

Extended Case Report. Bruxism with a History of Early Onset in a 25-Year-Old Male

Veronica Mercuț¹, Monica Scricciu², Sanda Mihaela Popescu³, Monica Crăitoiu⁴, Petre Mărășescu⁵, Roxana Marinescu⁵

¹ Ph.D. Professor of Prosthetic Dentistry, Faculty of Dental Medicine, University of Medicine and Pharmacy, Craiova, Romania.

² Ph.D. Associate Professor of Prosthetic Dentistry, Faculty of Dental Medicine, University of Medicine and Pharmacy, Craiova, Romania.

³ Ph.D. Associate Professor of Oral Rehabilitation, Faculty of Dental Medicine, University of Medicine and Pharmacy, Craiova, Romania.

⁴ Assistant Professor of Prosthetic Dentistry, Faculty of Dental Medicine, University of Medicine and Pharmacy, Craiova, Romania.

⁵ Ph.D.-Student. Department of Prosthetic Dentistry, Faculty of Dental Medicine, University of Medicine and Pharmacy, Craiova, Romania.

Abstract

This paper reports the case of a 25-year-old male patient with bruxism who has been monitored for more than 20 years. Once his permanent teeth had erupted, his bruxism led to advanced wear at the palatal aspect of the maxillary incisors. The patient is physically normally developed but has muscle hypertrophy of the scapulohumeral belt, without having practised any physical exercises for developing the muscles in this area. Psychologically, he is well balanced, very meticulous, conscientious, intelligent, and a cooperative patient. He reported that he was a great consumer of caffeine-based soda drinks (about 2l per day). He gave a history of grinding his teeth from a very early age while sleeping and stated that he used to wake up during these teeth-grinding episodes, with his jaws clenched. On the basis of the history, a provisional diagnosis of bruxism was made. Since he was 14 years old, his bruxism has been monitored by one of the authors of this case report and the minimal criteria for diagnosis set by American Sleep Disorder Association and revised by American Academy of Sleep Medicine have been applied. The patient presented three conclusive signs, included in these minimal criteria: tooth wear, teeth grinding, and jaw clenching. The major factor that triggered his bruxism could not be identified. He has worn a night guard, had topical fluoride applications, and has been advised to reduce his intake of caffeine and carbonated drinks and try to minimise stressful factors in his life.

Key Words: Bruxism, Early Onset

Introduction

Bruxism is currently considered the most serious occlusal parafunction. It is manifested by the hyperactivity of the masticatory muscles and affects the teeth, the periodontium and the temporomandibular joint. Clark & Ram (2007) considered bruxism a particular form of dystonia, a psychophysiological disorder, and a common condition of the masticatory system [1]. It can cause tooth wear and disorders of the masticatory muscles, jaw bones, and temporomandibular joint.

The earliest mention of bruxism dates back over 2,400 years to the ancient Greeks. Rozenzweig (1994) emphasised that “tooth wear was an expression of disorder of the soul” [2]. This aphorism indicates the complex nature of bruxism, whose

aetiology might be placed, to some extent, outside the orodental area. This is not a new concept, as Marie and Pietkiewicz (1907) suggested that tooth wear was associated with some injuries of the central nervous system and proposed the term *bruxomania* [3]. The first studies into the aetiology of bruxism took into account the disorders of occlusion and the function of the masticatory muscles.

More recently, Lavigne and Monplaisir (1995) concluded that the aetiology of bruxism should take behaviour and sleep disorder into account [4]. Brocard *et al.* (2007) reported that the American Academy of Psychiatry considered bruxism *as assimilated to a behaviour disorder* [5]. The American Academy of Sleep Medicine [6] stated that the nocturnal bruxism is a sleep disorder, and

Corresponding author: Veronica Mercuț, Faculty of Dental Medicine, University of Medicine and Pharmacy, 2-4 Petru Rares Street, Craiova, Romania; e-mail: veronica.mercut@yahoo.com

should be considered in the parasomnias category. Within this category, bruxism is the third most common condition, after snoring and talking during sleep. According to data from clinical studies, bruxism never occurs in isolation. It can be associated with the obstructive sleep apnoea syndrome, micro-awakening periods while sleeping, and excessive sleepiness during the day.

