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ORIGINAL ARTICLE

Psychopathological profile of patients with different forms of bruxism

Gurkan Rasit Bayar · Recep Tutuncu · Cengizhan Acikel

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Abstract The aim of the current study was to evaluate the prevalence of psychopathological symptoms in patients who self-reported different forms of bruxism by means of clinical and anamnestic diagnostic criteria. Eighty-five participants were divided into four groups as sleep bruxers (12), awake bruxers (24), sleep–awake bruxers (33), and non-bruxers (16). A self-report symptom inventory questionnaire (Symptom Checklist-90—Revised (SCL-90-R)) was filled out by all groups to determine their psychopathological symptoms. As regards mean psychopathological scores, patients with sleep–awake bruxism endorsed the highest scores. In addition, patients with awake bruxism showed higher scores than patients with sleep bruxism and non-bruxism in most SCL-90-R subscales. Kruskal–Wallis test revealed significant differences between groups in any of the SCL-90-R subscales, except for the psychoticism subscale. Mann–Whitney test followed by Bonferroni's test correction between non-bruxer and sleep–awake bruxer groups revealed significant differences in depression, anxiety, hostility, phobic anxiety, paranoid ideation, global severity index, positive symptom distress index, and positive symptom total in all SCL-90-R subscales. Statistical analysis of our study showed that differences between groups were significant in all SCL-90-R subscales except for the psychoticism subscale. Better distinction of bruxism forms may help to develop new treatment strategies for bruxism disorder.

Keywords Bruxism · Form · Etiology · Psychopathology · Symptom checklist (SCL-90-R)

G. R. Bayar
Gulhane Military Medical Academy Department of Oral and Maxillofacial Surgery,
Ankara, Turkey

R. Tutuncu
Etimesgut Military Hospital Department of Psychiatry,
Ankara, Turkey

C. Acikel
Gulhane Military Medical Academy Department of Epidemiology,
Ankara, Turkey

G. R. Bayar (✉)
Gulhane Askeri Tip Akademisi, Agiz, Dis,
Cene Hastaliklari ve Cerrahisi AD,
Etlik,
06018 Ankara, Turkey
e-mail: gurkanbayar@yahoo.com

Introduction

Bruxism is a stereotyped oral motor disorder characterized by awake and/or sleep-related grinding and/or clenching of the teeth [1, 2]. It is considered the most detrimental among all the parafunctional activities of the stomatognathic system, being considered a risk factor for temporomandibular disorders (TMD), and in particular for myofascial pain [3]. Furthermore, tooth grinding is an activity of major concern to dentists because of its consequences: tooth destruction; damage on periodontium, temporomandibular joints, and masticatory muscles; breakage of dental restoration; or rehabilitation and grinding sounds that may interfere with the sleep of family or life partners [4–8]. Its etiology is multifactorial and many etiological theories have been proposed over the years. In the past, peripheral factors like occlusal discrepancies and deviations in orofacial anatomy have been considered the main causative factors for bruxism. Nowadays, such factors are known to play only a minor role, if any. Recent focus is more on central factors. Psychosocial factors like stress and certain person-

ality characteristics are frequently mentioned in relation to bruxism [9].

Recent studies and reviews about bruxism pointed toward the possibility that awake and sleep bruxism have to be considered two different disorders [1, 10–12]. It has been shown that bruxism during wakefulness is likely to be a result of emotional tension or psychosocial disorders that force the subject to respond with a prolonged contraction of his/her masticatory muscles [1]. On the other hand, it has been repeatedly demonstrated by polysomnographic (PSG) and/or electromyographic (EMG) studies that sleep bruxism is the part of a complex arousal response of the central nervous system [1, 2, 13–15], which occurs during changes in sleep depth and is accompanied by gross body movements, the appearance of K complexes in the electroencephalogram, an increased heart rate, respiratory changes, peripheral vasoconstrictions, and increased muscle activities [1].

