Chapter

Bruxism: Innovative Perspectives in Assessment and Therapy

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Abstract

Bruxism is a prevalent parafunctional behavior marked by involuntary clenching, grinding, bracing, or thrusting of the mandible during wakefulness or sleep, whose cumulative mechanical load can surpass the adaptive capacity of dental and musculoskeletal tissues and precipitate tooth wear, orofacial pain, and temporomandibular disorders. Reported prevalence estimates range 3.5–96%, reflecting heterogeneity in diagnostic definitions, assessment methods, and study populations, with adult rates averaging approximately 7% and pediatric estimates spanning 3.5–40.6%. Etiologically, bruxism emerges from the interplay of peripheral factors—such as facial morphology and occlusal discrepancies—central neuromuscular dysregulation, psychosocial stressors, pharmacological influences, and genetic predisposition. An integrated diagnostic schema—categorizing cases as possible, probable, or definite based on self-report, clinical examination, electromyography, and polysomnography—enhances consistency across settings. Management prioritizes reversible measures, such as patient education, behavioral modification, physical therapy, pharmacotherapy, and occlusal splint therapy, to reduce parafunctional load, with occlusal adjustment, botulinum toxin injections, or surgical intervention reserved for refractory presentations.

Keywords: bruxism, sleep bruxism, temporomandibular disorders, occlusal splint therapy, pathophysiology of bruxism

1. Introduction

Bruxism is a general term referring to involuntary clenching or grinding movements of the teeth that result from non-functional activation of the masticatory muscles, either during sleep or wakefulness [1]. "Clenching" describes tonic contractions resulting in sustained contact between the upper and lower teeth, whereas "grinding" refers to rhythmic chewing-like movements occurring in the absence of food. First described in the early twentieth century under the term *la bruxomania*, the condition was later renamed "bruxism" and has since undergone evolving definitions, highlighting its complexity and clinical relevance [2].

The concept of parafunction encompasses all movements of the masticatory system that do not serve a physiological purpose, among which bruxism is the most

common and clinically significant example [3–6]. Even brief episodes of excessive force can result in damage to the periodontal ligament and alveolar bone, while some patients may remain asymptomatic, underscoring the importance of careful evaluation of dental surface wear, masticatory muscle tone, and temporomandibular joint (TMJ) function during oral examinations [7–11].

The prevalence of bruxism varies widely due to differences in definitions, diagnostic criteria, and populations studied. Overall, the prevalence tends to increase from adolescence to young adulthood, with considerable interindividual variability [12, 13].

The historical variability in the clinical definitions of bruxism also contributes to its heterogeneity. While Zarb and Carlsson define bruxism strictly as tooth clenching or grinding that occurs during sleep [14], Walsh reported that such behavior can also be observed during wakefulness [15]. Attanasio distinguished between static and dynamic relationships: clenching, representing a static relationship that involves sustained contact between the maxilla and mandible in centric or eccentric positions; in contrast, grinding reflects a dynamic relationship, characterized by forceful contact during lateral or protrusive movements of the mandible [16].

The historical variability in bruxism definitions illustrates its heterogeneity. Modern definitions emphasize its involuntary nature and the need for clinical management. For example, the American Academy of Prosthodontics defines bruxism as an "involuntary habit of clenching or grinding the teeth" [17], while the American Academy of Orofacial Pain stresses that these parafunctional activities can occur both nocturnally and diurnally [18]. The most recent definition by Kato et al. describes bruxism as "a condition characterized by excessive forceful and prolonged involuntary contact or friction between the upper and lower teeth, which must be monitored and managed" [19]. This perspective not only clarifies the mechanism of bruxism but also highlights the necessity for precise diagnostics and innovative management approaches in contemporary clinical practice.

