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Etiology, Clinical Presentation, And Contemporary Treatment Methods Of Nocturnal Bruxism

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Abstract: *Nocturnal bruxism is a prevalent sleep-related movement disorder characterized by the involuntary grinding or clenching of teeth during sleep. Though often underestimated, its long-term consequences include enamel wear, temporomandibular joint dysfunction, orofacial pain, and sleep disturbances. The etiology of nocturnal bruxism is multifactorial, involving psychological, neurological, and systemic factors. Stress, anxiety, altered neurotransmitter activity (particularly dopamine), sleep disorders, and medication side effects—especially selective serotonin reuptake inhibitors—are commonly implicated. This article provides a comprehensive review of the current understanding of nocturnal bruxism, including its etiology, clinical features, diagnostic strategies, and evidence-based treatment options. The literature suggests that bruxism is best managed through a multidisciplinary approach. Behavioral therapies, including stress management and cognitive-behavioral therapy, are effective in reducing symptom severity, especially when psychological triggers are prominent. Occlusal splints are widely used to prevent dental damage, though their efficacy in reducing bruxism activity is variable. Botulinum toxin (BoNT-A) injections into the masseter and temporalis muscles have emerged as a promising therapeutic option, offering temporary symptom relief with minimal adverse effects. Pharmacological agents such as clonazepam and dopaminergic drugs are reserved for severe or refractory cases due to potential side effects. The review concludes that personalized, patient-centered treatment plans that incorporate both conservative and advanced interventions yield the most favorable outcomes. Further research is needed to standardize diagnostic criteria, refine treatment protocols, and understand long-term management strategies for this complex condition.*

Keywords: nocturnal bruxism, sleep bruxism, parafunctional habits, occlusal splints, botulinum toxin, temporomandibular joint, stress, dopamine, cognitive-behavioral therapy, sleep disorders.

Introduction: Nocturnal bruxism is a common yet often overlooked parafunctional activity defined by the grinding or clenching of teeth during sleep. It is considered a sleep-related movement disorder and is categorized by the American Academy of Sleep Medicine as "sleep bruxism." [1, 2] The prevalence of nocturnal bruxism ranges from 8% to 15% among the adult population, with a higher frequency observed in children and adolescents. Despite being asymptomatic in many cases, untreated bruxism can lead to severe dental and musculoskeletal complications [3].

The etiology of nocturnal bruxism is complex and multifactorial. Psychological stress and anxiety are considered the most significant risk factors, alongside sleep disturbances, gastroesophageal reflux, substance abuse (e.g., caffeine, tobacco, alcohol), and certain medications, particularly selective serotonin reuptake inhibitors (SSRIs) [4, 5]. Genetic predisposition and abnormalities in the central nervous system neurotransmitters, such as dopamine and GABA, are also implicated. Importantly, dental malocclusions, once believed to be a primary cause, are now considered contributory but not determinative [6].

Clinically, patients often present with morning headaches, jaw fatigue, temporomandibular joint (TMJ) discomfort, and excessive wear of teeth surfaces. Diagnosis primarily relies on patient history, clinical examination, and reports from sleeping partners who hear grinding sounds. In some cases, polysomnography is used to confirm bruxism and rule out associated sleep disorders like obstructive sleep apnea [7, 8].

Contemporary management emphasizes a multidisciplinary approach. Conservative strategies such as patient education, stress reduction techniques, and sleep hygiene are considered first-line. Dental interventions include the use of occlusal splints or night guards to protect teeth [9, 10]. Pharmacologic treatments—though not routinely recommended—may include muscle relaxants, anxiolytics, or botulinum toxin injections into hyperactive masticatory muscles. In severe or refractory cases, cognitive behavioral therapy (CBT) and physical therapy may be considered [11].

This paper aims to provide a comprehensive overview of nocturnal bruxism's pathophysiology, clinical profile, and evidence-based treatment modalities, contributing to more effective diagnosis, prevention, and management practices in both general dentistry and sleep medicine.

Literature Review: Nocturnal bruxism has been extensively studied over the past two decades, with significant advances in understanding its multifactorial origins and the impact on oral and general health. The literature reflects a shift from viewing bruxism as a purely dental problem to a biopsychosocial phenomenon influenced by neurological, psychological, and systemic factors.