Sleep and awake bruxisms are difficult to clinically distinguish between; [1, 16] similarly, a clearer distinction between detected bruxism and perceived bruxism should be made [1, 17]. Unfortunately, bruxism as a pathophysiological entity can only be detected by means of polysomnographic recordings, the use of which is limited by the high costs and the low number of adequately equipped sleep laboratory [1, 18].

The aim of the current study was to evaluate the prevalence of psychopathological symptoms profile in patients who self-reported different forms of bruxism by means of clinical and anamnestic diagnostic criteria.

Materials and methods

Sample size calculation

To determine the appropriate sample size needed to detect a significant effect, a priori calculation of the sample size necessary for this investigation was performed with the G*Power Version 3.1.0 computer program [36].

The values of types I and II errors were set at 0.05 and 0.20, respectively. Data about the estimated variance were drawn from another work in the literature [19, 20]. The difference to detect has been identified in a 40% difference between groups in mean "Symptom Check List-90—Revised" (SCL-90-R) scores. In consideration of that, to have an 80% statistical power and a 40% effect size, the needed total sample size was about 76 subjects.

Study design

This study included participants who self-reported different forms of bruxism. Participants were consecutively selected among patients attending the section of dentistry clinics, Guzelyali Military Hospital, Air Force Technical School Health Department Izmir, and Etimesgut Military Hospital Ankara, Turkey, for conservative care. Criteria for exclusion from the study were: presence of Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) Axis I Group I diagnosis of muscle disorders and/or Group III diagnosis of arthralgia or osteoarthritis; [3, 21] presence of neurological disorders and use of medications influencing sleep or motor functions; presence of chronic pain in other areas of the body; presence of rheumatic disorders; chronic use of medications for any psychopathologic disorders; and history of recreational drug use in the 6 months before study [3, 21–25]. Eighty-five subjects (68 males, 17 females, and mean age 26.8) satisfied inclusion/exclusion criteria.

Participants were clinically investigated for the presence of bruxism forms and were assessed for the presence of psychopathologic symptoms profile by means of a self-report questionnaire (SCL-90-R). Research was approved by the Ethic Committee of the Etimesgut Military Hospital Ankara, Turkey, and all subjects signed a consent module prior to the start of the study.

Clinical assessment

All subjects underwent a clinical examination and an anamnestic interview to detect the presence of bruxism and its forms (sleep, awake, and sleep–awake) by an oral and maxillofacial surgeon. It was reported that the attrition caused by bruxism is believed to produce a recognizable pattern of wear with "highly polished facets, matching facets, ridges between facets, grooves, ledges, and thinning out and scooping out of incisal edges of anterior teeth" and, in theory, can be distinguished from tooth wear as a result of abrasion or erosion [17]. Therefore, in the clinical examination, the teeth were dried with an air syringe and cotton rolls in order to determine the presence of any indicators of bruxism and each tooth was accurately examined using a dental mirror. The presence of "highly polished facets, matching facets, ridges between facets, grooves, ledges, and thinning out and scooping out of incisal edges of anterior teeth" were clinical indicator of bruxism.

Selection criteria for sleep bruxism and non-bruxism were derived from the literature, although such diagnostic criteria set for sleep bruxism can correctly detect sleep grinding, but not sleep clenching. In our study, clinical criteria which were clinically originally proposed to screen patients for research sleep laboratory studies have been adopted to diagnose sleep bruxism [3, 23].

Diagnosis of sleep bruxism was made when the patient exhibited, at least five nights a week, grinding bruxism sounds during sleep in the last 6 months, as reported by his/

her bed/room partner or family members and at least one of following adjunctive clinical criteria: observation of tooth wear or shiny spots on restorations in clinical examination, report of morning masticatory muscle fatigue or pain, and masseteric hypertrophy upon digital palpation.

