluoxetine, and paroxetine on SB in patients with anxiety and depression.

Materials and methods: Thirty users of SSRIs for treatment of depression or anxiety were enrolled in the study. After clinical and anamnestic examination, 15 fluoxetine and 15 paroxetine users were included. For an objective evaluation of SB, a single-use disposable home screening device, BiteStrip, was used prior to the first SSRI intake and was repeated on the 7th and 15th days. Patients' self-reported data also were obtained for assessment of patient awareness.

Results: BiteStrip scores were significantly higher on the 7th and 15th days than the first measurement ($p < 0.01$). There was an increase in 26 (86.6%) patients' bruxism scores on the 7th day. There was also an increase in 27 (90%) patients' bruxism scores on the 15th day. But according to patients' self-reports, only 6 patients had an awareness that bruxism symptoms were initiated or exacerbated 15 days after starting fluoxetine or paroxetine.

Conclusion: Fluoxetine and paroxetine, SSRIs used for the treatment of anxiety and depression, may initiate or aggravate SB. Clinicians should consider that SSRIs may be the cause of SB when SSRI users are referred to dental clinics for SB symptoms. As there is a shortage of researches on this subject, further studies are necessary to confirm the existence of SSRI-induced SB.

1. Introduction

Sleep bruxism has been defined as a movement disease characterized by involuntary grinding or clenching of the teeth in sleep affecting approximately 8–21% of the population. This abnormal movement during sleep may cause severe destruction to oral and maxillofacial structures, such as the teeth, temporomandibular joint (TMJ), and masticatory muscles (Ahlberg et al., 2008; Kara et al., 2012).

Despite the great number of published researches, the actual etiology of SB has not yet been investigated. Although numerous types of theories have been suggested, the most rational speculation among them is the multifactorial model in which pathophysiological and psychosocial are considered to be the primary causative factors and morphological and peripheral factors are considered to play a small role (Lobbezoo, Van Der Zaag, & Naeije, 2006). Lastly, the main research area for investigators is the central dopaminergic system, but other factors including sleep disorder, chronic use of cigarettes and alcohol, and psychosocial factors involving personality and emotional stress also are thought to be precipitating factors in SB (Bayar, Tutuncu, & Acikel, 2012). Beside these, recent case reports (Bostwick & Jaffee, 1999; Kishi, 2007; Milanlioglu, 2012; Oulis, Dimitrakopoulos, Konstantakopoulos, Tsaltas, & Kollias, 2012; Sabuncuoglu, Ekinci, & Berkem, 2009; Soyata & Oflaz, 2015) have proposed that selective serotonin reuptake inhibitors (SSRIs) may constitute a different etiologic factor for SB through inducing or aggravating involuntary jaw movement during sleep.

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SSRIs are used widely for the treatment of depression and also have been popular for the treatment of anxiety due to their safety and effectiveness. However, despite their successful outcome, SSRIs may lead to some complications, including obstructive sleep apnea, restless legs syndrome, sleepwalking, and sleep bruxism (Drapier et al., 2016). SSRIs may evoke extraordinary serotonergic activation on the mesocortical neurons, a consequence of dopaminergic deficiency, which may lead to a particular form of akathisia-like movement of the jaw muscles, thus resulting in bruxism (Milanlioglu, 2012).

There is no consensus in the literature of the relationship between bruxism and SSRIs due to the lack of controlled, objective studies. Although a limited number of all case reports (Bostwick & Jaffee, 1999; Kishi, 2007; Milanlioglu, 2012; Oulis et al., 2012; Sabuncuoglu et al., 2009; Soyata & Oflaz, 2015) suggested SSRIs induced or aggravated SB, one study (Hermesh et al., 2015) asserted that no relationship between SSRIs and SB and another study (Lobbezoo, van Denderen, Verheij, & Naeije, 2001) demonstrated only 3.2% of SSRI users have encountered SB, based on family physicians' reports.

SSRIs have similar characteristics, but their pharmacological properties differ. For this reason, the capability of different SSRIs to stimulate unwanted adverse effects, such as bruxism, may differ, and SSRIs have to be examined individually in terms of side effects (Drapier et al., 2016). This study concentrates on fluoxetine and paroxetine since fluoxetine is thought to be the foremost antidepressant in the SSRI group and paroxetine has been awarded full marketing acceptance due to its anxiolytic characteristic. This study evaluates possible effects of fluoxetine and paroxetine on SB in patients diagnosed with depression and anxiety.

