Original Article

The Prevalence of Bruxism and Associated Factors Among Patients with Schizophrenia in Istanbul, Türkiye: A Cross-Sectional Study

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ABSTRACT

Background: Schizophrenia is a severe and chronic neuropsychiatric disorder that involves profound impairment of psychopathology in cognition, emotion, perception, and other aspects of behavior. Factors, such as the nature of the disease, length of hospital stay, duration of illness, and side effects of psychotropic drugs, may contribute to poor oral health and the risk of developing bruxism in patients with schizophrenia. **Aim:** To evaluate the prevalence of bruxism and associated factors in patients with schizophrenia. **Methods:** This cross-sectional study was conducted in a single center with 211 patients with schizophrenia. Study participants were graded according to "probable" bruxism based on positive clinical inspection, with or without a positive self-report. The type of antipsychotic treatment used in participants was evaluated in three categories: typical antipsychotic monotherapy, atypical antipsychotic monotherapy, and a combination of both. Binary logistic regression models were used to evaluate associations between probable bruxism and different factors. **Results:** The mean age of the study participants was 51.02 ± 9.29 years, and 112 (52.5%) were males. Probable bruxism was identified in 87 (41.2%) of the study participants. Younger age (AOR = 0.88, 95% CI = 0.838–0.928, $P < 0.001$), higher duration of illness (AOR = 1.50, 95% CI = 1.278–7.545, $P < 0.001$), and combination antipsychotic therapy (AOR = 3.042, 95% CI = 1.278–7.545, $P = 0.015$) were significant factors associated with probable bruxism among patients with schizophrenia on treatment. **Conclusion:** The relatively high prevalence of probable bruxism in patients with schizophrenia and its relation to antipsychotics was observed. There is a need for more research on the causes and treatment of bruxism in schizophrenia.

KEYWORDS: *Antipsychotic, atypical, bruxism, combination, monotherapy, schizophrenia, typical*

INTRODUCTION

Schizophrenia is a complex, heterogeneous, behavioral, and cognitive syndrome that results from a disruption in brain development caused by genetic or environmental factors or both. It is a severe mental disorder that has profound effects on both individuals and society.[1] Patients with schizophrenia, one of the often neglected disease groups, are at risk for oro-dental disease, temporomandibular disorders, and bruxism possibly due to both the emotional distress they experience and the side effects of psychotropic drugs.[2-5]

Bruxism is a repetitive masticatory muscle activity characterized by tooth clenching, grinding, or bracing or thrusting of the mandible, occurring during sleep (sleep bruxism) or wakefulness (awake bruxism). These are phenomena regulated by the central nervous system, of multifactorial origin, with peripheral factors playing a

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secondary role.[6] Recent literature defines bruxism as periodical movement of the lower jaw, which does not necessarily have to be pathological.[7] Primary bruxism has no identifiable biopsychosocial cause; secondary bruxism is associated with neurological disorders or considered an adverse effect of drugs.[8] The drugs that cause bruxism act mainly through dopamine, serotonin, norepinephrine, and histamine.[9] These medications alter the receptors of various neurotransmitters and modulate their responses.[10] There is literature on drug-induced bruxism, but it is still an under-recognized problem in dentistry.[9]

The most clinically relevant dopamine antagonists are antipsychotic drugs used to treat a wide range of psychiatric disorders, including schizophrenia, depression, anxiety, and dementia. Antipsychotics, the mainstay of schizophrenia treatment and inhibit dopamine receptors, are known to cause extrapyramidal symptoms and tardive and oral dyskinesia, but their effects on bruxism are contradictory.[9-11] The literature is substantial but controversial, and based mostly on anecdotal case reports.[12-14] Therefore, it is clear that there are insufficient evidence-based data to draw firm conclusions about antipsychotic drugs that trigger or aggravate bruxism.[15]

The relationship between schizophrenia and bruxism is not as well-known as other psychiatric disorders, because of the psychosocial factors in the etiology of bruxism that are generally associated with depression, anxiety disorders, and somatization.[16,17] In addition, dentists do not adequately recognize psychotic disorders. The study aimed to assess the prevalence of probable bruxism and associated factors in patients with schizophrenia who are on treatment.