Kato *et al.* (2003) proposed the following definition: “sleep bruxism is a parasomnia and an oral parafunctional activity characterized on one side by the jaws straining (tonic activity) and/or by a stage, repetitive activity of the masticatory muscles which is translated in terms of teeth grinding” [7].

As for its prevalence, many authors [8-11] have reported that 6% to 20% of adults have reported a few episodes of bruxism. Studies by Reding *et al.* (1966) [12] and Lavigne and Monplaisir (1994) [13] indicated that 6% of the patients studied had tonic activity of their masticatory muscles and 20% ground their teeth. The percentage of children bruxing was also reported to be high (15% prevalence between 15 and 17 years of age) [12]. However, another survey found a tendency for the frequency of bruxism to decline as children grew up [4].

Case study

This case study reports details of a 25-year-old male patient who has suffered from bruxism for virtually all his life. The patient gave his written informed consent for his details and images to be included in this report.

He gave a history that his mother was also a bruxist and drank excessive amounts of coffee during her pregnancy, prior to his birth. His mother mentioned that from the time of his birth, he had a muscle hypertonia and could keep his head up without support from her. Before he was four months old, he could sit up without being supported. When he was ten months old, at the same time as the eruption of two upper incisors, he made bruxist-like noises for two weeks. At five years old, he suffered a cranial trauma, with fractures to his occipital and temporal bones, but without any damage to his teeth or jaws. There was wear on the occlusal surfaces of all his deciduous teeth, which his dentists at that time considered to be a sign of functional occlusal remodelling. He did not have any dental caries or other oral disease.

The patient has been carefully monitored by one of the authors of this report since he was 14 years and 8 months old. At this age, he suffered

from painful dentine sensitivity to his upper incisors. A transparent area developed at the incisal tips of his upper central incisors, although, at this age, there was no wear at the recently erupted canines and premolars. However, there was advanced wear at the palatal aspects of his maxillary incisors. All of the enamel had worn away from the palatal surface of his upper incisors and the dentine was so thin that the red colour of the dental pulp could be seen. It seems likely that this was due to bruxism. When asked whether he ground his teeth while sleeping, the patient answered positively and added that he used to wake up during the teeth-grinding episodes to find his jaws clenched. He reported that he had a very high intake of caffeine-based soda drinks; on average, 2l per day.

A diagnosis of his bruxism was made on the basis of the history and the clinical examination. It seems very likely that the aetiology of this patient's tooth surface loss is related to his bruxism and daily intake of carbonated drinks. The patient had denied other causes for tooth wear, such as acid regurgitation, or vomiting.

The nature of the problem was explained to the patient and his parents and he was recommended to stop drinking carbonated drinks and caffeine-containing drinks. He was referred to a stress counsellor (psychologist) for the management of stress and his high intake of caffeine and carbonated drinks.

At this time, treatment included fluoride topical applications and a maxillary night guard made of hard acrylic was prescribed. The night guard has subsequently been changed once a year. For the hypersensitivity caused by tooth wear, a fluoride solution (natrium [sodium] fluoride) was prescribed, applied once a week for a month and repeated when necessary. The vitality of the upper incisors was also checked on a monthly basis (using an electric pulp tester). The initial scores were high but they decreased to normal values after a year. The vitality of the affected teeth has been preserved by the deposition of a layer of secondary dentine over the pulp chamber. The good cooperation of the patient and the fluoride solution appeared to be key factors in this improvement.

Over the next ten years, the patient experienced “calm periods” alternating with periods of stress when bruxism started again. As he grew older, as mentioned previously, replacement night guard appliances were made as his occlusion developed. When aged 24 years old, he tried to use a bruxism deconditioning system based on negative

feedback. However, he found it to be ineffective.

At present, the patient is 25 years old and attends courses at the Faculty of Medicine, preparing for his residency examination. He has noticed that his bruxism has started again. It is possible that it is associated with the stress of his forthcoming examinations. Clinically, he has muscle hypertrophy of the scapulohumeral belt, without practising any physical exercises to develop the muscles in this area. Other than this finding, he has no other relevant medical history. His masticatory muscles and temporomandibular joint were examined and his masseter and temporal muscles showed hypertonia (*Figure 1*). It was also noticed that he had pale lips. This may well have been because of hypertonia of his lip muscles (*Figure 1*). The examination of his temporomandibular joint revealed clicking during the movement of opening and closing his mouth. There was also an asymmetric movement of the two condyles when he opened and closed his mouth.