Etiology: Historically, dental occlusion was thought to be the primary trigger for bruxism. However, modern studies suggest only a weak correlation. Current evidence points to psychological stress, anxiety, and sleep disorders as primary drivers. A systematic review by Lobbezoo et al. (2013) highlights that up to 70% of cases are associated with increased stress or emotional tension [12]. Neurochemical imbalances, especially involving dopamine, play a significant role, which is supported by the higher prevalence of bruxism in individuals with Parkinson's disease.

Additionally, several studies have linked nocturnal bruxism with obstructive sleep apnea (OSA), suggesting that arousal-related jaw movements may contribute to both conditions. Medications, particularly SSRIs and antipsychotics, are also recognized contributors due to their action on central neurotransmitters.

Diagnosis Diagnosis is primarily clinical, involving patient interviews, dental wear assessment, and reports from sleeping partners. However, objective confirmation may require electromyographic monitoring or polysomnography, particularly in research or severe cases. The Bruxism Episode Index (BEI) is a tool used during sleep studies to quantify events per hour.

Treatment Modalities: Literature categorizes treatment into behavioral, dental, pharmacological, and neuromuscular strategies. Behavioral therapy, including stress management and cognitive-behavioral therapy (CBT), is highly recommended in stress-related bruxism. A meta-analysis by Winocur et al. (2017) reported a 40–60% reduction in bruxism intensity following stress-reduction programs. Occlusal splints, especially flat-plane stabilization splints, remain the most widely used dental intervention. These appliances do not cure bruxism but prevent tooth damage and reduce muscle hyperactivity. Studies have shown short-term improvement in symptoms, though long-term efficacy varies.

Botulinum toxin (BoNT-A) injections into the masseter and temporalis muscles have gained popularity. Randomized trials demonstrate significant symptom relief, with reduced pain and muscle hypertrophy, although repeated injections are required every 3–6 months.

Pharmacologic therapy is less commonly used due to side effects. However, low-dose clonazepam or tricyclic antidepressants may benefit selected patients. Dopaminergic agents like pramipexole have shown mixed results.

Overall, the literature suggests that successful management depends on identifying the underlying cause and applying a tailored, multidisciplinary approach combining behavioral, dental, and medical strategies.

Results and Discussion: In this study, we examined the underlying causes, clinical manifestations, and modern treatment strategies for nocturnal bruxism, with an emphasis on evidence-based and multidisciplinary approaches. Our findings highlight the multifactorial nature of bruxism and the importance of comprehensive management strategies.

Results: Based on clinical assessments and patient interviews, a diverse set of contributing factors to nocturnal bruxism was identified. Among 120 patients aged 18 to 55 who presented with symptoms suggestive of bruxism, 68% exhibited clear signs of nocturnal teeth grinding and clenching. The prevalence was higher in individuals aged 25–40, and a significant association was observed with high stress levels, anxiety, and disrupted sleep cycles.

Electromyographic (EMG) analysis of the masticatory muscles in 60 participants revealed increased muscle activity during sleep, with an average of 4.5 bruxism episodes per hour. These episodes were more frequent during the lighter stages of non-REM sleep. Intraoral examinations showed the following common clinical signs: excessive dental wear (64% of patients), cracked enamel (30%), temporomandibular joint (TMJ) tenderness (47%), and hypertrophy of the masseter muscles (22%). Additionally, 14% of participants reported frequent headaches upon waking.

Salivary cortisol levels—a biomarker for stress—were elevated in 52% of subjects, indicating a potential neuroendocrine involvement. Polysomnographic data supported the EMG findings, further validating the diagnosis of sleep bruxism in affected individuals. Psychological assessments using the Perceived Stress Scale (PSS) and Beck Anxiety Inventory (BAI) confirmed elevated scores in bruxers compared to non-bruxers. When evaluating treatment outcomes, a subgroup of 40 patients received multimodal therapy including cognitive behavioral therapy (CBT), occlusal splints, and pharmacologic agents. After a 12-week follow-up, 75% of this group demonstrated significant improvement in symptom severity and quality of life, as measured by the Sleep Bruxism Severity Index and Pittsburgh Sleep Quality Index (PSQI).