Understanding the neuroanatomical mechanisms underlying bruxism, including central cortico-cerebellar arousals and dopaminergic dysfunction, along with peripheral, genetic, and psychosocial factors, is essential for developing effective management strategies. Although no definitive treatment currently exists to eliminate bruxism permanently, clinical management aims to reduce the parafunctional load, protect dental and jaw structures, prevent pain and TMJ dysfunction, and improve patient quality of life [20].

2. Epidemiology and prevalence

Most adults, including children, exhibit nocturnal bruxism of varying intensity at some point in their lives. Although the tissues of the masticatory system generally adapt to this parafunctional activity, in some individuals, the cumulative forces may exceed physiological limits, leading to pain and functional impairment [20].

To date, the use of subjective assessment criteria has hindered the establishment of a globally standardized diagnostic protocol for bruxism. This issue also applies to determine its prevalence.

Consistent findings regarding the prevalence and characteristics of bruxism episodes remain elusive due to methodological variability among studies, including differences in diagnostic criteria, age groups, and sample types. As a result, reported prevalence rates in both adult and pediatric populations range widely from 7% to 88% [20].

In adults, bruxism is estimated to affect approximately 7.4% of the general population [21]. In studies focusing on sleep bruxism, particularly in children, prevalence rates have been reported to range from 3.5% to 40.6%, with a general trend of decline observed with advancing age [4]. In older populations, prevalence rates may drop as low as 3% [22].

There is also no clear consensus regarding sex-related differences. Some studies report that bruxism is three to nine times more prevalent in females compared to males [19, 23], whereas others have found no statistically significant difference between sexes [2, 4].

Polysomnographic studies reveal that the frequency and duration of bruxism episodes throughout the night vary significantly among individuals. On average, approximately five episodes occur during an 8-hour sleep period, with each episode lasting around 8 seconds. Consequently, a duration of 8–9 seconds has become the generally accepted range for a bruxism episode in the literature [20, 24]. However, some studies have reported that longer episodes may also occur. Furthermore, in individuals with temporomandibular disorders (TMDs), the cumulative duration of tooth contact during an 8-hour sleep period may reach up to 38.7 minutes, whereas in health controls, this average remains limited to 5.4 minutes [25]. These findings demonstrate that the duration and intensity of bruxism activity can vary widely depending on both physiological and pathological conditions, and that the resulting load may exceed the adaptive capacity of the musculoskeletal system.

3. Etiology

The origin of bruxism remains not fully understood, and current evidence suggests that it has a multifactorial etiology [3, 7, 26]. These factors are generally categorized into two groups: morphological (peripheral) and central [27]. Peripheral factors include facial morphology, condylar asymmetry, variations in dental arch form, malocclusion, discrepancies between centric relation and maximum intercuspation, and general occlusal irregularities. Central factors encompass psychological, pathophysiological, neurological, genetic, and systemic elements, as well as side effects of medications. Recent review studies indicate that pathophysiological components account for nearly 70% of bruxism cases, psychosocial factors account for approximately 20%, and morphological factors account for about 10%.

3.1 Morphological factors

Although skeletal deviations and occlusal discrepancies were initially considered the primary causes of bruxism, they are now believed to play only a limited role. No reliable association has been established between occlusal interferences and bruxism, and the elimination of these interferences has not been shown to affect parafunctional activity [28, 29] significantly. Nonetheless, achieving harmony between centric relation and maximum intercuspation is considered clinically beneficial [30].

3.2 Psychosocial factors

Although psychological factors have long been proposed as potential triggers for bruxism, no consistent personality profile unique to "bruxists" has yet been defined [31]. Questionnaire-based and self-reported studies suggest that individuals with a history of clenching or grinding may be more prone to anxiety, hyperactivity, or

aggression, although these findings lack consistency [32, 33]. The current theoretical framework posits that daily life stressors may increase both the frequency and severity of bruxism episodes. This model suggests that the limbic system and hypothalamus may trigger parafunctional activity by sending unnecessary stimuli to the masticatory muscles via the gamma-efferent pathway [34]. Indeed, isolated EMG recordings have shown a notable increase in masseter muscle activity during stressful periods, such as exams, arguments, or high workloads; however, these observations are mostly case-based, and their interpretation remains at a hypothetical level [35].

