Sleep Bruxism and Obstructive Sleep Apnea. Prescription of a Mandibular Advancement Device from a Dental Perspective *

Bruxismo del sueño y apnea obstructiva del sueño. Prescripción de un dispositivo de avance mandibular desde una perspectiva dental

Bruxismo do sono e apneia obstrutiva do sono. Prescrição de um dispositivo de avanço mandibular sob a perspectiva odontológica

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ABSTRACT

Bruxism and obstructive sleep apnea are two entities that occur together in a high number of patients. The consequences of these are relevant both for oral health and for the general health and quality of life of those affected. Identification, diagnosis and treatment are an essential part of the clinical activity of dentists. The use by dentists of mandibular advancement devices allows these two problems to be addressed with a single device. For the proper treatment and management of these patients, it is necessary for the dentist to have adequate training. In this review article we present an updated summary of the scientific evidence on the association of bruxism and apnea, as well as management by the dentist with mandibular advancement devices.

Keywords: bruxism; dentistry; mandibular advancement device; obstructive sleep apnea; sleep bruxism; sleep disorders; occlusal splint

RESUMEN

El bruxismo y la apnea obstructiva del sueño son dos entidades que se presentan conjuntamente en un elevado número de pacientes. Las consecuencias de estas son relevantes tanto para la salud oral como para la salud general y la calidad de vida de los afectados. La identificación, el diagnóstico y el tratamiento forma parte esencial de la actividad clínica de los odontólogos. El uso por parte de los odontólogos de los dispositivos de avance mandibular permite el abordaje de estos dos problemas con un único dispositivo. Para el adecuado tratamiento y manejo de estos pacientes es necesario que el odontólogo tenga una adecuada formación. En este artículo de revisión presentamos un resumen actualizado de la evidencia científica sobre la asociación de bruxismo y apnea, así como del manejo por parte del odontólogo con dispositivos de avance mandibular.

Palabras clave: apnea obstructiva del sueño; bruxismo; bruxismo del sueño; desórdenes del sueño; dispositivo de avance mandibular; férula oclusal; odontología

RESUMO

O bruxismo e a apneia obstrutiva do sono são duas entidades que ocorrem juntas num elevado número de pacientes. As consequências destas são relevantes tanto para a saúde oral como para a saúde geral e qualidade de vida das pessoas afetadas. A identificação, o diagnóstico e o tratamento são parte essencial da atividade clínica do médico dentista. A utilização por dentistas de dispositivos de avanço mandibular permite que esses dois problemas sejam resolvidos com um único dispositivo. Para o tratamento e manejo adequado desses pacientes é necessário que o dentista tenha formação adequada. Neste artigo de revisão apresentamos um resumo atualizado das evidências científicas sobre a associação de bruxismo e apneia, bem como o manejo pelo dentista com dispositivos de avanço mandibular.

Palavras-chave: apneia obstrutiva do sono; bruxismo; bruxismo do sono; dispositivo de avanço mandibular; distúrbios do sono; odontologia; tala oclusal

INTRODUCTION

Dentists see hundreds of patients daily. Some of them come with dental wear, abrasions, cracks, and even broken teeth, dentures and implants. It is evident that the cause of many of these clinical signs is bruxism. It is also likely that many patients with these conditions have sleep apnea, which is the cause of bruxism, as many clinical studies show. The purpose of this literature review supported by a case description was to analyze and synthesize the current evidence on bruxism and its association with sleep apnea, and how the use of mandibular advancement devices can mitigate the negative effects of both pathologies. Therefore, the prescription or use of a mandibular advancement device can be done from an oral health care perspective.

Sleep Bruxism

Sleep bruxism (SB) is defined as chewing muscle activity that occurs during sleep and is characterized by rhythmic or non-rhythmic chewing muscle activity (RMMA) (1-3). BS is classified as centric bruxism, which involves the involuntary clenching of the upper and lower teeth in a dental contact position, and eccentric bruxism, in which teeth grinding occurs in lateral or sliding movements (1). The prevalence of SB ranges between 22 % and 30 % in adults, is higher in men than in women, and tends to decrease with age (3-4). The etiology of BS is multifactorial and may include genetics and neuromuscular, emotional, and environmental factors (5).

BS can have negative consequences on teeth such as wear, fractures, fissures, breakage or loss of restorations or implants, and increased tooth sensitivity (1,6-8). In oral tissues, BS can be the cause of muscle tension, orofacial pain, and joint dysfunction (1). In addition, it can contribute to sleep disorders such as insomnia and headaches, which impact quality of life (1).

