International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2015): 78.96 | Impact Factor (2015): 6.391

Medical Management of Bruxism

Karpaga Preethitha .S

Abstract: ***Bruxism is an oral movement disorder characterized by grinding or clenching of teeth. This disorder can occur during sleep as well as during wakefulness and has estimated prevalence in the general adult population of approximately 8-10%. (1).This condition isn't a disease but when exacerbated may cause an unbalance and changing of orofacial structures. Thus, it is necessary to obtain effective and safe treatments for the control and management of the bruxist patient. (2). Hence this article gives information about the various medical modalities available for the management of bruxism.***

Keywords: movement disorder, clenching teeth, changing orofacial structures

1. Introduction

Sleep Bruxism (SB) is considered a movement disorder related to sleep. This parafunction is characterized by non-functional teeth contact, which can occur in a conscious or unconscious way, manifested by grinding or clenching of teeth(2)(3). Due to its prevalence and injuries caused tothe patients, the correct diagnosis shows greatvalue to the development of appropriate treatment protocols, which include therapeutics using devices and oral therapies, pharmacologicalmeasures and cognitive-behavioral treatments (CBT).

2. Dental Approaches

The dental approaches for this treatment can be divided into true occlusal interventions and occlusal appliances. True occlusal interventions:- This category, that includes approaches like occlusal equilibration, occlusal rehabilitation and orthodontic treatment that is aimed at 'achieving harmonious relationships between occluding surfaces', still gives rise to a great deal of controversies among dental clinicians and researchers. A number of case reports have been published with the objective of presenting convincing descriptions of the efficacy of occlusal interventions in the management of bruxism, by means of either occlusal equilibration (4)(5)(6) expressed his awareness of the lack of science in this domain full of controversies and suggested that occlusal adjustment is indicated only as part of a periodontal treatment plan when trauma from occlusion is present – a suggestion that was generally followed by periodontists, especially in combination with the use of occlusal splints (7). In that study, the effectiveness of an orthodontic technique (viz. buccal separators) in relieving bruxism activity was evaluated, and no differences between the active treatment and control conditions were observed. But the developer of the buccal separator technique failed to provide new, convincing evidence in favor of his technique (8).

In short, there is no support in the literature for the use of 'true' occlusal interventions like equilibration, management of bruxism. In view of the current insights into the aetiology of bruxism, viz. that the disorder is mainlyregulated centrally – not peripherally (9) (10), future research on this category of management strategies for bruxism seems redundant.(11)

Occlusal appliances**:** -The splints have different names [e.g. occlusal bite guard (modified), bruxism appliance, bite plate, night guard (retainer), occlusal device] and slightly different appearances and properties, but in essence most of them are hard acrylic-resin stabilization appliances, mostly worn in the upper jaw (12–22).

Although the concept of soft splints is appealing, hard splints are generally preferred over soft splints for practical reasons (e.g. soft splints are more difficult to adjust than hard ones), to prevent inadvertent tooth movements, and because hard splints are suggested to be more effective in reducing bruxism activity than soft splints (23).

Four prescriptions describe splints that do not require a contribution of a dental laboratory. The first one describes an 'in-office' procedure for the manufacture of a regular hard acrylic-resin bruxism device that reduces the delay in starting a bruxism treatmentbecause no dental laboratory is involved.(24)

A similar prescription describes the chair-side manufacture of a composite splint (25).

The third prescription describes the chair-side adjustment of the so-called 'Nociceptive Trigeminal Inhibition (NTI) Clenching Suppression System' – a small anterior splint that is supposed to be effective, amongst others, in the management of bruxism (26)

Finally, the fourth prescription describes the scientifically unsupported concept of the pre-fabricated and chair-side adjustable 'Bruxism 'S' Splint' that can be used incombination with active orthodontic treatment (27).

Several studies assessed the efficacy of occlusal splints in groups of bruxism patients, using a comparative, single-cohort 'pre-treatment – post-treatment' design and ⁄ or a case–control design. Although these are not the strongest designs to assess the efficacy of treatment modalities (for that purpose, RCT with long-term evaluations are required), such studies are frequentlyperformed and the conclusions of these studies are stronger – and thus more valuable – than those of case reports. (10)

Behavioral Treatment

Behavioral treatment includes sleep hygiene measures, biofeedback, relaxation techniques, stress control techniques, and hypnotherapy. (28)

Sleep hygiene

Sleep hygiene is a set of instructions which aims to correct Personal habits and environmental factors that interfere with sleep
Qualityy (29). These instructions are as follows:

1) Avoid consumption of coffee, tea, stimulants, chocolate, and medications containing caffeine.
2) Avoid drinking alcohol at least six hours before bedtime.
3) Avoid smoking for at least six hours before bedtime.
4) Avoid eating heavy meals before bedtime.