Methods

This cross-sectional study was conducted over a period from September 2019 to January 2020 at the Training and Research Hospital for Psychiatry, Neurology, and Neurosurgery. The study population included inpatients with a diagnosis of schizophrenia according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria.[18] Inclusion criteria for study participants were as follows: age over 18 years, maintained antipsychotic treatment, presence of natural teeth (at least four teeth on each sextant), no history of alcohol or substance use, no history of facial or cervical injury, and no history of general neurologic disturbances, neoplasm. Other dental exclusion criteria were as follows: the presence of a removable prosthesis or extensive prosthetic restoration, fixed orthodontic treatment, the presence of TMDs requiring treatment, and the presence of a gross malocclusion (i.e., open anterior bite).

Clinical examination

The diagnosis of probable bruxism was based on a positive clinical inspection, with or without a positive self-report.[6] Permission was obtained from the authorities, and clinical examinations were conducted at the hospital wards. For examination, one or more of the subsequent were considered: severe wearing of the teeth, jaw muscle discomfort, transient morning jaw muscle pain or fatigue, temporal headache, jaw locking upon awakening, or masseter muscle hypertrophy caused by voluntary forceful clenching. The presence of occlusal tooth wear to at least the extent of dentin exposure was measured.[19] The highest score was noted for each sextant. History and clinical examinations were performed by a single examiner (Ö.O. Prosthodontist) throughout the study. Reliability studies were conducted using a second assessor (G.M. Prosthodontist) who was blinded to group allocation. The second assessor separately examined randomly selected subjects from this study sample using the same protocols.

Ethical approval

The Ethics Committee of Bakirkoy Prof. Mazhar Osman Training and Research Hospital for Psychiatry, Neurology, and Neurosurgery, Istanbul-Türkiye, approved the study protocol (number: 31066). The approval conforms to the provisions of the Declaration of Helsinki (version 2008). The study participants were fully informed of the nature of the study and provided verbal and written consent for maturation. Research data were stored on password-protected computers. Access to study data was restricted to only a few members of the study team.

Statistical examination

Data were analyzed using the Statistical Package for Social Sciences (SPSS) for Windows (version 26.0, SPSS Inc. Chicago, IL, USA). Kappa statistic was used to measure the interobserver agreement for probable bruxism. The Shapiro-Wilk test was applied to test if the data were normally distributed. A comparison of continuous variables between the groups was conducted using the Mann–Whitney U test for nonparametric distribution. To compare categorical data, a Chi-square test was performed. In addition, binary logistic regression analysis was conducted to analyze the relationship between bruxism and different factors among the study sample. The adjusted odds ratios (AOR) with confidence intervals (CI) were calculated. *P* values less than 0.05 were considered statistically significant.

Results

Table 1 summarizes the demographic and clinical characteristics of the study population. A total of

211 patients with schizophrenia, with a mean age of 51.02 ± 9.29 years, comprising 99 females (49.2%) and 112 males (50.8%) were included in this study. These patients were being treated in the chronic clinics of the hospital for an average of approximately 13.5 years, and the duration of illness was 15.64 ± 7.32 years. Of the 211 patients with schizophrenia, 56 (25.6%) were taking general medications: hematology (1.9%), endocrine (9%), neurology (2.4%), cardiovascular (12.3%), and respiratory (2.8%).