2. Materials and methods

The study was designed in the Department of Oral and Maxillofacial Surgery, Faculty of Dentistry, Izmir Katip Celebi University, Izmir, Turkey. Permission for the study was obtained from the Human Ethical Committee of Izmir Katip Celebi University. Detailed explanations related to the study were given to all the participants, and written informed consent was provided by all volunteers before starting the study.

This was a multidisciplinary, clinical study. The participants were males and females and were over the age of 18. Thirty patients prescribed paroxetine or fluoxetine for the first time due to diagnosis of mild or moderate depression or an anxiety disorder were enrolled in the study. The presence of depression or anxiety was diagnosed by a psychiatric specialist at the Government Hospital of Karsıyaka in a short diagnostic interview using the State-Trait Anxiety Inventory (STAI) I and II and Beck Depression Inventory (BDI) tests. The STAI is a self-report psychometric test that evaluates the patient's perceptions and sensory experiences and aims to measure both state and trait anxiety. This test involves 40 questions regarding anxiety. To assess the severity of depression and characteristic attitude, the BDI was applied. The BDI has 21 items, with each question receiving a score of 0–3. An increase in scores shows an increase in depression.

Patients prescribed paroxetine or fluoxetine were sent to our clinic for an assessment of whether they were suitable for inclusion in the study. Patients who had serious medical problems and major psychiatric diseases or presence of neurological disorders, including Parkinson's disease, Alzheimer's disease, or epilepsy, were excluded from the study. Also excluded were patients reporting heavy smoking, alcohol or drug abuse, pregnancy or breast-feeding, and a history of sleep problems. Patients who had moderate or severe bruxism, as evaluated by the clinical evaluation and patients' history, more than two missing posterior teeth, or severe occlusal problems also were not included in the study.

An examination was done to determine existing SB prior to the study. To determine existing bruxism, patients were assessed through clinical examination and their medical history according to the criteria of the American Sleep Disorder Association (ASDA). Participants were considered to have existing bruxism if they had a history of grinding or clenching their teeth at least five nights a week, as reported by a sleeping partner or family member, and who had at least one of the following clinical symptoms: common masticatory muscle hypertrophy, stiffness, discomfort, fatigue, pain (particularly in the morning), abnormal tooth wear, and shiny spots on dental restorations. In the current study, each patient served as his or her own control.

A single-use disposable home screening device (BiteStrip) was used by each participant who was not diagnosed with bruxism after clinical examination. The BiteStrip has two electromyographic electrodes that detect the presence and frequency of bruxism, is similar to surface electromyography (EMG), and includes a computer chip that records the number of times the masseter muscle contracts throughout a 5-h sleep period. This device was applied to the patient's cheek (left masseter region). The patients were given exhaustive information according to the manufacturer's instructions. All patients also received a CD with a step-by-step video explaining the proper use of this device.

According to the manufacturer, the device scores were indicated as follows: 0 = very low (less than 30 contractions), 1 = mild (between 31 and 60 contractions), 2 = moderate (between 61 and 100 contractions), 3 = severe (more than 100 contractions), and E = error (no maximal voluntary clenches detected or no skin conductivity). Patients with E outcomes were renewed by another device for a subsequent night.

The BiteStrip device was applied before the fluoxetine or paroxetine use (T1), and it was repeated 7 (T2) and 15 days (T3) later to assess changes in bruxism. In order to determine if patients had any SB symptoms, patient self-reports were obtained before using fluoxetine or paroxetine and only 15 days later for a subjective assessment of bruxism changes in the volunteers.

2.1. Statistical analysis

Because there was no similar study found in the literature, the first 10 observations were considered as a pilot sample. Based on the pilot sample of 10, the effect size (Phi coefficient = 0.55) for a chi-square test with 2° of freedom indicated that a minimum sample size of 27 would yield at least 80% statistical power. Considering possible drop-outs, a sample size of 30 was determined for the study.

The data analysis used the SPSS (SPSS 14.0; SPSS, Chicago, IL, USA) software program. For all the analyses, based on the nominal variables, Pearson chi-square tests were implemented. The Phi coefficient for each analysis was considered to be effect sizes. The column proportions were compared using Z-tests regarding measurement times. The descriptive statistics were reported as counts and percents in tables. A p value of 0.05 was considered statistically significant. Control confounders for SB were not considered due to the single cohort before-and-after design of this study, in which each volunteer acted as his or her own control (Tables 1 and 2).

3. Results

For obtaining 30 participants for this study, 227 patients with depression and anxiety who had been prescribed SSRIs, fluoxetine or paroxetine, for the first time were examined. A total of 118 patients were excluded because of additional prescribed drugs beside the SSRIs. After clinical and anamnestic examination, 53 patients also were excluded due to existing bruxism symptoms.