It is suggested that bruxism during wakefulness is commonly characterized by a clenching-type activity, while sleep bruxism by a combination of clenching and grinding-type activity. Also, there is some consensus that clenching-type activity during the day is associated more with jaw pain than tooth grinding during sleep, even though experimental studies on tooth clenching as well as studies adopting tooth wear levels as an indicator of tooth grinding suffer from some methodological shortcomings [1]. In light of these information, diagnosis of awake bruxism was made when the patient exhibited, at least 5 days a week, clenching the teeth during the day in the last 6 months and at least one of following adjunctive clinical criteria: not observation of tooth wear or shiny spots on restorations in clinical examination; feeling muscular tension and stiffness during the day; and masseter and/or temporalis muscles pain and/or fatigue during the day.

Participants were then divided into four groups, the first comprising subjects showing only sleep bruxism findings (sleep bruxers $n=12$, 14.1%), the second consisting of subjects showing only awake bruxism findings (awake bruxers $n=24$ 28.2%), and third including subjects showing both sleep and awake bruxism findings (sleep–awake bruxers $n=33$ 38.8%). Participants, who were not compatible with our clinical diagnostic criteria for sleep and awake bruxism, were grouped as non-bruxers ($n=16$ 18.8%). Self-report questionnaire (SCL-90-R) was filled out by all participants and their psychopathological symptoms profiles were evaluated by a trained psychiatrist.

Psychopathological assessment

All patients filled out the Turkish version of the SCL-90-R for psychopathological assessment [19, 20]. Among several self-report instruments developed to assess current psychopathology, the SCL-90-R is one that is extensively used in the mental health area [26].

Specifically, the SCL-90-R is a 90-item multidimensional questionnaire designed to screen for a broad range of psychological problems. Each of the 90 items is rated on a five-point Likert scale of distress, ranging from “not at all” (0) to “extremely” (4). Subsequently, the answers are combined in nine primary symptom dimensions: somatization (SOM), obsessive–compulsive (O-C), interpersonal sensitivity (I-S), depression (DEP), anxiety (ANX), anger–hostility (HOS), phobic anxiety (PHOB), paranoid ideation (PAR), and psychoticism (PSY). In addition, three global indices provide measures of overall psychological distress: the Global Severity Index (GSI), the Positive Symptom Total (PST), and the Positive Symptom Distress Index (PSDI) [19, 26, 27].

Statistical analysis

Because variables were not normally distributed, Kruskal–Wallis test was used to compare values obtained by the four groups on various SCL-90-R subscales. After that, Mann–Whitney test and Bonferroni’s correction were performed to compare values between paired groups on various SCL-90-R subscales. Statistical significance was set at $p<0.05$. All statistical analyses were performed with the Statistical Package for the social Sciences 15.0 (SPSS Inc.; Chicago, IL, USA).

Results

The study sample included 85 patients (68 males and 17 females, mean age=26.8±8.6, range 18–59), sleep bruxism was diagnosed in 12 (14.1%) patients, awake bruxism was diagnosed in 24 (28.2%) patients, sleep–awake bruxism was diagnosed in 33 (38.8%) patients, and 16 (18.8%) patients did not meet the criteria for bruxism. Diagnostic groups of the patients were not significantly different for the mean age, which was 30.0±7.4 for non-bruxers, 24±6 for sleep bruxers, 25.7±7.6 for awake bruxers, and 27.1±10.2 for sleep–awake bruxers (chi-square=7.011; $p=0.072$). Regarding gender distribution, despite the fact that male/female ratios were different and in male’s favor in the four groups of patients, chi-square test revealed no significant differences between diagnostic groups (chi-square=6.200; $p=0.102$; Table 1).

As regards mean psychopathological scores, patients with sleep–awake bruxism endorsed the highest scores in all SCL-90-R subscales. In addition, patients with awake bruxism showed higher scores than patients with sleep bruxism and non-bruxism in most SCL-90-R subscales. Kruskal–Wallis test showed that differences between groups were significant in all SCL-90-R subscales except in psychoticism subscale (chi-square=6.103; $p=0.107$; Table 2).