Table 1: Clinical and demographic characteristics of the study participants

Study sample, *n*=211		
Variable	**Mean±SD**	**Min-max**
Age, yrs	51.02±9.29	24-71
Duration of illness, yrs	15.64±7.32	1-38
Length of hospitalization, yrs	13.47±8.27	1-38
Number of psychotropic drugs	2.60±0.97	1-5
Variable	***n***	**%**
Sex		
Male	112	53.1
Female	99	46.9
Type of antipsychotic treatment		
Typical antipsychotic monotherapy	98	46.5
Atypical antipsychotic monotherapy	72	34.1
Combination antipsychotic therapy	41	19.4
Smoking habits	133	63.0
Systemic disease	54	25.6
Antidepressants	10	4.7
Anxiolytics or mood stabilizers	26	12.3
Anticholinergic drugs	143	67.8
Benzodiazepines	42	19.9

SD: standard deviation, yrs: years, min-max: minimum-maximum, *n*: number, %: percentage

The types of antipsychotic treatments used by participants with schizophrenia were analyzed in three categories: typical antipsychotic monotherapy (46.5%), atypical antipsychotic monotherapy (34.1%), and a combination of both (19.4%). Some of the patients with schizophrenia receiving antipsychotic treatment were using additional adjuvant antidepressants (4.7%), anxiolytics or mood stabilizers (12.3%), anticholinergic drugs (67.8%), and benzodiazepines (19.9%) [Table 1].

Probable bruxism was identified in 87 (41.2%) of the study participants. The presence of probable bruxism in the interobserver examination indicated substantial to almost perfect agreement between them, as assessed by the Kappa coefficient (0.73 to 0.81). For analysis, the study participants were categorized into two groups, with and without bruxism. Table 2 presents statistical comparisons of the clinical and demographic characteristics of the study participants with and without bruxism. The two groups were statistically compared for age, sex, number of psychotropic drugs used, duration of illness, length of hospitalization, smoking, the presence of systemic disease, the types of antipsychotic treatment, and adjunctive psychotropic drugs [Table 2]. Based on the results of bivariate comparisons, no

Table 2: Comparisons of the different variables in the study participants with and without bruxism

Variable	**Schizophrenia patients without bruxism *n*=124, 58.8%**		**Schizophrenia patients with bruxism *n*=87, 41.1%**		***P***
	Mean±SD	**min-max**	**Mean±SD**	**min-max**	
Age, yrs	51.88±8.07	28-67	49.80±10.72	24-71	†0.050
Duration of illness, yrs	14.91±7.54	1-38	16.67±6.91	3-30	†0.060
Length of hospitalization, yrs	13.70±8.47	1-38	13.15±8.03	1-30	†0.726
Number of psychotropic drugs	2.54±0.90	1-5	2.68±1,05	1-5	†0.505
Variable	***n***	**%**	***n***	**%**	***P***
Sex					
Male	63	50.8	49	56.3	‡0.429
Female	61	49.2	38	43.7	
Type of antipsychotic treatment					
Typical antipsychotic monotherapy	60	48.4	38	43.7	‡0.037*
Atypical antipsychotic monotherapy	47	37.9	25	28.7	
Combination antipsychotic therapy	17	13.7	24	27.6	
Smoking habits	83	66.9	50	57.5	‡0.161
Systemic disease	37	29.8	17	19.5	‡0.092
Antidepressants	7	5.6	3	3.4	‡0.460
Anxiolytics or mood stabilizers	13	10.5	13	14.9	‡0.332
Anticholinergic drugs	83	66.9	60	69.0	‡0.756
Benzodiazepines	28	22.6	14	16.1	‡0.245

SD: standard deviation, yrs: years, min-max: minimum-maximum, *n*: number,%: percentage, †Mann-Whitney *U* test, ‡Pearson's Chi-square test, *statistically significant difference at the 0.05 level

