SOPIO DALALISHVILI, VLADIMER MARGVELASHVILI

BRUXISM IN DENTAL PRACTICE: DIAGNOSTIC PERSPECTIVES, SYSTEMIC ASSOCIATIONS AND MODERN THERAPEUTIC INTERVENTIONS

Ivane Javakhishvili State University, Tbilisi, Georgia

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სოფიო დალალიშვილი, ვლადიმერ მარგველაშვილი

ბრუქსიზმი სტომატოლოგიურ პრაქტიკაში: დიაგნოსტიკური პერსპექტივები, სისტემური ასოციაციები და თანამედროვე თერაპიული ჩარევები

ივანე ჯავახიშვილის სახელმწიფო უნივერსიტეტი, თბილისი, საქართველო

რეზიუმე

ბრუქსიზმი პარაფუნქციური აქტივობაა, რომელიც ხასიათდება კბილების უნებლიე ღრჭენით ან კრაჭუნით, რომელიც ხდება სიფხიზლის ან ძილის დროს. მიუხედავად იმისა, რომ ადრე ბრუქსიზმი ძირითადად სტომატოლოგიურ პრობლემად იყო კლასიფიცირებული, ამჟამად აღიარებულია, როგორც მრავალფაქტორიანი დარღვევა რთული ნევროლოგიური, ფსიქოლოგიური და ბიომექანიკური საფუძვლებით. ეს მიმოხილვა ასახავს მიმდინარე დიაგნოსტიკურ კრიტერიუმებს, რომლებიც განასხვავებენ ფხიზელი და ძილის ბრუქსიზმს და იკვლევს ეტიოლოგიურ ფაქტორებს, მათ შორის ემოციურ სტრესს, ოკლუზიურ შეუსაბამობებს, ნეიროქიმიურ დისბალანსს და სისტემურ მდგომარეობებს, როგორიცაა გასტროეზოფაგური რეფლუქსი და ძილის აპნოე.

მკურნალობის გარეშე, ბრუქსიზმა შეიძლება გამოიწვიოს მნიშვნელოვანი სტომატოლოგიური და კუნთოვანი გართულებები, მათ შორის მინანქრის ცვეთა, კბილის მოტეხილობა, პაროდონტის დაზიანება, საფეთქელ-ქვედა ყბის სახსრის დისფუნქცია და ოროფაციური ტკივილი. მართვის თანამედროვე მიდგომები მრავალგანზომილებიანია, პაციენტის განათლებიდან და ქცევითი თერაპიიდან დაწყებული ფარმაკოლოგიური მკურნალობითა და ოკლუზიური სპლიტური თერაპიით დამთავრებული. ზოგიერთ შემთხვევაში, ბოტულინის ტოქსინის გამოყენებამ აჩვენა პერსპექტიული შედეგები კუნთების ჰიპერაქტიურობისა და ტკივილის შემცირებაში. სტატიის მიზანია ბრუქსიზმის პათოფიზიოლოგიის, კლინიკური შედეგებისა და მართვის შესახებ არსებული მტკიცებულებების სინთეზირება.

Introduction. Bruxism is defined as a parafunctional activity involving involuntary clenching, grinding, or gnashing of the teeth, and is typically classified as either sleep bruxism or awake bruxism, depending on its circadian presentation [1]. Historically viewed as a purely dental phenomenon, bruxism is now recognized as a multifactorial disorder with implications across dentistry, neurology, psychiatry, and sleep medicine [2]. The etiology of bruxism remains complex and not fully understood. While occlusal disturbances were once considered primary causative factors, contemporary research emphasizes the role of psychological stress, neurochemical dysfunctions, and central nervous system dysregulation [3]. The condition has also been associated with comorbidities such as gastroesophageal reflux, obstructive sleep apnea, and certain neurodegenerative diseases, indicating systemic involvement [4,5].

Epidemiological data suggest that bruxism affects approximately 8–31% of the general population, with a notable prevalence among children and young adults [6]. Sleep bruxism is often episodic and asymptomatic in its early stages, making it difficult to detect clinically. Over time, however, it can contribute to tooth wear, fractures, temporomandibular joint (TMJ) dysfunction, and facial pain, significantly impairing quality of life [7]. Due to the complex and multifactorial nature of the disorder, multidisciplinary approaches to treatment have become the standard. These include behavioral interventions, psychological therapy, occlusal appliance therapy, pharmacological agents, and, in refractory cases, the use of botulinum toxin injections to manage muscular hyperactivity [8,9]. This review aims to summarize current knowledge on the classification, pathophysiology, and complications of bruxism and to provide an overview of contemporary therapeutic strategies available in clinical practice.