The lack of large-scale polysomnography-supported studies limits our understanding of the actual impact of psychological stress on nocturnal bruxism. Therefore, while stress, anxiety, and personality traits are accepted as potential contributors, their precise role in the development of bruxism has yet to be quantified. Further objective, longitudinal, and multi-center studies are needed to clarify these associations.

3.3 Pathophysiological factors

Sleep is divided into two primary phases: non-rapid eye movement (NREM) and REM, which alternate cyclically throughout the night [32, 36–38]. Polysomnographic data demonstrate that bruxism-related masticatory muscle activity can occur in all stages of the sleep cycle. However, approximately 60–80% of bruxism episodes in adults are concentrated in the first two stages of NREM sleep (N1–N2) [32, 36–38]. Although episodes occurring during the REM phase are less frequent, they are considered the most destructive form due to the intensity of muscle contractions and their prolonged duration [39].

It has been suggested that autonomic arousal and cortical reactivation during micro-arousals may trigger nocturnal bruxism by stimulating the masticatory muscles. Moreover, shortening of slow-wave sleep (N3) has been reported to facilitate this triggering [40, 41]. In individuals with snoring or obstructive sleep apnea syndrome (OSAS), both of which cause sleep fragmentation, bruxism risk has been shown to increase significantly [42, 43].

Recent studies highlight that centrally mediated autonomic nervous system mechanisms, modulated by psychological stress, play a more substantial role in the pathophysiology of bruxism than previously assumed [29, 30].

3.4 Drug-related side effects

Various studies have suggested that the side effects of substances, such as alcohol, caffeine, tobacco, and prescription medications for systemic diseases may contribute to the development of bruxism [44–46]. Lobbezoo et al. reported a reduction in bruxism activity in patients receiving low doses of levodopa. In contrast, long-term use of levodopa, particularly in conditions such as Parkinson's disease, was associated with increased clenching and grinding [47]. The same research group also proposed that selective serotonin reuptake inhibitors (SSRIs) may trigger bruxism when used long term [48]. Although there is a widespread belief regarding the adverse effect of SSRIs on bruxism, controlled clinical data confirming this association remain insufficient.

3.5 Genetic factors

Evidence concerning the genetic predisposition to bruxism appears to be conflicting. An extensive survey involving over 4,000 twin pairs reported that 39–64% of the

variance in bruxism could be attributed to genetic factors [49]. In contrast, another study with 250 twin pairs found no evidence of heritability [50]. On a molecular level, a specific polymorphism in the HTR2A gene has been associated with an increased susceptibility to sleep bruxism [51]. Additionally, between 20% and 50% of individuals with nocturnal bruxism report a family history of teeth grinding [31]. A meta-analysis reviewing 10 genetic studies supports the hypothesis that bruxism is partially heritable [52].

4. Diagnosis

Bruxism can be challenging to diagnose due to the lack of awareness by the patient or their surroundings, as well as the clinician's limited attention to subtle signs and symptoms.

The American Academy of Sleep Medicine (AASM) updated its diagnostic criteria in 2014, summarized as follows (**Table 1**) [53]:

Due to the lack of a universally accepted diagnostic standard, an international consensus meeting was held in 2013 to address this uncertainty. The expert panel defined bruxism as "a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible." The consensus also emphasized the circadian classification of bruxism into two distinct types: sleep bruxism and awake bruxism. To facilitate use in both clinical and research settings, a three-level diagnostic grading system was proposed (**Table 2**) [54].

Since no single test is considered sufficient to confirm the diagnosis of bruxism, multiple approaches are often used in combination. According to Koyano et al. [55, 56], five main diagnostic strategies are commonly employed in clinical practice.