The diagnosis of BS is based on clinical evaluation, medical and dental history, and observation of characteristic signs and symptoms (2). Sleep monitoring techniques, such as electromyography and polysomnography, can provide objective data on muscle activity during sleep and help confirm the diagnosis (9-10). Some disadvantages of these tests are that polysomnography is expensive, time-consuming, and has been associated with the risk of misdiagnosis in the absence of bruxism (8). In practice, dentists frequently use the presence of clinical signs of occlusal wear patterns on natural teeth or restorative materials to diagnose bruxism. Likewise, patients recognize that they grind or clench their teeth and report it to the dentist (8).

The management of SB includes multidisciplinary approaches that address both neuromuscular aspects and emotional and lifestyle factors. Intraoral devices, cognitive behavioral therapy, relaxation techniques, and sleep hygiene education may be part of the therapeutic strategies. Individualizing treatment is essential since the causes and manifestations of BS can vary widely between people (1,11).

Treatment of BS with Splints

The treatment of SB with occlusal splints is one of the most used strategies to address this neuromuscular disorder (11). Occlusal splints are custom oral devices designed to modify the relationship between the upper and lower dental arches and relieve excessive tension in the chewing muscles during sleep. This reduces involuntary clenching and grinding of the teeth (12). The mechanism of action of occlusal splints in the treatment of BS includes relieving muscle tension by creating a uniform contact surface and reducing premature contact points between teeth; protect teeth by acting as a physical barrier between dental arches to prevent damage to dental structures; distribute chewing forces in a more balanced way; minimize excessive loading on specific dental areas; and reposition the jaw to a stable position that reduces pressure on the joint (13).

The treatment of BS has also been carried out with mandibular advancement devices (MAD) with the same purpose as occlusal splints, that is, to reduce the number of bruxism events, as well as to protect the teeth (10-11,14-16). The use of a MAD is associated with a notable reduction in BS motor activity (14). The number of sleep bruxism events per hour shows a clinically significant reduction after the use of a MAD (15). Additionally, it has been proven that MAD reduces the intensity of SB (10).

Snoring and Obstructive Sleep Apnea (OSA)

Snoring is a characteristic noise during breathing due to the vibration of the tissues due to the partial obstruction of the air flow and that originates at the level of the upper airway (17). OSA can be an intermittent, repetitive, partial (hypopnea) or total (apnea) obstruction of the upper airway during sleep. Such pauses can last several seconds and lead to pauses in breathing and episodes of hypoxia (decreased oxygen levels in the blood). Apnea episodes are usually followed by brief awakenings that allow people to regain normal breathing, although they are often unaware of such awakenings (18).

OSA is an independent risk factor for a number of conditions including hypertension, heart disease, stroke, and type 2 diabetes. It is also a risk for motor vehicle accidents and all-cause mortality (19-22). OSA is common and it has recently been estimated that more than one billion people worldwide suffer from it (18).

As mentioned before, the diagnosis of OSA is made through sleep studies with polygraphy or polysomnography, which record a series of physiological parameters during sleep, such as brain activity, muscle activity, heart rate, and oxygen saturation (23). Polysomnography is the test of choice, since it is the most complete to evaluate all sleep pathologies, although more expensive, while polygraphy is a simplified study for respiratory disorders (24). The severity of OSA is determined by clinical and polysomnographic parameters with the apnea-hypopnea index (AHI), which refers to the number of apneas and hypopneas per hour of sleep and allows cases to be classified into different degrees of severity: mild from 5 to 15, moderate from 15 to 30, and severe with more than 30 events per hour (24). Classification based on AHI is only one of the criteria used to evaluate the severity of OSA. Other factors such as blood oxygen saturation, duration of breathing pauses, and the presence of symptoms and comorbidities are also considered when determining severity and establishing the appropriate treatment approach (24). Regarding the pathophysiology of OSA, 4 key factors or phenotypes that contribute to its pathogenesis have been described. The first factor is the increased collapsibility of the airway due to the narrowing of the pharynx (anatomical factor), such as obesity or hypertrophy of the tonsils. The other three factors are functional (not anatomical) and consist of the response of the muscles during sleep (muscle factor), the hypersensitivity of the chemoreceptors to hypoxia and hypercapnia (instability of respiratory control or loop gain), and the ease that the individual has a transient or arousal awakening that allows the passage of air to open (awakening threshold) (25).