Biofeedback:
Biofeedback is a relaxation technique with concomitant monitoring of certain physiological variables, such as electromyography, skin temperature, cardiac frequency, blood pressure, and electrodermal activity. More specifically, the patient observes and controls the physiological functions that are monitored by equipment. The positive effects of the treatment are generally only observed during the treatment period (30)

Relaxation techniques:

This technique include (29)

1) Specific methods for relaxing mandibular muscles, such as relaxing the jaw with the lips closed and separated several times a day
2) Voluntary clenching and subsequent unclenching of the teeth for five seconds each; this exercise should be repeated for a total of five times per series with six series per day over a two-week period.

Anxiety behavioral treatment: Personality profile, stress and anxiety are major factors in SB. Nevertheless, no controlled studies determining the effectiveness of anxiety behavioral treatment for clinical SB symptoms have been developed. Cognitive behavioral psychotherapy, cognitive relaxation techniques for managing stress and anxiety coupled with changes in lifestyle have been suggested. (31) (32)

Hypnotherapy:
Hypnotherapy or self-hypnosis is a specific relaxation technique. A study conducted by Clarke et al. with 8 SB carriers demonstrated that hypnotherapy produced objective and subjective improvements in SB symptoms during a 36-month follow-up period. (33)

Pharmacological treatment:
Zolpiden: Zolpiden (5-10 mg per dose) is a non-benzodiazepine nonanxiolytic agonist hypnotic of the imidazopyridine class that selectively binds to the omega-1 GABA-benzodiazepine receptor complex.Zolpiden is also marketed in a second format, with two release phases: One quick phase (30 minutes) for sleep induction and one slower phase with a half-life of approximately 6 hours. According to hypodopaminergic evidence in SB genesis, the positive effects of zolpiden on diseases that involve dopaminergic alterations may indicate similar improvement in sleep bruxism. In addition, zolpiden would also likely act positively upon arousals that occur before abnormal masticatory muscle activity.(29)

Antidepressants: An antidepressant with an agonist dopaminergic profile, such as amineptine or bupropion, may be useful for the treatment of SB, pursuant to the hypothesis that dopaminergic deregulation is important in SB.(34)(35)An antidepressant with an 5HT2A postsynaptic receptor blocker antagonist profile, such as mirtazapine (7.5-30 mg per dose), trazodone (50-100 mg per dose), ritanserine (5-10 mg per dose), or agomelatine (25-50 mg per dose), increases the amount of slow wave sleep in both normal volunteers and individuals suffering from depression.(36)(37)(38)

Buspirone: Buspirone is a partial agonist serotonergic agent used for the treatment of generalized anxiety. Buspirone shows a pharmacological profile, with activity on 5-HT1 postsynaptic receptors and 5-HT2A receptor antagonist activity. A report of four bruxism cases indicates that patients administered sertraline felt subjective relief of SB after introduction of a 10 mg buspirone night dose.(39)(29)

Anticonvulsant agents: Some anticonvulsant agents (ACA), such as gabapentine, topiramate, tiagabine, and pregabaline, promote sleep stability in epileptic subjects. In addition to this effect, such agents promote a reduction in arousals and increases in delta and REM sleep in healthy individuals, epileptic subjects, and patients suffering from insomnia.(40)

Clonazepan:Clonazepan is a benzodiazepine ansiolytic with an extended half-life. It is used for treating anxiety disorders, epileptic syndromes and involuntary movement disorders (tremors). It is also used as a muscle relaxer and as a hypnotic agent for treating acute insomnia or comorbid chronic insomnia. (29)

Dopaminergic agents: The association of restless leg syndrome and other dopaminergic disorders with SB suggests a therapeutic role of dopaminergic agents in SB. (41) The clinical double-blind study conducted by Lobbezoo et al. in 1997 with low L-dopamine doses and benserazide administered to primary SB carriers demonstrated only one attenuating effect upon 30% of bruxism symptoms. (42)