Table 3: Results of binary logistic regression analysis

Dependent variable	Independent variables	ß	SE	AOR (95% CI)	*P*
Probable bruxism: No/Yes	Sex	-0.141	0.360	0.869 (0.429-1.760)	0.696
	Age	-0.126	0.026	0.882 (0.838-0.928)	<0.001*
	Duration of illness	0.406	0.073	1.500 (1.300-1.732)	<0.001*
	Length of hospitalization	-0.271	0.056	0.762 (0.683-0.851)	<0.001*
	Number of drugs	0.005	0.182	1.005 (0.704-1.436)	0.977
	Type of antipsychotic treatment				0.049*
	Typical antipsychotic monotherapy				
	Atypical antipsychotic monotherapy	0.325	0.376	1.384 (0.662-2.895)	0.388
	Combination antipsychotic therapy	1.113	0.458	3.042 (1.239-7.471)	0.015*
	Smoking habit	-0.623	0.358	0.537 (0.266-1.083)	0.082
	Systemic disease	-0.360	0.405	0.698 (0.315-1.544)	0.374

Reference group: Schizophrenia patients with bruxism, ß: beta coefficient, SE: standard error, AOR: adjusted odds ratios with 95% confidence intervals (CI), * statistically significant difference at the 0.05 level

statistically significant differences were identified in demographic and clinical variables between the patients with schizophrenia with and without bruxism, except for antipsychotic treatment regimens (P = 0.037). Patients with schizophrenia who were on a combination of typical plus atypical antipsychotics had a significantly higher prevalence of bruxism compared to patients on typical monotherapy (P = 0.031) or atypical monotherapy (P = 0.01) [Table 2].

Binary logistic regression analysis showed that younger age (AOR = 0.88, 95% CI = 0.838–0.928, P < 0.001), more prolonged duration of illness (AOR = 1.50, 95% CI = 1.278–7.545, P < 0.001), and combination antipsychotic therapy (AOR = 3.042, 95% CI = 1.278–7.545, P = 0.015) were significant factors associated with the probable bruxism among patients with schizophrenia in treatment [Table 3].

Discussion

This study focused on the prevalence of probable bruxism and associated variables in patients with schizophrenia in treatment. The main finding was a relatively high prevalence of probable bruxism among the study participants. In addition, the presence of probable bruxism was significantly associated with the type of antipsychotic treatment used, age of the patient, and duration of illness.

The prevalence of probable bruxism in this study was 41.2%. Severe dental attrition has been reported as bruxism in previous studies.[3-5,20] These studies reported high prevalence of bruxism in psychiatric patients, the majority of whom had schizophrenia.[3,4,20] Rekha *et al.*[20] reported significantly higher degree of abnormal dental attrition among psychiatric patients compared to control (52.8% vs. 18.6%). Winocur *et al.*[3] also reported severe attrition in 46.8% of the psychiatric patients compared with 20% in the controls. Significant differences in mean muscle sensitivity to palpation, temporomandibular joint sensitivity to palpation, and range of mouth opening were also observed. Another study by Gurbuz *et al.*[4] in Türkiye concluded that the prevalence of severe tooth wear was significantly higher in patients than in controls (39.2% vs. 21.2%). In this study, duration of illness was found to be a significant factor for the presence of probable bruxism in study sample. This association can be interpreted as the cumulative effect of both the disease and its prolonged treatment with the use of psychotropic drugs on bruxism. These observations support the idea that the central mechanism of bruxism in patients with schizophrenia may not be similar to the general population. The existence of a mental disorder may act as a trigger point for deregulation in the central nervous system.[21]

According to our findings, using typical plus atypical antipsychotic combination therapy was a significant factor in the presence of probable bruxism among participants. Patients with schizophrenia who were treated with typical monotherapy or atypical monotherapy had a lower prevalence of bruxism compared to patients treated with a combination of both. Several interpretations on this issue can be suggested, such as a relationship between the higher prevalence of acute extrapyramidal side effects, which are commonly seen with the combined use of typical and atypical antipsychotic compared to the use of these drugs separately or a possible effect of the interaction between the two drugs in a combinatorial treatment.[22] The nigrostriatal dopamine pathway may have been activated upon drug-drug interaction, which may have increased the prevalence of bruxism in patients using a combinatorial treatment.[23]

