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Sleep Dentistry: Using Supplements for Bruxism

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Abstract

Introduction: *There are few studies that consider nutrient insufficiency in the pathophysiology of bruxism, and this review draws attention to these factors, which interfere with the main causal component of bruxism, which is stress.*

Objective: *The objective of this study was to investigate the relationships between sleep bruxism the main vitamin and nutrient deficiencies that are associated with psychological symptoms and bruxism.*

Methodology: *The following databases were searched to select articles: PubMed until December 2024, with a total of 3272 articles were found for the terms sleep bruxism, until 12/31/2024. The selection of articles on bruxism and vitamins, by title and abstract, included 316 articles. Finally, after reading the full articles, a total of 20 studies were included in this review.*

Results: *Due to the scarcity of clinical evidence to evaluate the nutritional importance in BS, we carried out a synthesis with a literature review on the importance of understanding the influence of nutrients on neuropsychological mechanisms in BS, which provides valuable insights into the effects of nutritional interventions on deep craniofacial nociception and stress.*

Discussion: *Exogenous oxidants from unhealthy dietary patterns may contribute to peripheral and central pro-inflammatory immune signaling, leading to peripheral and central sensitization. Furthermore, diets rich in bioactive compounds are suggested to contribute to CPC pain control. High dietary intake of ultra-processed foods impacts diet quality and shows adverse health outcomes. In this context, the role of nutrition in TMD is neglected.*

Conclusion: *Instead of focusing on pharmacological agents, we draw attention to homeostatic disorders and deficiencies in important elements such as vitamin D, magnesium, and omega-3 fatty acids for the treatment of bruxism of sleep.*

Keywords: *Sleep bruxism, vitamin D, nutrients, magnesium.*

Introduction

Sleep bruxism (SB) is defined as involuntary and repetitive activity of the jaw muscles characterized by clenching or grinding of the teeth and/or contracting or thrusting of the jaw, which can occur during sleep. It is characterized as rhythmic (phasic) or non-rhythmic (tonic). [1-3] BS can result in deleterious effects, including loss of tooth enamel, fracture of teeth or restorations, hypersensitivity or pain in the teeth, and headache. [1-3] A systematic review evaluated the global prevalence of sleep bruxism and awake bruxism in pediatric and adult populations and showed that the global prevalence of sleep bruxism is 21% and the awake prevalence is 23%. The occurrence of sleep bruxism, based on polysomnography, was estimated at 43%. It has been observed that age is a significant factor in the associated with deprivation of the deep sleep phase. [1-3] *Fulek M et al*. highlighted differences in sleep architecture and deprivation of the deep sleep phase, in which stage 3 non-REM sleep was significantly shorter in severe bruxers. Differences were also noted in stage 2 non-REM sleep and in the REM sleep phase. [1-3]

It is well recognized that stress plays a significant role in the etiopathogenesis of bruxism. Activation of the hypothalamic-pituitary-adrenal (HPA) axis and subsequent release of corticosteroids leads to increased muscle activity. [3-6]

Neurological studies have shown that chronic exposure to stress induces neurodegeneration of important neuronal structures and destabilization of the mesocortical dopaminergic pathway. [5-9] These disruptions impair the ability to counteract HPA axis hyperactivity and disinhibit involuntary muscle activity while at the same time activating the amygdala. Recent evidence shows that hyperactivation of the amygdala under stressful stimuli causes rhythmic jaw muscle activity by overactivating the midbrain and trigeminal motor nuclei. [3-7]

In addition to neurophysiopathological mechanisms, there are negative effects of certain vitamin and mineral deficiencies, such as vitamin D, magnesium, and omega-3 fatty acids, on the central nervous system. Increased sensitivity to stress reduces the ability to respond effectively to overactivation of the sympathetic nervous system and can also lead to vitamin and mineral deficiencies. [4-9]

Studies have shown that calcium and magnesium deficiencies may be implicated in the development of bruxism through regulation of the nervous system and muscle function. There is also epidemiological data linking low concentrations of vitamin D to various types of sleep problems, such as sleep bruxism. [4-9]

Physiological factors, including sleep disorders, abnormal levels of neurotransmitters, and specific medications, may contribute to the development of bruxism. Lifestyle factors such as alcohol, caffeine, tobacco, and drug abuse have also been associated with bruxism. [10-13]

There are few studies that consider nutrient insufficiency for the pathophysiology of bruxism, and this review draws attention to these factors, which interfere in the main causal component of bruxism, which is stress. [10-13]

Objective

The aim of this study was to investigate the relationships between sleep bruxism and the main vitamin and nutrient deficiencies that are associated with psychological symptoms and bruxism.

Methodology

The following databases were searched for article selection: PubMed until December 2024, with a total of 3272 articles found for the terms 'sleep bruxism' until 12/31/2024. The selection of articles on bruxism and vitamins, by title and abstract, included 316 articles. Finally, after reading the full articles, a total of 20 studies were included in this review.