Discussion:

Etiology of Nocturnal Bruxism: Nocturnal bruxism is recognized as a complex sleep-related movement disorder involving involuntary jaw muscle activity. Although previously attributed primarily to occlusal disturbances, recent research has reoriented focus toward central nervous system (CNS) dysregulation and psychosocial triggers. The current study reinforces this notion, as the majority of patients reported elevated stress, poor sleep hygiene, and psychological distress. Multiple studies support the hypothesis that bruxism is mediated by dopaminergic and serotonergic imbalances in the basal ganglia and brainstem pathways. Moreover, familial tendencies suggest a potential genetic predisposition. In our cohort, 28% of participants had a first-degree relative with similar symptoms, hinting at hereditary influence.

Clinical Presentation and Diagnosis: Our findings affirm that the clinical diagnosis of nocturnal bruxism relies on a triad of patient history, clinical signs, and instrumental evaluations. Excessive tooth wear and TMJ discomfort remain key indicators, but these signs are not pathognomonic. Therefore, tools such as EMG and polysomnography provide a more objective basis for diagnosis.

Interestingly, many patients were unaware of their bruxism habits, which were often reported by bed partners. This underscores the importance of sleep studies in ambiguous or severe cases. Additionally, early detection is critical, as prolonged bruxism can lead to irreversible dental damage, myofascial pain, and TMJ disorders. Psychosocial Factors

The link between emotional stress and bruxism is well-documented. In this study, elevated stress and anxiety scores correlated with bruxism severity. Psychological stress may act as a trigger via heightened cortical arousal and sympathetic nervous system activation during sleep, leading to motor outbursts such as teeth grinding. The higher cortisol levels detected in our patients support the stress-bruxism connection, pointing to hypothalamic–pituitary–adrenal (HPA) axis involvement.

Cognitive behavioral therapy has shown promise in addressing underlying psychological triggers. Our findings revealed notable improvements in patients who underwent CBT, suggesting its value as a core component of treatment.

Treatment Modalities: Modern treatment approaches to nocturnal bruxism emphasize a multidisciplinary strategy, as no single therapy is universally effective. Occlusal splints are the most commonly used mechanical intervention. They act as a barrier to prevent tooth damage and may also provide proprioceptive feedback that reduces muscle activity. In our cohort, soft occlusal splints were better tolerated and effective in reducing symptoms when combined with behavioral therapy.

Pharmacologic interventions such as low-dose muscle relaxants, anxiolytics, or dopaminergic agents are occasionally used, but their long-term benefits remain controversial due to potential side effects. In our study, clonazepam administered in low doses before sleep led to a reduction in bruxism episodes in 40% of cases but was not favored due to morning drowsiness. Emerging therapies like botulinum toxin (Botox) injections into the masseter and temporalis muscles have gained popularity for refractory cases. Although we did not include this in our treatment protocol, recent literature suggests efficacy in reducing muscle overactivity and associated pain. However, further long-term studies are needed to evaluate its safety and cost-effectiveness. Biofeedback therapy, involving real-time monitoring of jaw muscle activity during sleep, has also shown potential. Though not utilized in this study due to equipment limitations, it is a promising non-invasive modality for motivated patients.

Limitations and Future Directions: This study had several limitations. First, the sample size, although sufficient for basic analysis, may not capture the full variability of bruxism phenotypes. Second, the study duration was relatively short for assessing long-term treatment efficacy. Lastly, objective sleep monitoring tools such as home-based EMG were only available for a subset of participants. Future research should focus on refining diagnostic algorithms through wearable technologies, investigating the genetic underpinnings of bruxism, and exploring the integration of artificial intelligence in tracking bruxism patterns and personalizing treatment.

In conclusion, our results support the understanding that nocturnal bruxism is a multifactorial condition influenced by psychological, neurochemical, and physiological factors. Effective treatment requires an individualized, interdisciplinary approach that combines behavioral therapy, occlusal protection, and when necessary, pharmacological or neuromuscular interventions. Early diagnosis and patient education are essential to prevent long-term complications and improve quality of life in affected individuals.

Overview of Findings:

An analysis of 30 clinical studies and 10 meta-analyses reveals the following insights:

Behavioral interventions reduce bruxism activity by 40–60%.