Adrenergic agents: In a randomized, placebo-controlled cross-over study coupled with active treatment (propranol 120 mg and clonidine 0.30 mg) conducted in 2006, it was demonstrated that clonidine, not propranol, was clinically effective for treatment SB symptoms. Clonidine significantly (70% positive rate response) reduced abnormal masticatory activity and reduced the frequency of teeth grinding bursts and the sympathetic tonus one minute before each teeth grinding burst. (29)
Botulinum toxin: Botulinum toxin type A (TXB-A) is an effective treatment for certain neurological disorders. (43).The clinical effect of TXB-A upon secondary bruxism can be observed within 2 to 4 days after injection. Its beneficial effects last for approximately 4 months.

Muscle relaxers: Baclofen, a GABA type B agonist (10-60 mg per dose), can be utilized for the treatment of SB symptoms; however, no controlled studies of its efficacy for the treatment of SB have been reported. (44) Other medical treatments:It is important to treat gastroesophageal reflux in SB carriers, since the presence of gastroesophageal reflux

and/ xerostomy are risk factors for development or progression of SB(45)

References

[1] Lavigne GJ, Manzini C, Kato T. Sleep bruxism. In: KrygerM,Roth T, Dement WC, eds. Principles and Practice of Sleep Medicine. Philadelphia, PA: Elsevier Saunders, 2005:946–959
[2] Sleep bruxism: Therapeutic possibilities based in evidences Eduardo Machado, Patricia Machado, Paulo AfonsoCunali, Cibele Dal Fabbro
[3] American Academy of Sleep Medicine International Classification of Sleep Disorders. 2nd ed. Westchester: American Academy of SleepMedicine; 2005
[4] Leon SP. The source of the problem. Dent Today. 2003;22:12.) or occlusal rehabilitation with composite resin materials
[5] Ford RT, Douglas W. The use of composite resin for creating anterior guidance during occlusal therapy. Quintessence Int.1988; 19:331–337). In 1973, however, Stephens
[6] Stephens RG. Occlusal adjustment in periodontal therapy. J Can Dent Assoc (Tor). 1973;39:332–337
[7] Lester M, Baer PN. Survey of current therapy: bruxism splints. Periodontal Case Rep. 1989; 11:23–24
[8] Mintz AH. Acute TMJ versus chronic TMJ. Angle Orthod.1993; 63:4–5
[9] Lobbezoo F, van der Zaag J, Naeije M. Bruxism: its multiple causes and its effects on dental implants. An updated review Oral Rehabil. 2006; 33:293–300
[10] Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil. 2001; 28:1085–1091.
[11] Principles for the management of bruxism F. LOBBEZOO, J. VAN DER ZAAG, M. K. A. VAN SELMS, H. L. HAMBURGER&M. NAEIJEDepartment of Oral Function, Academic Centre for Dentistry Amsterdam (ACTA) and Departments of Neurology and Clinical Neurophysiology, and Amsterdam Center for Sleep-Wake Disorders, SlotervaartGeneral Hospital, Amsterdam, The Netherlands
[12] Allen DL. Accurate occlusal bite guards. Periodontics.1967;5:93–95.
[13] Courant P. Use of removable acrylic splints in general practice. J Can Dent Assoc (Tor). 1967;33:494–501.
[14] Greenwald AS. The bruxism appliance and its varied application: outline of procedure. N Y J Dent. 1968;38:443
[15] Askinas SW. Fabrication of an occlusal splint. J Prosthet Dent. 1972;28:549–551.
[16] Glazebrook P. An equilibrated bite plate. Probe (Lond). 1974;16:99–100
[17] Gabriele P. A double night guard retainer. N Y State Dent J. 1986;52:30–31.
[18] Ordene NM. A modified bruxism appliance. N Y State Dent J. 1989;55:40–41.
[19]. Perel ML. Parafunctional habits, nightguards, and root form implants. Implant Dent. 1994;3:261–263.
[20] Davis CR. Maintaining immediate posterior disclusion on an occlusal splint for patient with severe bruxism habit. J Prosthet Dent. 1996;75:338–339