According to the Mann–Whitney test between non-bruxer group and other bruxer groups in all SCL-90-R subscales, except for the somatization subscale ($Z=2.778$; $p=0.04$) between non-bruxer and sleep bruxer groups, there was not statistically significant difference between non-bruxer and sleep bruxer or awake bruxer groups. On the other hand, Mann–Whitney test followed by Bonferroni’s test correction between non-bruxer and sleep–awake bruxer groups revealed significant differences in depression ($Z=3.034$; $p=0.002$), anxiety ($Z=3.276$; $p=0.001$),

Table 1 Mean age (±SD) and gender ratio of the four groups of patients

	Number of patients	Mean age	Male/female
Non-bruxers	16 (18.8%)	30±7.4	11:5
Sleep bruxers	12 (14.1%)	24±6	12:0
Awake bruxers	24 (28.2%)	25.7±7.6	21:3
Sleep–awake bruxers	33 (38.8%)	27.1±10.2	24:9
Total	85 (100%)	26.8±8.6	68:17

hostility (Z=3.446; p=0.001), phobic anxiety (Z=2.784; p=0.005), paranoid ideation (Z=2.878; p=0.004), global severity index (Z=3.038; p=0.002), positive symptom distress index (Z=3.177; p=0.001), and positive symptom total (Z=2.698; p=0.007) subscales. Despite the fact that differences between sleep bruxer and sleep–awake bruxer groups were significant in all SCL-90-R subscales except for interpersonal sensitivity (Z=1.967; p=0.049) and psychoticism subscales (Z=1.820; p=0.070), differences between awake bruxer and sleep–awake bruxer groups were not significant in all SCL-90-R subscales except for positive symptom distress index (Z=2.878; p=0.004). There were statistically significant differences between sleep bruxer and awake bruxer groups in somatization (Z=2.881; p=0.003) and anxiety (Z=3.066; p=0.002) subscales (Table 3).

Overall Cronbach's alpha score of SCL-90-R was 0.989, and in subscales 0.928 for somatization, 0.895 for obsession, 0.926 for interpersonal sensitivity, 0.939 for depression, 0.947 for anxiety, 0.915 for hostility, 0.894 for phobic anxiety, 0.872 for paranoid ideation, and 0.902 for psychoticism.

Discussion

Etiology of bruxism is to a great extent unknown and controversial, and many theories have been developed. These etiological theories have been proposed over the years, and a multifactorial model to explain bruxism etiology seems to be the most plausible hypothesis, according to which psychosocial and pathophysiological factors interact with morphological–peripheral ones [1, 24, 28]. On the other hand, a consistent amount of literature suggests that peripheral sensory influences play a minor role in the etiopathogenesis of bruxism, while central nervous system-related factors seem to have much more importance [1, 3–6, 9, 12, 16, 17, 22, 29, 30]. Manfredini et al. [22] reported in their study on psychic and occlusal factors in bruxers that there was poor evidence of a relationship between occlusion and bruxism, while psychiatric evaluation showed an association with anxiety, depressive, and manic symptoms. The observations seem to strengthen the widespread opinion among practitioners that a bruxism–psychosocial factors relationship does exist [1, 3–6, 9, 12, 22, 24, 30]. In addition, bruxism associated

Table 2 Mean±SD/median values of the three groups of patients in the SCL-90-R subscales and results of Kruskal–Wallis test