Treatment for sleep-disordered breathing can vary depending on the severity and underlying cause. It includes general measures, such as losing weight, avoiding alcohol and tobacco before bed, and using mandibular advancement devices, continuous positive airway pressure (CPAP), and surgery (24).

Treatment of OSA with MAD

MADs are a treatment alternative for mild, moderate, and severe snoring and sleep apnea. MADs modify the position of the jaw, soft palate and tongue, keeping the airways open and reducing the obstruction that causes snoring and apnea. The main effect of MAD is anatomical, in particular, thanks to the increase in lateral velopharyngeal diameter, improvement in airway patency, and reduction in collapsibility and obstructive respiratory events (26). The prescription of a MAD for the treatment of OSA should be made by a sleep specialist and the treatment should be performed by a dentist with adequate sleep training (23,27).

Anteroinferior displacement of the mandible increases the space available for soft tissues (28). The chin, tongue, and soft palate move in the same direction (29), which places greater stress on these structures and makes them less likely to block the airway (30). The displacement of the mandible pulls the superior constrictor muscle laterally and the lateral pharyngeal wall, widening the airway (31). The displacement of the mandible in turn changes the position of the hyoid bone to a more anterior and superior position (32). The distance between the mandible and the hyoid is decreased by pulling the hyoid muscles forward and improving the space of the upper airways (33). The MAD also has an effect on the epiglottis since the displacement of the hyoid affects the hyoepiglottic ligament, changing its position and inclination (34). Added to this is that MAD reduces collapse at higher levels, all of which helps reduce epiglottis collapse (34).

What was described in the previous paragraph increases the anteroposterior and lateral diameters, as well as the area and volume of the airways in the oropharynx and velopharynx (30-31, 35). The increase in volume, area, and diameter has been confirmed by 3D video fluoroscopy, computed tomography, computational fluid dynamics analysis, magnetic resonance imaging (MRI), and drug-induced sleep endoscopy (DISE) (28,31,34). These changes improve muscle function (36). A larger, less collapsible upper airway requires less activity of the dilator muscles to keep the air passage open (36). The MAD gives the muscles a better position, with the proper direction and length of the muscle fibers to fulfill their function (37). Additionally, functional changes have been observed with the use of MADs by reducing resistance in the velopharyngeal, increasing inspiratory flow and making the airway less collapsible (30,34).

Association Between SB and OSA

OSA and SB are two disorders that may share a complex relationship in the context of sleep. Lagana, *et al.* (38) found a significant correlation between bruxism and OSA in almost 20% of a group of participants with bruxism who also breathed through their mouth during sleep. On the other hand, Winck, *et al.* (39) found a close relationship between SB and OSA, with a statistically significant positive association between the rate of bruxism and the rate of phasic events during OSA episodes. Although some researchers have suggested that SB may be a compensatory mechanism in response to upper airway obstruction in patients with OSA, others have proposed that the two conditions may share a common etiology related to sleep disorders or neurobiological factors (4). The significant prevalence of SB in patients with OSA has important clinical implications for management and highlights the need for dentists to evaluate and manage SB in patients with OSA, and conversely, evaluate the presence of OSA in patients with clear signs of bruxism (4).

Several studies in patients with OSA have observed that apnea and hypopnea events are frequently followed by RMMA (9). Those studies suggested that RMMA occurs to reposition the jaw and prevent the

upper airway from collapsing (9). In patients with SB and OSA, 55 % of respiratory events preceded rhythmic RMMA, 25 % of events were the opposite, while 20 % of events had no temporal association (40).

There are several alternatives for the joint diagnosis of SB and OSA. One is polysomnography, which presents difficulties in accurately diagnosing BS in a single night, apart from being expensive, time-consuming, and associated with the risk of misdiagnosis (8). On the other hand, the diagnosis of BS by identifying dental wear is immediate, is less expensive than polysomnography, and is made by inspecting the tooth surface (6). Furthermore, a significant correlation has been established between clenching in dex and AHI (41) and the severity of tooth wear with the severity of OSA (8).

Finally, occlusal splints used in bruxism to protect the teeth are associated, according to several studies, with a risk of worsening OSA (42-43).

Treatment of Bruxism and OSA with MAD

Scientific evidence highlights that the use of MAD significantly decreases RMMA (3), as well as time-related RMMA for respiratory awakenings in patients with OSA (9-10). For those non-respiratory events, MAD had no significant effects. When comparing the effectiveness in reducing RMMA between an occlusal splint and a MAD, the latter showed a significantly higher rate of reduction in RMMA episodes than occlusal splints (13).