4.1 Questionnaires

Questionnaires are widely used in both clinical practice and field studies to screen for bruxism. Their primary advantage is their ability to assess large populations in a short time. The most frequently used formats include yes/no responses to questions such as:

- Do you feel pain or fatigue in your jaw muscles upon waking?
- Do you experience tooth pain in the morning?
- Has anyone ever noticed that you grind your teeth during sleep?

Criterion	Description / Clinical sign
A—sleep sound	Repetitive or regular grinding/clenching sounds during sleep, reported by a bed partner or observer.
B—clinical findings *(at least one must accompany A)*	1. Pathological teeth consistent with sleep bruxism
	2. Morning jaw muscle pain or fatigue, or temporal headache
	3. Sensation of jaw locking or tightness upon awakening

Table 1.
Diagnostic criteria for sleep bruxism (American Academy of Sleep Medicine, 2014).

Level	Basis of diagnosis
Possible	Self-report only (questionnaire or interview)
Probable	Self-report + clinical examination showing bruxism-specific findings
Definite	Self-report + clinical signs + polysomnography (preferably with audio-video recording)

Table 2.
International consensus-based grading of bruxism.

- Have you ever noticed yourself grinding your teeth during the day?
- Do you catch yourself clenching your teeth during the day?
- Do you experience temporal headaches upon awakening?

According to Pintado et al., individuals clinically identified as bruxists answered "yes" to at least two of the six questions above. However, it is important to note that these diagnoses were not always confirmed using additional objective methods [57].

4.2 Clinical examination

The clinical diagnosis of bruxism is typically based on a combination of patient-reported symptoms and morphological and functional findings observed during examination. Key indicators that may support suspicion of parafunctional activity include:

- Reported grinding or clenching sounds during sleep by others
- Masseter muscle hypertrophy
- Morning masticatory muscle pain, fatigue, or temporal headache
- Temporomandibular joint clicking or locking
- Thermal sensitivity and pathological tooth wear
- Mucosal indentations on the cheek or tongue (**Figure 1**)
- Wear patterns consistent with normal or eccentric mandibular movements.

Sleep bruxism is most often brought to clinical attention by the patient's partner or family member. During a clinical examination, the presence of pathological wear on the incisal-labial and palatal surfaces of the anterior teeth, or the occlusal surfaces of the posterior teeth, typically supports the diagnosis. The severity of tooth wear can range from small, polished spots on the enamel to deep surface loss exposing secondary dentin (**Figure 2**). Experimental data have shown that intra-tissue fluid pressure increases during bruxism episodes, leading to muscle edema, which is expected to contribute to pain perception [58].

Another common objective sign is masseter muscle hypertrophy (**Figure 3**) [55]. While hypertrophy typically appears as a bilateral and symmetrical enlargement, unilateral cases have also been reported. Therefore, to determine whether the observed hypertrophy is indeed related to bruxism, a comprehensive clinical examination is necessary, and ambiguous cases should be referred to the appropriate medical specialty.

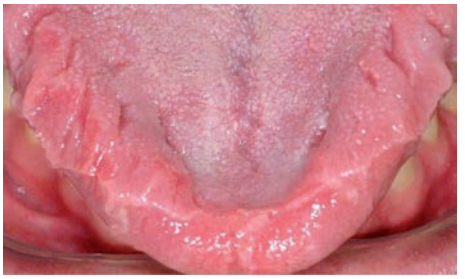

Figure 1.
Scalloped tongue (indentation marks on the lateral side).

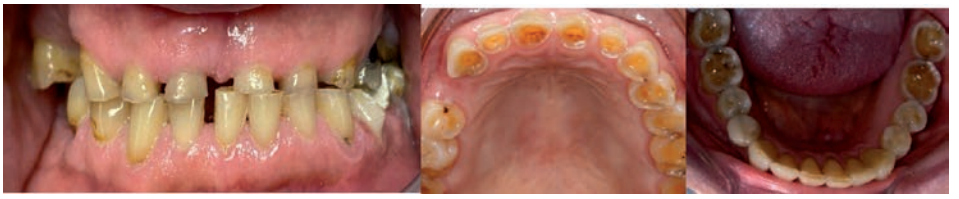

Figure 2.
Pathological wear patterns on incisal-labial and palatal surfaces of anterior teeth and occlusal surfaces of posterior teeth.