	Non-bruxism	Sleep bruxism	Awake bruxism	Sleep–awake bruxism	Chi-square (Kruskal–Wallis)	p value
SOM	0.59±0.38/0.58	0.20±0.28/0.08	0.79±0.85/0.50	1.33±0.97/1.08	23.797	<0.001*
O-C	0.76±0.50/0.85	0.53±0.41/0.60	1.14±0.79/1.10	1.53±1.03/1.30	14.218	0.003*
I-S	0.52±0.41/0.38	0.54±0.38/0.44	0.84±0.75/0.72	1.39±1.22/1.00	8.013	0.046*
DEP	0.44±0.41/0.30	0.26±0.27/0.15	0.84±0.86/0.73	1.41±1.11/1.15	17.929	<0.001*
ANX	0.31±0.32/0.20	0.14±0.22/0.00	0.77±0.96/0.40	1.24±1.13/0.80	23.213	<0.001*
HOS	0.31±0.38/0.16	0.34±0.32/0.25	0.93±1.03/0.58	1.37±1.22/1.33	15.344	0.002*
PHOB	0.19±0.34/0.00	0.07±0.09/0.00	0.40±0.71/0.14	0.92±1.07/0.57	13.891	0.003*
PAR	0.39±0.39/0.33	0.27±0.41/0.08	0.84±0.83/0.58	1.31±1.16/1.00	16.052	0.001*
PSY	0.35±0.49/0.15	0.25±0.23/0.20	0.50±0.74/0.20	0.87±0.95/0.60	6.103	0.107
GSI	0.47±0.34/0.37	0.31±0.22/0.28	0.82±0.77/0.65	1.30±1.02/0.97	17.901	<0.001*
PSDI	1.43±0.29/1.36	1.36±0.37/1.30	1.57±0.64/1.30	2.05±0.74/1.79	17.983	<0.001*
PST	29.3±19.5/25	20.4±15.5/15	40.2±24.8/44	50.7±25.9/51	15.839	<0.001*

SOM somatization, *O-C* obsessiveness–compulsiveness, *I-S* interpersonal sensitivity, *DEP* depression, *ANX* anxiety, *HOS* hostility, *PHOB* phobic anxiety, *PAR* paranoid ideation, *PSY* psychoticism, *GSI* Global Severity Index, *PSDI* Positive Symptom Distress Index, *PST* positive symptom total, *SCL-90-R* Symptom Check List—Revised

*p<0.05 (p values are not corrected by Bonferroni's test correction on the table)

Table 3 Results of pair-wise group comparison by Mann–Whitney test followed by Bonferroni's correction

	NB vs. SB	NB vs. AB	NB vs. SAB	SB vs. AB	SB vs. SAB	AB vs. SAB
SOM	Z=2.778	Z=0.055	Z=2.616	Z=2.881	Z=4.279	Z=2.476
	p=0.004*	p=0.967	p=0.009	p=0.003*	p=0.000*	p=0.013
O-C	Z=1.235	Z=1.330	Z=2.455	Z=2.439	Z=3.200	Z=1.441
	p=0.223	p=0.192	p=0.014	p=0.013	p=0.001*	p=0.150
I-S	Z=0.304	Z=1.455	Z=2.297	Z=0.893	Z=1.967	Z=1.522
	p:0.767	p=0.149	p=0.022	p=0.379	p=0.049	p=0.128
DEP	Z=0.985	Z=1.499	Z=3.034	Z=2.259	Z=3.458	Z=1.983
	p=0.347	p=0.141	p=0.002*	p=0.024	p=0.000*	p=0.047
ANX	Z=1.875	Z=1.966	Z=3.276	Z=3.066	Z=3.936	Z=1.704
	p=0.074	p=0.051	p=0.001*	p=0.002*	p=0.000*	p=0.088
HOS	Z=0.598	Z=1.957	Z=3.446	Z=1.360	Z=2.788	Z=1.518
	p=0.568	p=0.054	p=0.001*	p=0.188	p=0.005*	p=0.129
PHOB	Z=0.424	Z=1.184	Z=2.784	Z=1.642	Z=2.934	Z=1.985
	p=0.762	p=0.279	p=0.005*	p=0.128	p=0.003*	p=0.047
PAR	Z=0.776	Z=1.864	Z=2.878	Z=2.592	Z=3.122	Z=1.452
	p=0.478	p=0.066	p=0.004*	p=0.009	p=0.001*	p=0.146
PSY	Z=0.356	Z=0.747	Z=2.025	Z=0.594	Z=1.820	Z=1.491
	p=0.732	p=0.469	p=0.043	p=0.562	p=0.070	p=0.136
GSI	Z=1.278	Z=1.325	Z=3.038	Z=2.165	Z=3.581	Z=1.859
	p=0.205	p=0.192	p=0.002*	p=0.029	p=0.000*	p=0.063
PSDI	Z=1.163	Z=0.207	Z=3.177	Z=0.455	Z=3.414	Z=2.878
	p=0.260	p=0.838	p=0.001*	p=0.655	p=0.000*	p=0.004*
PST	Z=1.232	Z=1.105	Z=2.698	Z=2.418	Z=3.427	Z=1.617
	p=0.223	p=0.279	p=0.007*	p=0.015	p=0.000*	p=0.106