Additionally, the MAD functions similarly to an occlusal splint by allowing precise occlusal contact of the upper and lower splint, eliminating occlusal interferences, modifying the position of the condyle, reducing loading pressure on the retrodiscal tissue, elongating the muscle, and reducing sensory feedback on periodontal receptors (44). A good design and management of the MAD favors good posterior support and balanced occlusal forces between the upper and lower splints, while the patient is in a resting position (45-48). MADs promote a forward and stable mandibular position, which can help reduce muscle tension and prevent clenching and grinding (48). Therefore, the use of MAD helps reduce SB events in those cases where the events are associated with breathing or OSA-related awakening, while in other SB events, the MAD protects the teeth. like an occlusal splint.

DESCRIPTION OF A CLINICAL CASE

With the presentation of this clinical case, we want to illustrate what is described in the present scoping review of the literature of this article. This was a 46-year-old male patient who came to the clinic with obvious signs of bruxism, missing molars due to fracture, and wear facets on several incisors. The patient was asked about snoring, observed apneas, and drowsiness during the day, answering positively to all three: snoring of more than 5 years of evolution that occurred daily and of a strong nature, observed by the patient's partner, and moderate drowsiness during the day (Epworth 12). An intraoral assessment was performed, and it was classified on the Friedman scale of the palate and tongue as grade 3, and the Friedman scale of the palate tonsils as grade 1.

Given the suspicion of moderate OSA, home respiratory polygraphy (PG) was performed with a result of AHI 57, that is, very severe OSA. Complete rehabilitation was carried out and, in order to protect the teeth and the dental prosthesis from bruxism, a MAD was placed. After signs and symptoms of OSA were detected, the patient was referred to a sleep specialist who confirmed the diagnosis and prescribed MAD for treatment. Once the MAD was in place, a control PG was performed, which gave an AHI of 7.3, that is, resolution of the OSA problem. Currently, the patient is asymptomatic and undergoes control of the OSA with the doctor, as well as control of the MAD, his teeth, and rehabilitation with the dentists.

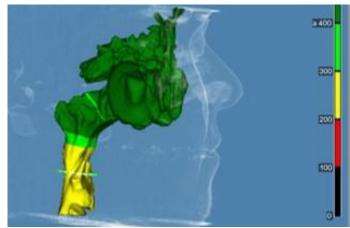


FIGURE 1 Upper airway image: color scale illustrates area in mm²

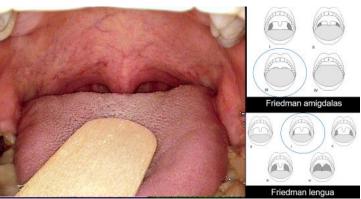


FIGURE 2 Friedman classification of the patient at the beginning of treatment



FIGURE 3

Clinical case. Top left, profile photo; in the center, teleradiography in which the narrow airway can be seen in an anteroposterior direction; top right, panoramic x-ray showing the absence of some molars. Bottom left and center, intraoral diagnostic photo, with wear and gingival retraction; bottom right, photo with MAD after rehabilitation



FIGURE 4

Diagnostic polygraphy and control polygraphy. Left, the AHI diagnostic polygraph of 55.9. Right, polygraphy with MAD AHI 7.3

CONCLUSIONS AND RECOMMENDATIONS

Bruxism and apnea are two independent entities correlated according to current scientific evidence. Dentists should be aware of the important role they play in the detection and appropriate treatment of SB and OSA in all patients who visit the dental office.

The diagnosis of OSA and the prescription of a MAD for the treatment of OSA corresponds to the sleep doctor and the placement and management of the MAD is the responsibility of the dentist with adequate training in sleep. It is important that the work protocol be multidisciplinary, both for diagnosis and for short, medium, and long-term follow-up.

The dental consequences of SB are important, and its prevention and management constitute a point of special interest for dentists who want to offer the best treatment to their patients with stable solutions in the short, medium, and long term, both in oral and dental health. durability of them. Assessing breathing, sleep, and occlusion as well as the orofacial joint and musculature is essential.

Scientific evidence highlights the importance of prescribing a MAD from a dental point of view to protect the teeth in patients with bruxism and OSA. Likewise, dentists can inform the patient about sleep hygiene techniques that, together with the device, will help reduce the problem.

In the case of starting treatment with MAD due to bruxism, the patient must continue with the complete diagnosis of OSA by a sleep doctor, with adequate prescription and control of the treatment.

More clinical studies are needed to show the positive relationship of long-term MAD in reducing the signs, symptoms, and consequences of SB.

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