Etiology and Pathogenesis. The etiology of bruxism is multifactorial, involving an interplay of central and peripheral mechanisms. Early theories attributed bruxism primarily to occlusal disharmony or abnormal dental contacts; however, more recent studies have demonstrated that occlusal factors alone are insufficient to explain the complexity of this disorder [10]. Bruxism is now recognized as a centrally mediated condition influenced by neurological, psychological, and behavioral elements.

One of the primary contributing factors is psychological stress, which has been positively correlated with both awake and sleeps bruxism. Emotional stress, anxiety, and personality traits such as hyperactivity or aggression can modulate muscle tone and central nervous system arousal, leading to parafunctional activities like clenching and grinding [11]. This association is particularly pronounced in awake bruxism, where cognitive awareness and emotional response directly influence mandibular activity.

Neurochemical dysregulation also plays a role in the pathogenesis of sleep bruxism. Studies have suggested that alterations in neurotransmitters - particularly dopamine - may affect motor regulation during sleep and contribute to bruxism episodes. Pharmacological evidence supports this theory, as dopaminergic agents have been shown to modulate bruxism activity in susceptible individuals.

In addition, sleep-related arousal phenomena such as micro-awakenings have been implicated in sleep bruxism. These episodes are associated with transient increases in heart rate, respiratory activity, and muscle tone, often preceding bruxism events. Comorbid sleep disorders, including obstructive sleep apnea (OSA), have been shown to increase the frequency and intensity of bruxism episodes, suggesting a shared pathophysiological substrate [12].

Bruxism has also been linked with systemic and neurological conditions, including Parkinson's disease, gastroesophageal reflux disease (GERD), and certain forms of epilepsy, further reinforcing its multifaceted nature. Figure 1. illustrates a fractured distopalatal cusp, a common clinical consequence of chronic bruxism-related occlusal stress. In children, bruxism may reflect both developmental neuromuscular factors and behavioral responses to stress or environmental stimuli. Pediatric bruxism is frequently observed in conjunction with attention-deficit/hyperactivity disorder (ADHD), sleep disturbances, and anxiety disorders [13]. Taken together, these findings support a model in which bruxism is not a single-pathway disorder but rather a multifactorial phenomenon resulting from complex interactions between psychological, neurophysiological, and behavioral processes.

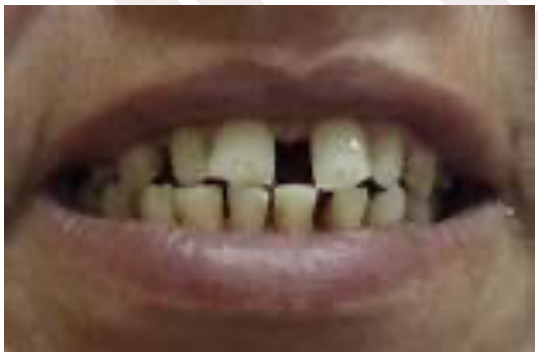

Figure 1. Fractured Distopalatal Cusp in a Bruxist Patient

Classification of Bruxism. Bruxism is broadly classified based on its occurrence during the sleep–wake cycle. According to the international consensus published in 2013, it is divided into two primary types: sleep bruxism and awake bruxism [14]. Sleep bruxism is defined as a masticatory muscle activity occurring during sleep and characterized as either rhythmic (phasic) or non-rhythmic (tonic). It is regarded as a sleep-related movement disorder and often coincides with micro-arousals, increased autonomic nervous system activity, and changes in sleep architecture.

Awake bruxism, in contrast, is defined as a masticatory muscle activity during wakefulness, typically manifested by repetitive or sustained tooth contact, clenching, or bracing of the mandible. Unlike sleep bruxism, which is often involuntary and unnoticed by the patient, awake bruxism may be more consciously regulated and associated with stress, concentration, or emotional states.

A third category, known as mixed bruxism, refers to the coexistence of both awake and sleep bruxism in the same individual. This form may be more clinically complex, as it combines elements of both voluntary and involuntary behaviors, often requiring an interdisciplinary diagnostic and therapeutic approach.