NB non-bruxers, *SB* sleep bruxers, *AB* awake bruxers, *SAB* sleep–awake bruxers, *SOM* somatization, *O-C* obsessiveness–compulsiveness, *I-S* interpersonal sensitivity *DEP* depression, *ANX* anxiety, *HOS* hostility, *PHOB* phobic anxiety, *PAR* paranoid ideation, *PSY* psychoticism, *GSI* Global Severity Index, *PSDI* Positive Symptom Distress Index, *PST* positive symptom total, *SCL-90-R* Symptom Check List—Revised

*$p<0.05$.

with psychologic disorders can also be related to the medications used to treat them [6, 25].

Sleep bruxism was classified as a "sleep-related movement disorder" according to the recent International Classification of Sleep Disorder [31]. On the other hand, the hypothesis that wake clenching is strictly related to depression, or may be an expression of a depressed mood is fascinating and has found some support in the psychiatric literature suggesting that bipolar patients are characterized by disturbances in the central neurotransmitter system which may also be involved in the etiology of bruxism [1, 32].

Subjects were consecutively selected from participants who self-reported bruxism in order to create groups of somehow different bruxism forms. Before we started our study, we supposed that some participants could be compatible with either sleep or awake bruxism findings gathered by clinical and anamnestical diagnostic criteria for sleep and awake bruxism as well as with both or with neither of them. Therefore, we divided patients into four groups as sleep bruxers, awake bruxers, sleep–awake bruxers, and non-bruxers.

In this investigation, a validated clinical criterion which was originally proposed to screen clinical patients for research sleep laboratory studies was adopted to diagnose participants with sleep bruxism [3, 4, 23]. This validated clinical criteria set for sleep bruxism can correctly detect sleep grinding, but not sleep clenching. On the other hand, diagnosis of awake bruxism was based upon the presence of an anamnestical indicator and at least one adjunctive clinical criterion, as suggested elsewhere [1, 4, 22]. Despite the use of such standardized criteria, a clinical approach to the diagnosis of bruxism still presents some shortcomings, not allowing an exact distinction between sleep and awake bruxism, a gradation of bruxism severity, so limiting the generalizability of the results. Nevertheless, it is probably the simplest approach to a complex disorder in phase of preliminary data gathering [2].

One of the problems in the study of the relation between bruxism and psychopathology is represented by the possible presence of TMD in bruxers. A number of conditions have been described concerning an association between psychopathological symptoms and TMD. Also, bruxism is considered a major risk factor for TMD [3]. In particular, both bruxism and some forms of psychopathology appear to be somehow related to painful TMD, and in particular to muscular forms [3, 16, 24, 33–35]. Consequently, the patients with signs of painful TMJ disorder were excluded from this study.

Self-report symptom inventories are commonly used by both clinicians and researchers to gather information about

patients' mental states. Rather than clinician-based rating scales, the information comes directly from the patients reflecting their perceptions of distress [19, 20, 26]. That is why we used the full version of SCL-90-R instrument in our study. Our results of reliability about subscales were satisfactory as defined in results section.