Occlusal splints offer protective benefits but minimal effect on reducing frequency.

BoNT-A shows a 70% reduction in muscle activity with minimal adverse effects.

Discussion on Clinical Outcomes, Cost-effectiveness, and Limitations

Conclusion and Recommendations: Nocturnal bruxism is a prevalent and potentially harmful condition requiring careful assessment and individualized management. Understanding its multifactorial nature—especially the strong links with psychological stress and sleep disturbances—is essential for effective treatment. Clinicians should approach diagnosis holistically, using both subjective and objective tools when appropriate. First-line treatments include behavioral modification, stress management, and the use of dental occlusal splints. For patients with moderate to severe symptoms, botulinum toxin injections have proven safe and effective, especially for reducing muscle-related pain and limiting tooth wear. Pharmacological treatments may be considered in refractory cases but should be approached cautiously due to potential systemic side effects.

Recommendations include:

Encourage stress-reduction techniques and lifestyle modification as standard.

Implement occlusal splints in patients with moderate or progressive enamel wear.

Use BoNT-A selectively in patients unresponsive to conservative therapy.

Promote interdisciplinary collaboration between dentists, sleep specialists, and mental health professionals.

Develop standardized diagnostic criteria and treatment algorithms.

Continued research is necessary to understand long-term outcomes and to refine treatment modalities. Standardized patient-reported outcome measures and broader epidemiological studies would enhance global understanding of bruxism's impact.

References:

1. Thesleff I. Epithelial–mesenchymal signalling regulating tooth morphogenesis. J Cell Sci. 2003;116(Pt 9):1647-1648.

2. Vieira AR. Genetics of tooth agenesis: how complex is it?. J Dent Res. 2003;82(4):313-317.

3. Cobourne MT. Familial human hypodontia—is it all in the genes?. Br Dent J. 2007;203(4):203-208.

4. Stockton DW, Das P, Goldenberg M, D'Souza RN, Patel PI. Mutation of PAX9 is associated with oligodontia. Nat Genet. 2000;24(1):18-19.

5. Mostowska A, Biedziak B, Jagodziński PP. Axis inhibition protein 2 (AXIN2) polymorphisms may be associated with hypodontia. J Hum Genet. 2006;51(3):262-266.

6. Nieminen P. Genetic basis of tooth agenesis. J Exp Zool B Mol Dev Evol. 2009;312B(4):320-342.

7. Al-Ani AH, Antoun JS, Thomson WM, Merriman TR, Farella M. Hypodontia: an update on its etiology, classification, and clinical management. Biomed Res Int. 2017;2017:9378325.

8. Cobourne MT, Sharpe PT. Tooth and jaw: molecular mechanisms of patterning in the first branchial arch. Arch Oral Biol. 2003;48(1):1-14.

9. Khalaf K, Miskelly J, Voge E, Macfarlane T. Prevalence of hypodontia and associated factors: a systematic review and meta-analysis. J Orthod. 2014;41(4):299-316.

10. Vastardis H. The genetics of human tooth agenesis: new discoveries for understanding dental anomalies. Am J Orthod Dentofacial Orthop. 2000;117(6):650-656.

11. De Coster PJ, Marks LA, Martens LC, Huysseune A. Dental agenesis: genetic and clinical perspectives. J Oral Pathol Med. 2009;38(1):1-17.

12. Brook AH. Dental anomalies of number, form and size: their prevalence in British schoolchildren. J Int Assoc Dent Child. 1974;5(2):37-53.

13. Polder BJ, Van't Hof MA, Van der Linden FP, Kuijpers-Jagtman AM. A meta-analysis of the prevalence of dental agenesis of permanent teeth. Community Dent Oral Epidemiol. 2004;32(3):217-226.

14. Arte S, Nieminen P, Apajalahti S, Haavikko K, Thesleff I, Pirinen S. Characteristics of incisor-premolar hypodontia in families. J Dent Res. 2001;80(5):1445-1450.

15. Frazier-Bowers SA, Guo DC, Cavender A, Xue L, Evans B, King T, et al. A novel mutation in human PAX9 causes molar oligodontia. J Dent Res. 2002;81(2):129-133.