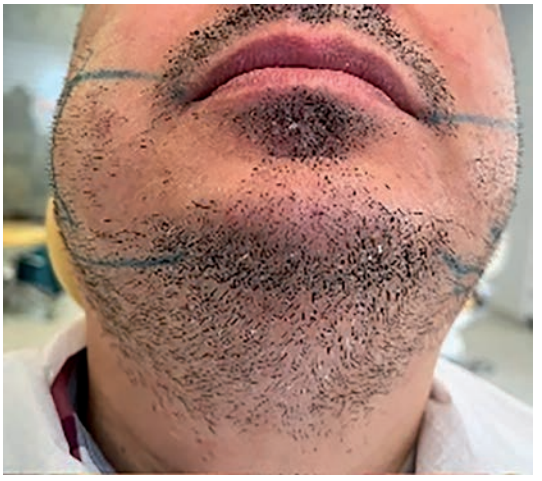

Figure 3.
Masseter muscle hypertrophy.

4.3 Objective measurement with intraoral appliances

To strengthen the diagnosis of bruxism in research settings, objective data beyond clinical observation are required. One such method involves analyzing wear marks on occlusal splints, which can provide quantitative insights into the presence and severity of bruxism [59, 60]. In more advanced setups, pressure sensors embedded in the splints dynamically record biting force, offering a detailed profile of parafunctional activity [61, 62]. These approaches improve diagnostic accuracy by enabling both wear pattern analysis and real-time force measurement.

4.4 Polysomnographic assessment

Polysomnography (PSG) is regarded as the gold standard for diagnosing bruxism. However, its use is limited by high equipment costs and the requirement for a sleep laboratory setting, which may alter the patient's natural parafunctional behavior due to the unfamiliar environment [55, 63]. The primary diagnostic marker for nocturnal bruxism in PSG is the electromyographic (EMG) activity of the jaw muscles. Additionally, PSG enables differentiation between bruxism and other orofacial activities, such as snoring, swallowing, or coughing, and can detect co-existing sleep disorders, such as obstructive sleep apnea (OSA) or insomnia. Furthermore, PSG allows for the simultaneous monitoring of cardiovascular and respiratory changes associated with sleep bruxism [38, 64, 65].

5. Treatment

The management of bruxism primarily targets three anatomical and functional domains: the masticatory muscles, the TMJ, and the dentition. Disorders originating from the TMJ or masticatory muscles are collectively classified under the umbrella term temporomandibular disorder. The origins of this concept trace back to James Costen's description of otological symptoms associated with TMJ dysfunction. Later, Ramfjord and Ash proposed categorizing these conditions as functional joint disorders [66]. Both the American Academy of Orofacial Pain (AAOP) and the American Dental Association (ADA) have adopted the term TMD to encompass all dysfunctions of the masticatory system.

According to the classification framework developed by J.P. Okeson, which is widely used for diagnostic and therapeutic guidance, TMDs are grouped into four major categories:

a. Masticatory muscle disorders

b. Temporomandibular joint disorders

c. Chronic mandibular hypomobility

d. Developmental anomalies.

The treatment of bruxism is tailored based on which of these groups is predominantly involved. For instance, in cases of muscle hyperactivity, physical therapy, biofeedback, or botulinum toxin injections may be utilized. When joint dysfunction is evident, occlusal splints, manual therapy, or intra-articular injections may be indicated. In the presence of tooth wear or malocclusion, occlusal adjustment or restorative approaches may be necessary. Treatment protocols should be individualized based on symptom severity, distribution of etiological factors, and the patient's general health status.