Contrary to the current paradigm, no significant difference was identified in the prevalence of probable bruxism between the study participants on typical

monotherapy versus those on atypical monotherapy. Previously published case reports suggest that long-term treatment with typical antipsychotics may cause permanent changes in the brain's dopaminergic pathways.[24] In contrast, it has been reported that patients who developed awake bruxism after exposure to typical antipsychotics can be successfully treated with atypical antipsychotics.[12] Moreover, a recent meta-analysis indicated that typical antipsychotic or atypical antipsychotic does not affect sleep bruxism, while atypical can reduce awake bruxism, and typical antipsychotic can increase awake bruxism.[10] Our findings showed that atypical antipsychotic monotherapy or typical antipsychotic monotherapy was not associated with probable bruxism in the study participants, but the prevalence of bruxism in participants using typical antipsychotic monotherapy was higher than that in those using atypical monotherapy and lower than that in those using combination antipsychotic treatment. Atypical antipsychotics are considered to be less likely to cause bruxism in patients and may even have more therapeutic benefits compared to typical antipsychotics.[9-11,25] The antagonistic effect of atypical antipsychotics on 5-hydroxytryptamine (5-HT) receptors may contribute toward their therapeutic benefits.[11] Drugs that inhibit serotonergic neurotransmission, such as atypical antipsychotics, suppress bruxism by the reverse mechanism, normalizing the activity of the mesocortical pathway.[12] Regional differences in dopamine receptor pharmacology have been suggested as the reason for bruxism in both hyper- and hypodopaminergic states.[26] The literature suggests that the relationship between dopaminergic drugs and bruxism may be complicated by multiple dopamine-related circuits that can trigger or suppress if altered in one or the other direction.[10] From another point of view, it can be interpreted as the presence of schizophrenia being the determinant factor for the increase in parafunction rather than an influence of drugs on the central nervous system. In other words, schizophrenia may make the patients more susceptible to the development of extrapyramidal movements that originate from or are exacerbated by the medication. Bruxism could also be an unusual manifestation of dyskinesia in vulnerable individuals with schizophrenia. There is a need for further studies to evaluate the etiology of the development of bruxism in patients with schizophrenia.

This study has some limitations. The cross-sectional design of the included studies weakens the level of inference that can be drawn. In addition, the diagnosis of bruxism in this study was based on patient history, self-reporting, and clinical evaluation. Bruxism can be diagnosed definitively by electrophysiological tools, such as polysomnography or an ambulatory recording system, but it cannot be easily applicable to this study group, suggesting concerns about the diagnostic accuracy of the method used in this study. In addition, we were unable to distinguish sleep bruxism from awake bruxism in this study, which can be considered another important limitation. Even though sleep bruxism and awake bruxism are considered different entities, a co-occurrence of up to 20% has been reported pointing toward shared etiology.[27] In addition, the dosage of medication used was not queried in this study; therefore, analysis of a dose-response association with probable bruxism was not feasible. Despite these limitations, the results of this study are important in the field, as it is one of the few studies that assess the prevalence of probable bruxism and associated factors among patients with schizophrenia in treatment.

Conclusion

In conclusion, the relatively high prevalence of probable bruxism in patients with schizophrenia and its relation to antipsychotics deserves attention. From a bruxism perspective, monotherapy with atypical or typical antipsychotic seems to be superior to atypical plus typical antipsychotic combination therapy. Further studies are needed to evaluate the possible relationship between schizophrenia, its treatment, and bruxism.

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Ethical approval

The protocol for this research project has been approved by the ethics committee of the study and by Bakirkoy Prof. Mazhar Osman Research and Training Hospital for Psychiatry, Neurology, and Neurosurgery in Istanbul/Türkiye (B10.4. ISM 04.34.26.08-113, number: 31066). The approval conforms to the provisions of the Declaration of Helsinki (version 2008).

Informed consent

After providing participants with a complete description of the study, informed consent was obtained from each participant or from their legal guardians for individuals under guardianship.

Data availability

The data supporting this study's findings are available from the corresponding author on reasonable request.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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