In terms of severity, bruxism can be further subclassified as:

- Mild: occasional occurrences without functional consequences
- Moderate: frequent episodes with mild dental wear or muscle fatigue
- Severe: persistent activity leading to significant dental damage, temporomandibular disorders (TMD), or orofacial pain

The most widely accepted tools for classification include patient self-reports, clinical examination, and where necessary, polysomnographic recordings. Recent developments also support the use of ambulatory electromyography (EMG) and smartphone-based behavioral monitoring as adjunctive diagnostic tools.

Understanding the type and severity of bruxism is crucial for developing individualized treatment plans and assessing the risk of long-term complications, another example of occlusal damage in a bruxist patient is shown in Figure 2., further highlighting the destructive potential of sustained parafunctional activity.

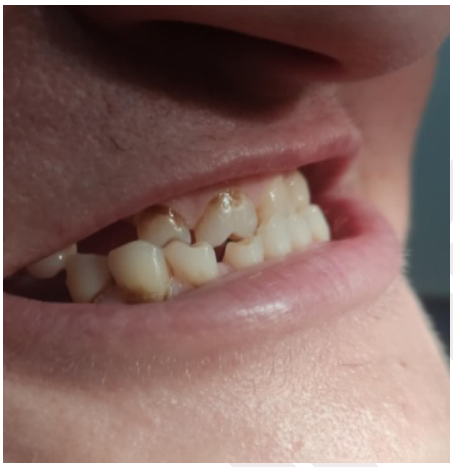

Figure 2. Additional Fractured Cusp in a Bruxist Patient

Clinical Manifestations and Complications. Bruxism presents with a broad spectrum of clinical signs and symptoms, varying by duration, severity, and whether the activity occurs during wakefulness or sleep. In early stages, the condition may be asymptomatic and detected only incidentally during routine dental examinations. However, chronic or severe bruxism can lead to a cascade of functional and structural complications affecting the teeth, masticatory muscles, temporomandibular joints (TMJs), and associated craniofacial structures. Dental complications are among the most common and include enamel attrition, tooth fractures, loss of vertical dimension, and hypersensitivity due to dentin exposure. In cases of prolonged bruxism, restorative dental work may also become compromised, leading to recurrent prosthetic failures.

Muscular symptoms often involve the masseter, temporalis, and medial pterygoid muscles, presenting as morning muscle fatigue, facial tension, or pain upon palpation. Overuse of these muscles can lead to myofascial pain syndrome and contribute to headaches, particularly tension-type headaches.

Temporomandibular joint disorders (TMD) represent another significant consequence. Bruxism can cause or exacerbate TMD symptoms, including joint clicking, restricted mandibular movement, and pain during function. Chronic loading of the TMJs may also accelerate degenerative changes, leading to internal derangement or osteoarthritis. In pediatric populations, bruxism may manifest as tooth wear, sleep disturbances, or behavioral issues. Though often transient, pediatric bruxism warrants monitoring due to its potential connection with neurodevelopmental or psychosocial factors. In severe or untreated cases, bruxism can significantly impact quality of life, contributing to disturbed sleep, impaired mastication, reduced aesthetic confidence, and chronic orofacial pain. Psychological effects such as irritability, anxiety, and depression may arise secondary to persistent discomfort or frustration with dental complications.

Early diagnosis and intervention are essential to prevent progression from subclinical activity to overt pathology. The presence of any of the above symptoms, particularly when accompanied by evident tooth wear or jaw discomfort. Figure 3 depicts dentition misalignment, which may act as both a contributing factor to and a consequence of chronic bruxism.

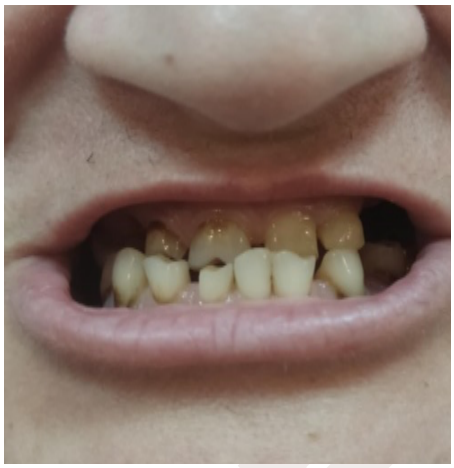

Figure 3. Malocclusion and Dentition Irregularity

Diagnosis of Bruxism. The diagnosis of bruxism is based on a combination of patient-reported symptoms, clinical examination, and, when necessary, instrumental assessments. The international consensus guidelines published in 2013 categorize the diagnostic certainty of bruxism into three levels: possible, probable, and definite.