Results

Due to the scarcity of clinical evidence to assess the nutritional importance in SB, we performed a synthesis with a literature review on the importance of understanding the influence of nutrients on neuropsychological mechanisms in SB, which provides valuable insights into the effects of nutritional interventions on deep craniofacial nociception. and stress. [10-13]

Neuro Pathophysiology of Sleep Bruxism

My etiology is related to my activities during sleep.The sleep cycle is composed of non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. [14-17]

The natural sleep cycle begins with deep NREM sleep (stages 1 and 2; stages N1 and N2), reaches deep NREM (stages 3 and 4; stage N3), and then transitions to NREM and light REM sleep. The BS is followed by microarousal, representing temporary activity of the central and sympathetic nerves. [14-17]

SB and microarousals are highest during stages 1 and 2 of NREM. [14-17] Slow-wave sleep, an index of deep sleep, decreases after the onset of SB. A shift in the sympathetic-vagal balance toward increased sympathetic activity begins about 8 minutes before the onset of SB, which highlights poor sleep quality. [14-17] *Fritze et al.* found increased levels of salivary cortisol in bruxers, which were correlated with strong circadian rhythms associated with peak levels during periods of activation. Using experimental stress, they observed increased masseter activity, which returned to baseline levels after relaxation. [14-17]

Stress-induced muscle hyperactivity has been associated with panic attacks, and teeth grinding, bruxism, has been shown to be more frequent. [14-17]

Stress has been well documented as playing a significant role in the etiopathogenesis of bruxism. Activation of the hypothalamic-pituitary-adrenal (HPA) axis and subsequent release of corticosteroids leads to increased muscle activity. [14-17]

Neurological studies have shown that chronic exposure to stress induces neurodegeneration of important neuronal structures and destabilization of the mesocortical dopaminergic pathway. [14-17]

These disruptions impair the ability to counteract HPA axis hyperactivity and disinhibit involuntary muscle activity while at the same time activating the amygdala. [14-17]

Recent evidence shows that hyperactivation of the amygdala under stressful stimuli causes rhythmic jaw muscle activity by overactivating the midbrain and trigeminal motor nuclei. [14-17]

Nutritional deficiency

Nutrients play an important role in regulating sleep well-being. Foods and meals that contain enough protein, carbohydrates, and fats are essential for maintaining quality sleep. The amino acid tryptophan, the precursor of melatonin, has a positive effect on sleep. [17-20] Meals containing carbohydrates with a low glycemic index, low glycemic load, and high fiber content can improve sleep quality. Caffeine causes a decrease in the total amount and quality of sleep and delays sleep induction. Carbohydrates, lipids, amino acids, and vitamins are related to sleep disorders. [17-20]

Overwhelming evidence points to insufficiencies and deficiencies of elements necessary for homeostasis and normal function of the central nervous system (CNS), with vitamin D (vit. D) being the most important; calcium, magnesium, and omega-3 fatty acids are the most studied. [17-20]

Sufficient concentrations of vitamin D and calcium are necessary to regulate nervous system and muscle function, including jaw muscle contraction. [17-20]

Although there is no evidence showing the exact mechanism linking low vitamin D to muscle disorders, of sleep. The problem appears to be multifactorial. Some studies have shown a link between deficient vitamin D levels and nonspecific skeletal pain, which can disrupt sleep. [17-20]

Other studies have shown that vitamin D supplementation can relieve pain and improve sleep in individuals complaining of chronic pain. [17-20] Furthermore, vitamin D has been reported to

play a role in immune regulation, and vitamin D deficiency may alter immunomodulation and increase the production of cytokines that have roles in sleep. [17-20]

A narrative review aims to assess the current understanding of the effectiveness of dietary habits and nutritional approaches for the treatment of painful TMDs, elucidating the following nutritional elements: Vitamin D, vitamin B, vitamin C, magnesium, zinc, omega-3 fatty acids, sulfur, citral, crocin, lutein, limonide, and quercetin. [17-20]

Association of vitamin D with bruxism

Vitamin D is necessary for a healthy musculoskeletal system, as it plays an important role in the metabolism of calcium and phosphorus, not only by increasing their intestinal absorption and regulating the production of parathyroid hormone (PTH). [17-20]

Recent evidence has revealed a strong association between bruxism, particularly sleep (central) bruxism, and vitamin D deficiency. *Alkhatatbeh MJ et al*. demonstrated that sleep bruxism was associated with vitamin D deficiency and low calcium intake, and frequent headache was reported in individuals with sleep bruxism compared to controls. [17-20]

Alkhatatbehet al. showed that there is a significant link between vitamin D deficiency and sleep bruxism, with 60% of patients with bruxism exhibiting low vitamin D levels compared to 34% of controls. [17-20]

Furthermore, they reported that only 26% of bruxers had a daily dietary calcium intake of >600 mg/day, compared with 42% of controls, which may be explained by the neuroprotective role of vitamin D, as low levels of vitamin D disrupt calcium homeostasis, affecting neuron excitability. [17-20]