Given the multifactorial nature of TMD, no single therapy is sufficient for all cases, and interdisciplinary collaboration is often essential. A comprehensive treatment plan combining non-invasive and invasive strategies is typically required.

5.1 Reversible therapies

- Patient education and habit modification

- Physical therapy interventions
- Pharmacotherapy
- Psychological support
- Occlusal splint therapy.

5.2 Irreversible (invasive) interventions

- Occlusal adjustment
- Surgical procedures.

5.3 Alternative techniques

- Botulinum toxin
- Biofeedback therapy
- Hypnotherapy
- Anti-snoring devices.

This stepwise protocol advocates initiating treatment with low-risk conservative methods, progressing to more extensive interventions only when necessary.

5.4 Reversible treatment approaches

5.4.1 Patient education and behavioral modification

Patients are educated to recognize their bruxism habits and taught self-monitoring and behavioral control techniques. Exercises involving the tongue and palate, as well as stress reduction strategies, such as yoga or psychotherapy, may be recommended [67, 68].

5.4.2 Physical therapy

Modalities such as transcutaneous electrical nerve stimulation (TENS), ultrasound, and massage may improve muscle relaxation and circulation [69].

5.4.3 Psychiatric support

Counseling or psychotherapy focused on anxiety and stress management can enhance the patient's awareness of parafunctional habits, thereby helping reduce the frequency of clenching and grinding [68].

5.4.4 Pharmacotherapy

Medications such as muscle relaxants, benzodiazepines, and tricyclic antidepressants are commonly used for orofacial pain and sleep bruxism. In some cases,

dopamine agonists, propranolol, or botulinum toxin A may be added. However, the number of well-controlled studies supporting their efficacy remains limited [70].

5.4.5 Occlusal splint therapy

Among conservative treatments for the masticatory system, the use of interocclusal appliances (occlusal splints or night guards) is the most common approach [9]. Initially developed by Karolyi in 1901, splint design has evolved, with rigid acrylic appliances now preferred over soft materials due to superior stability, wear resistance, and adjustability [71, 72]. Soft splints are prone to deformation, discoloration, and lack of retention, leading to reduced clinical longevity [73–78].

The primary therapeutic goals of splints include [71–79]:

- Eliminating premature contacts
- Protecting teeth from wear
- Promoting muscle relaxation
- Reducing TMJ stress
- Supporting proper condylar positioning
- Monitoring adaptation to occlusal or vertical changes.

Their effect stems from occlusal redistribution, reduced proprioceptive drive, joint stabilization, and increased behavioral awareness. A minor vertical increase promotes muscle relaxation by restoring optimal fiber length. The perception of the splint as a "treatment device" may also elicit a placebo effect (Hawthorne effect) [80].

According to Laskin's classification, four main types are used clinically: stabilization splints, anterior/posterior bite plates, anterior repositioning splints, and soft splints [81].

5.4.5.1 Stabilization splints

Made of hard acrylic and covering the entire arch, these splints—referred to in the literature as Michigan, Fox, Tanner, flat-plane, or Ramfjord appliances—promote stable centric occlusion and provide anterior guidance during dynamic movements. While typically applied to the arch with more missing teeth, selection depends on skeletal and arch morphology [82]. It is one of the first options in temporomandibular disorders accompanied by pain and functional limitations [83].

5.4.5.2 Anterior bite plates

These thin acrylic appliances cover the upper anterior teeth and contact only the mandibular anterior teeth. By eliminating posterior contacts, they effectively reduce muscle hyperactivity in a short period. They are typically used for 3–6 days during initial therapy. Extended use may cause supraeruption of posterior teeth, leading to anterior open bite [11].

5.4.5.3 Anterior repositioning splints

These full-arch appliances guide the mandible slightly forward and downward from the intercuspal position, stabilizing the condyle-disc complex and allowing time for retrodiscal tissue adaptation. Some authors recommend continuous wear (including during meals) for 6–7 weeks, while others suggest nighttime-only use for 2–6 months [82].