- A possible diagnosis is based solely on self-report, typically gathered through questionnaires or patient interviews. Patients may report clenching, grinding sounds during sleep (noted by a bed partner), morning jaw discomfort, or awareness of parafunctional habits during the day.
- A probable diagnosis is established when self-reported symptoms are corroborated by clinical signs. These may include tooth wear facets, hypertrophy of the masseter muscles, tongue or cheek indentations, and temporomandibular joint tenderness [2].
- A definite diagnosis requires instrumental confirmation, such as polysomnography with audiovisual recordings or electromyographic (EMG) monitoring. These methods are typically reserved for research settings or complex cases due to cost and limited availability.

For sleep bruxism, polysomnography (PSG) remains the gold standard. It allows for the detection of rhythmic masticatory muscle activity (RMMA), often occurring in association with sleep arousals, increased heart rate, and other physiological markers. For awake bruxism, diagnosis relies primarily on behavioral monitoring and patient awareness, although recent developments in mobile applications and wearable EMG devices show promise for enhancing objectivity.

Differential diagnosis is also important, as bruxism may be mistaken for other conditions such as oromandibular dystonia, gastroesophageal reflux disease, or epileptic seizures, particularly when nocturnal movement is reported without confirmatory observation [4,14].

Ultimately, clinical judgment remains central to diagnosis, particularly in general dental practice, where access to advanced diagnostics may be limited. Combining self-reported behavior with observable signs of dental or muscular damage provides a pragmatic and effective approach in most cases, Figure 4 summarizes the clinical profile of bruxism and illustrates common diagnostic considerations.

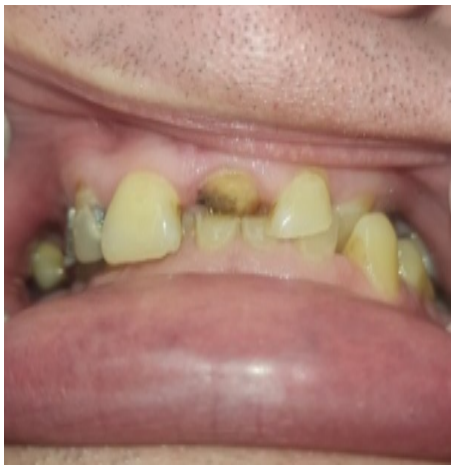

Figure 4. Diagnostic Overview of Bruxism

Management and Treatment of Bruxism. The treatment of bruxism depends on its type, severity, underlying cause, and whether it leads to clinical consequences. Because bruxism is often multifactorial in origin, management typically involves a multidisciplinary approach, integrating behavioral strategies, occlusal interventions, pharmacotherapy, and - when indicated - neuromodulatory techniques.

1. Behavioral and Psychosocial Interventions

In cases where bruxism is associated with stress, anxiety, or maladaptive coping mechanisms, cognitive behavioral therapy (CBT), biofeedback training, and relaxation techniques have shown positive effects in reducing awake bruxism. Patient education regarding parafunctional habits, posture awareness, and sleep hygiene is an essential component of initial management, particularly in children and adolescents [15.]

2. Occlusal Splint Therapy

Occlusal splints (also known as night guards) remain one of the most widely used interventions for sleep bruxism. These custom-made acrylic appliances are worn over the upper or lower teeth to reduce tooth wear, protect restorations, and alleviate muscle strain. While they do not eliminate the motor activity of bruxism, splints reduce the damaging effects of grinding and clenching. There is ongoing debate about the optimal splint design - hard versus soft, maxillary versus mandibular - and therapy must be tailored to the patient's dentition, muscle symptoms, and presence of temporomandibular joint disorders.

3. Pharmacological Treatment

Although no medication has been officially approved specifically for bruxism, pharmacologic agents may be considered in selected cases. Muscle relaxants, benzodiazepines, tricyclic antidepressants, and dopamine agonists have been used off-label, primarily in sleep bruxism with comorbid insomnia, anxiety, or neurodegenerative conditions. However, long-term use is limited by concerns regarding tolerance, dependence, and side effects.