Furthermore, since vitamin D is responsible for calcium homeostasis, low levels of vitamin D will result in low serum calcium levels, hypocalcemia, which has an immediate effect on neuromuscular function and the potential to cause spasms and muscle cramps. [17-20]

According to *Allaf* and *Abdul-Haket et al.,* vitamin D insufficiency or deficiency was shown to increase with the severity of bruxism, while in non-bruxists, insufficiency and deficiency were present in 41 and 16% of individuals, respectively. [17-20]

In individuals with mild bruxism, these numbers appeared to increase to 50% and 30%, respectively, and in moderate and severe bruxism to 58%, while in extremely severe bruxism, insufficiency and deficiency may reach 72% in total. [17-20]

Implication of agnesium in bruxism

The potentially beneficial impact of Mg on glucose and lipid metabolism, innate and adaptive immunity, and the nervous system deserves special attention. [17-20]

The possible involvement of magnesium in the pathogenesis of bruxism may be further supported by the fact that hypomagnesemia is observed in burning mouth syndrome (BMS), particularly in cases where the tongue is involved. BMS is caused by hyperactivity of the somatosensory fibers of the trigeminal nerve and loss of central inhibition. This hyperactivity can be caused by parafunctional habits. [17-20]

Another important function of Mg is that it exhibits a direct enhancing effect on serotonin 5-HT1A receptor transmission by acting as a cofactor for tryptophan hydroxylase, which plays an important role in the dopaminergic system. Mg levels are inversely correlated with estrogen levels, showing a sex-related difference. [17-20]

Experimentally induced Mg deficiency in animal studies has been associated with disrupted sleep patterns, while an increase in the amplitude of daily sleep change and delta power of slow-wave sleep has also been observed. [17-20]

Chronic sleep deprivation in humans is associated with progressive decreases in intracellular Mg levels, reduced duration of cardiopulmonary exercise, and increased hypersensitivity to sympathetic nerve stimulation. [17-20]
According to *Sarchielli et al.*, patients who suffered from migraine and tension headache, very common symptoms in patients with bruxism and TMD, presented significantly lower levels of serum and salivary Mg. [17-20]

This occurs because hypomagnesemia makes cerebral arteries more sensitive to CO2, which promotes cerebral vasospasm and headache. [17-20] Hypomagnesemia is also associated with exacerbated neural excitability, migraine, orofacial tardive dyskinesia, and increased anxiety, symptoms that can be improved by combined supplementation of Mg and B6. Mg functions as a structural or catalytic component of enzymes, as well as substrates, in hundreds of enzymatic processes. [17-20]

It is involved in several processes: ion channel activity and signal transduction, membrane stabilization, aerobic and anaerobic metabolism, and proliferation, cell proliferation, differentiation and apoptosis, and angiogenesis. [17-20]

Mg deficiency is associated with headaches, hyperemotionality, generalized anxiety, insomnia, asthenia, depressive states, muscle weakness, numbness and cramps, exacerbations of bronchial asthma, increased risk of stroke, progression of diabetes mellitus, and heart failure. congestive, worse control of arterial hypertension. [17-20]

Discussion

The present review has certain limitations, while there is also a lack of data on the optimal duration of nutrient supplementation to achieve the desired outcome. [17-20] Exogenous oxidants from unhealthy dietary patterns may contribute to peripheral and central proinflammatory immune signaling, leading to peripheral and central sensitization. [17-20]

Furthermore, diets rich in bioactive compounds are suggested to contribute to the control of CPC pain. High dietary intake of ultra-processed foods impacts diet quality and shows adverse health outcomes. [17-20]

In this context, the role of nutrition in bruxism is neglected. [17-20] However, despite the small number of studies addressing these issues and their associated limitations, the existing evidence supports the rationale that further research should be conducted in this field, with the aim of further increasing the current understanding of this complex and multifactorial condition and, hopefully, providing affected individuals with alternative and more effective therapeutic modalities. [17-20] Future studies should focus on assessing whether concentration changes have a causal effect or are the result of chronic stress exposure and whether restoring nutrient balance has a significant impact on the pathogenesis and chronicity of bruxism. [17-20]

Conclusion

Rather than focusing on pharmacological agents, we draw attention to homeostatic disturbances and deficiencies in important elements such as vitamin D, magnesium, and omega-3 fatty acids for the treatment of sleep bruxism.

Its neuroprotective properties, its ability to reduce oxidative stress to suppress the HPA and LC axes, and its positive action on neuroplasticity and neuronal growth in the hippocampus, particularly in the case of omega-3.

A decrease in the levels of these elements in the brain is associated with increased anxiety, increased neuromuscular excitability, muscle spasms, pain, and thus bruxism.

It is worth noting that appropriate and individualized supplementation of these nutrients appears to reduce or alleviate the neurological and musculoskeletal symptoms of bruxism.

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