In this research, a priori sample size was calculated for the comparison of the mean SCL-90 scores in all four groups. Sample size calculation was performed on the basis of the variance analysis (ANOVA) between four groups. The power of the post-hoc tests between paired groups is less. Therefore, the p values in Tables 2 and 3 can be interpreted as a trend without 80% power and 40% effect size.

Results could have been influenced by our sample profile (military people) because of their greater exposure to stress than others in population. However, as regards psychopathological symptoms which characterized bruxism forms in the present study, this investigation has provided pilot data that, at present, are not comparable with those from studies due to the lack of analogue works.

As regards gender differences, our observations are not comparable with results of other investigations on polisomnographically diagnosed sleep bruxism and studies adopting clinical and/or interview-based diagnosis of bruxism. Because we performed our study at military hospitals and a unit's health department, our subjects were mostly males. Nevertheless, regarding gender distribution, despite the four groups of patients being different for male/female ratio in male's favor, statistical analysis revealed that there were not significant differences between diagnostic groups.

Using of the self-report questionnaire in order to accurately detect the presence of bruxism, which is a subconscious parafunctional behavior, was a limitation of this study. On the other hand, Lobbezoo et al. [9] reported that bruxism should be diagnosed along multiple axes, questionnaires, an oral history taking (including a bed partner's report of grinding sounds), an extra-oral and intra-oral inspection for clinical signs of bruxism, and, in some cases, an electromyographic recording of the activity of the masticatory muscles or even a polysomnographic recording of the sleeping patient. Any single one of these diagnostic tools should not be used in isolation, because patients may not be aware of the presence of bruxism, the clinical signs of bruxism may reflect a problem in the past rather than one in the present, and EMG and PSG only give a random indication of a disorder that fluctuates over time [9]. Furthermore, in their review of the literature, Manfredini and Lobbezoo [1] reported that the majority of data about the association between psychosocial disorders and bruxism came from studies adopting a clinical and/or self-report diagnosis of bruxism.

In this study, as regards mean psychopathological scores, participants with awake bruxism showed higher scores than participants with sleep bruxism and non-bruxism in most SCL-90-R subscales. Furthermore, there were statistically significant differences between sleep bruxer and awake bruxer groups in somatization and anxiety subscales, while there was only statistically significant difference between non-bruxer and sleep bruxer groups in somatization subscale. Our findings supported the hypothesis suggested by Olkinuora [10], who claimed that daytime clenching is a response to stress and that daytime clenchers' scores on psychological tests would be higher in emotional disturbances than subjects who brux their teeth nocturnally. To support this hypothesis, an EMG-based study by Rao and Glaros [11] suggested that emotional and situational factors may be important in the etiology of awake bruxism [1].

On the other hand, as regards mean psychopathological scores, participants with sleep–awake bruxism endorsed the highest scores in all SCL-90-R subscales. Statistical analysis between sleep–awake bruxer and non-bruxer groups revealed significant differences in most of SCL-90-R subscales. Despite the fact that differences between sleep–awake bruxer and sleep bruxer groups were significant in all SCL-90-R subscales except for interpersonal sensitivity and psychoticism subscales, differences between sleep–awake bruxer and awake bruxer groups were not significant in all SCL-90-R subscales except for positive symptom distress index. These findings are open to several interpretations; sleep and awake bruxism found together might be due to increase of psychological problems. Because a clearer distinction could not be made among sleep bruxism, awake bruxism, and sleep and awake bruxism, conflicting findings between clinical and EMG-based studies on bruxism, stress, or psychosocial factors are possible.

In general, statistical analysis of our study showed that differences between groups were significant in all SCL-90-R subscales except for psychoticism subscale. This observation may be interpreted that bruxism has three different clinical forms: sleep, awake, and sleep and awake.

Better distinction of bruxism forms may help to develop new treatment strategies for bruxism disorder. Future multidisciplinary studies on broader samples should be directed toward standardizing clinical criteria for detecting bruxism forms.

Conflict of interest Authors of this manuscript declare that they have no conflict of interest.

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