4. Botulinum Toxin (Botox) Injections

In cases of severe, refractory bruxism - particularly where muscle hypertrophy and myofascial pain are prominent - botulinum toxin type A injections into the masseter and temporalis muscles have shown efficacy in reducing muscle activity and pain. Although not curative, Botox offers relief for patients

who have not responded to conservative treatment and is increasingly recognized as a valuable adjunct in bruxism management, Figure 5 shows advanced occlusal wear, a hallmark of long-standing bruxism requiring comprehensive rehabilitative management.

5. Management of Secondary Bruxism

When bruxism is secondary to conditions such as obstructive sleep apnea (OSA) or gastroesophageal reflux disease (GERD), treatment of the underlying disorder often leads to a reduction or even elimination of the bruxism. This is because the bruxism in these cases in often a symptom of the sleep or gastrointestinal issue, rather than a primary condition.

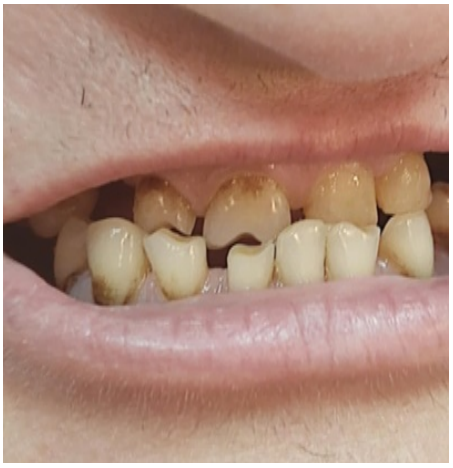

Figure 5. Severe Tooth Wear from Chronic Bruxism

Conclusion. Bruxism is a complex, multifactorial condition that extends beyond its traditional dental classification to encompass neurophysiological, psychological, and systemic dimensions. It can occur during sleep or wakefulness, often resulting in significant dental, muscular, and temporomandibular complications if left untreated. Although frequently asymptomatic in its early stages, chronic bruxism can progress to impair quality of life through pain, dysfunction, and aesthetic compromise. Effective diagnosis requires a careful combination of self-reported history, clinical examination, and, in select cases, instrumental assessment. While definitive diagnostic tools such as polysomnography and electromyography are reserved for complex or research settings, most cases can be reliably identified through clinical judgment and corroborative findings.

The management of bruxism is inherently interdisciplinary. Treatment strategies must be tailored to each patient's clinical presentation and etiological profile, often integrating behavioral therapy, occlusal splinting, pharmacologic agents, and - when warranted - botulinum toxin injections. Addressing comorbid conditions such as sleep apnea, GERD, or psychological distress is also critical to successful long-term outcomes.

As research continues to elucidate the mechanisms underlying bruxism and its varied manifestations, clinicians are encouraged to adopt a comprehensive, patient-centered approach. Early recognition and individualized intervention remain the cornerstones of preventing progression and minimizing the functional and structural impact of this prevalent disorder.

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BRUXISM IN DENTAL PRACTICE: DIAGNOSTIC PERSPECTIVES, SYSTEMIC ASSOCIATIONS AND MODERN THERAPEUTIC INTERVENTIONS

Javakhishvili Tbilisi State University

SUMMARY

Bruxism is a parafunctional activity characterized by involuntary grinding, clenching, or gnashing of teeth, occurring during wakefulness or sleep. While previously classified primarily as a dental concern, bruxism is now recognized as a multifactorial disorder with complex neurological, psychological, and biomechanical underpinnings. This review outlines current diagnostic criteria distinguishing awake and sleep bruxism and explores etiological factors including emotional stress, occlusal discrepancies, neurochemical imbalances, and systemic conditions such as gastroesophageal reflux and sleep apnea.

Left untreated, bruxism may lead to significant dental and musculoskeletal complications, including enamel wear, tooth fractures, periodontal injury, temporomandibular joint dysfunction, and orofacial pain. Contemporary approaches to management are multidimensional, ranging from patient education and behavioral therapy to pharmacological treatment and occlusal splint therapy. In select cases, the use of botulinum toxin has demonstrated promising outcomes in reducing muscle hyperactivity and pain.

This article aims to synthesize current evidence on the pathophysiology, clinical consequences, and management of bruxism, emphasizing the importance of individualized, interdisciplinary care in improving patient outcomes.

Keywords: bruxism, diagnosis, systemic associations, therapy