5.4.5.4 Soft splints

Soft splints are appliances made of thermoplastic material. They have a specific thickness and do not require balancing or adjustment with opposing teeth. They maintain continuous contact with the opposing teeth. This splint is used for urgent intervention in patients with pain and dysfunction. Wright reported that occlusal changes occur in users of soft splints, and symptoms increase in patients using non-adjusted appliances. Additionally, it is noted that soft splints may trigger parafunctional activity [84, 85].

5.5 Alternative techniques

5.5.1 Botulinum toxin

Botulinum toxin (Botox) is an exotoxin produced by *Clostridium botulinum*. This toxin causes temporary inactivation of muscles and glands by blocking the release of acetylcholine at the neuromuscular junction, also known as the cholinergic motor nerve terminals. In the treatment of bruxism, Botox injections are typically applied to the masseter muscle. However, in studies considering the synergistic effect of the masseter and temporal muscles, injections have been administered to both muscles.

Treatment option	Indications	Evidence level	Notes
Behavioral counseling	Mild bruxism, awake episodes	Moderate	First-line approach; includes patient education, stress management, and habit reversal techniques
Physical therapy	Muscle pain, hyperactivity	Moderate	Jaw exercises, relaxation techniques, and posture correction, often combined with behavioral therapy
Occlusal splints (stabilization splints)	Sleep bruxism, tooth wear, TMJ overload	High	Reversible, protective; reduces dental wear and may alleviate muscle hyperactivity
Botulinum toxin injections	Resistant muscular hyperactivity, severe jaw pain	Low to moderate	Adjunctive therapy; effects are temporary and require repeat administration
Pharmacological therapy	Severe cases with pain or comorbidities	Low	Analgesics, muscle relaxants, or anxiolytics; use with caution due to side effects
Surgical intervention	Rare, severe TMJ dysfunction	Very low	Last resort; irreversible and high-risk; limited supporting evidence

Table 3.
Summary of bruxism treatment options, indications, and evidence levels.

There is a lack of sufficient studies comparing these two approaches. In recent years, the use of Botox in the treatment of both daytime and sleep bruxism has been a topic of discussion. It has been proven that botulinum toxin reduces bruxism symptoms [86]. The toxin inhibits the release of acetylcholine, a neurotransmitter responsible for glandular secretion and muscle contraction. This results in a reduction in muscle tone in the injected area. The substance effectively weakens the muscle for 3–4 months [87].

Table 3 summarizes the primary treatment modalities, their clinical indications, and levels of supporting evidence to aid clinical decision-making.

6. Conclusion

Bruxism is a multifactorial, dynamic condition influenced by anatomical, neurological, psychological, and genetic components [3, 7, 26]. Recent diagnostic advances, including classification systems and technological tools, have improved diagnostic accuracy and therapeutic targeting [53–55].

Conservative approaches—such as behavioral therapy, physical rehabilitation, and stabilization splints—remain the foundation of treatment [9, 66, 71]. In cases resistant to conventional methods, botulinum toxin serves as a viable adjunct [86]. Pharmacologic and surgical interventions are reserved for specific, severe presentations.

A multidisciplinary, personalized treatment plan based on the patient's clinical and psychological profile is essential for long-term success.

Future research on bruxism should aim to clarify the neurophysiological pathways involved in both sleep and awake forms, using longitudinal study designs that capture variation over time. The integration of wearable diagnostic technologies, including EMG sensors and bite force detectors, may offer real-time, ecologically valid data. Additionally, advances in genetic analysis and neuroimaging can enhance precision diagnostics and help define personalized treatment pathways. Standardization of outcome measures and longer follow-up periods is essential to assess the durability and safety of both conventional and emerging therapies.

Conflict of interest

The authors declare no conflict of interest.

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