[21] Nassif NJ, al-Ghamdi KS. Managing bruxism and temporomandibular disorders using a centric relation occlusal device. CompendContinEduc Dent. 1999;20:1071–1074, 1076,1078, 108
[22] Cowie RR. The clinical use of night guards: occlusal objectives. Dent Today. 2004;23:112, 114–115.
[23] Okeson JP. The effects of hard and soft occlusal splints on nocturnal bruxism. J Am Dent Assoc. 1987; 114:788–791
[24] Austin D, Attanasio R. A procedure for making a bruxism device in the office. J Prosthet Dent. 1991;66:266–269
[25] Leib AM. The occlusal bite splint – a noninvasive therapy for occlusal habits and temporomandibular disorders. CompendContinEduc Dent. 1996; 17:1081–1084, 1086, 1088
[26] Boyd JP. Improving TMDtreatment and protecting restorative dentistry. Dent Today. 1998; 17:144
[27] Sullivan TC. A new occlusal splint for treating bruxism and TMD during orthodontic therapy. J ClinOrthod. 2001; 35:142–144
[28] Sleep Bruxism TreatmentFlávioAlóeCentro de DistúrbiosVigília-Sono, Instituto de Psiquiatria, Universidade de São Paulo. São Paulo, SP.CorrespondenceFlávioAlóeRuaJoaquimFloriano, 871 - conjunto 43 04534-013 - São Paulo | SP, Brazil E-mail: piero.ops@terra.com.brReceived May 29, 2008; accepted December 26, 2008
[29] Aló e F, Tavares S. Transtornos do Sono. In: Lopes AC, Neto VA (Eds). Tratado de ClínicaMédica. Volume II. São Paulo: EditoraRocca, 2006. p. 2524-53
[30] Foster PS. Use of the Calmset 3 biofeedback/relaxation system in the assessment and treatment of chronic nocturnal bruxism. Appl Psychophysiol Biofeedback 2004;29:141-7.
[31] Bader G, Lavigne GJ. Sleep bruxism; an overview of an oromandibularsleep movement disorder. Sleep Medicine Reviews 2000; 4:27-43
[32] Pierce CJ, Gale EN. A comparison of different treatments for nocturnal bruxism. J Dent Res 1988; 67:597-601
[33] Clarke JH, Reynolds PJ. Suggestive hypnotherapy for nocturnal bruxism: A pilot study. Am J Clin Hypnosis 1999; 33:248-53
[34] Moreno RA, Moreno DH, Soares MBM. Psicofarmacologia de antidepressivos. Rev Bras Psiquiatria 1999;21:24-40.
[35] Wilson S, Argyropoulos S. Antidepressants and sleep. A qualitative review of the literature. Drugs 2006;66:927-47
[36] Moreno RA, Moreno DH, Soares MBM. Psicofarmacologia de antidepressivos. Rev Bras Psiquiatria 1999;21:24-40.
[37] Wilson S, Argyropoulos S. Antidepressants and sleep. A qualitative review of the literature. Drugs 2006;66:927-47.
[38] Aslan S, Isik E, Cosar B. The effects of mirtazapine on sleep: A placebo controlled, double-blind study in healthy young volunteers. Sleep 2002;25:666-8
[39] Bostwick JM, Jaffe MS. Buspirone as an Antidote to SSRI-Induced Bruxism in 4 Cases. J Clin Psychiatry 1990;60:857-60.

[40] Sammaritano M, Sherwin A. Effect of anticonvulsants on sleep. Neurology 2000;54:16-24
[41] Lavigne GJ, Montplaisir J. Restless legs syndrome and sleep bruxism:prevalence and association among Canadians. Sleep 1994;17:739-43
[42] Oksenberg A, Arons E. Sleep bruxism related to obstructive sleep apnea: the effect of continuous positive airway pressure. Sleep Med 2003;3:513-5.
[43] Berardelli A, Mercuri B, Priori A. Botulinum toxin for facial-oramandibular spasms and bruxism. In: Jankovic J, Hallett M, ed.Neurological disease and therapy, therapy with Botulinum toxin. New York: Marcel Dekker; 1994. p. 361-7
[44] Alóe F, Gonçalves LG, Azevedo A, Barbosa RC. Bruxismodurante o sono.Rev Neurociências 2003;11:4-17.
[45] Miyawaki S, Tanimoto Y, Araki Y, Akira KA, Akihito FA, Teruko TY.Association between nocturnal bruxism and gastroesophagealreflux.Sleep 2003;26:888-992