Figure 1. Patient's face with masseter and temporal muscle hypertonia.

An intra-oral examination revealed a complete dentition with a healthy oral mucosa and periodontium. He had well-aligned teeth, with no caries, restorations or other lesions except for tooth wear (*Figure 2*). There is moderate wear at the canines, premolars and molars and advanced wear of the palatal aspects of his maxillary incisors. The patient currently has generalised tooth wear of the palatal surfaces of his maxillary teeth (*Figure 3*) and of the occlusal surfaces of his lower teeth (*Figure 4*).

However, there has been no loss of enamel or dentine interdentally and the width of the teeth has not been reduced (*Figure 5*).



Figure 2. Casts of the patient's teeth at 25 years of age.



Figure 3. Maxillary teeth with palatal wear at 25 years of age.



Figure 4. Mandibular teeth with occlusal wear at 25 years of age.



Figure 5. Panoramic radiograph of patient at 25 years of age.

The patient met the minimal criteria for the diagnosis of bruxism set by American Sleep Disorder Association and revised by American Academy of Sleep Medicine [6], in that he presented with the three conclusive signs for this diagnosis: tooth wear, tooth grinding, and jaw clenching. Other diagnoses such as gastro-oesophageal reflux, tooth enamel defects, and other parasomnias were ruled out.

The patient currently wears a new maxillary night guard made of hard acrylic. It has been explained to him that nocturnal bruxism is now considered a sleep disorder. The patient reported that he associated his tooth grinding with the moments when he had nightmares. The grinding wakes him up and then he does not know whether the dream events are real. The patient continues to be monitored at monthly intervals.

Discussion

This case has presented a young patient suffering from bruxism, in which almost all the aetiological factors reported in the specialty literature appear to be involved in the triggering the tooth grinding:

inheritance of bruxism, muscle hypertonia, cranial trauma, occlusal trauma, jaw-clenching, intake of caffeine-based soda drinks, stress. The recently published literature suggests a consensus that the aetiology of bruxism is multifactorial [14]. Attanasio (1991) [15] and Lobbezoo *et al.* (2006), [16] cited by Nascimento *et al.* (2008) [17], consider that the aetiology of bruxism involves local, systemic, psychological and hereditary factors. As for the local occlusal aetiology of bruxism, there have been different opinions over the course of time. Ramfjord and Ash (1966) [18] considered that occlusal factors, especially occlusal interferences, played a major role. Rugh *et al.* (1984) [19] created experimental occlusal interferences and found that the role of a lack of occlusal harmony was secondary because when the occlusal interferences were corrected, bruxism did not disappear. The same situation occurs in patients with bruxism who are made edentulous because, following their prosthetic rehabilitation, bruxism may occur again [20,21].

Some authors have associated the pathology of muscle contraction with bruxism. Wolf (1959) [22], cited by Popa (2006) [23], suggested that the muscle forces exerted during bruxism exceed in intensity the forces that are exerted during the mastication. Other authors have considered that the increase in the tonus of the masticatory muscles during bruxism is associated with the change in the position of the mandible and the reduction of the physiologic vertical dimension [24]. We consider the muscle hypertonia of this patient to be a predisposing factor for his bruxism. The hypertrophy of the masticatory muscles is rather a consequence and not a cause of bruxism.

An important development in understanding bruxism was the appreciation that stress played a major role. Rugh and Solberg (1976) [25] were the first to notice that as stress increased, bruxism also increased in intensity. A number of other workers have subsequently investigated the role of cognitive-behavioural factors, such as stress, anxiety, and personality, in the aetiology of bruxism [26-29]. They have concluded that patients diagnosed with bruxism have an anxious personality and a considerable drive to reach their personal goals, when compared with the rest of the population. These features are seen in the patient presented in this case report. Lavigne *et al.* (2003) [30] suggested that nocturnal bruxism is related to anxiety, and is a secondary aspect of excitation when sleeping,

Sleep excitation is defined as a transitory growth in the electrical activity of the brain and heart. These symptoms are followed by tooth grinding.

In 2005, the American Academy of Sleep Medicine proposed an International Classification of Sleep Disorders and defined nocturnal bruxism as a disorder of the stereotypical movements while sleeping, characterised by teeth grinding and/or their clenching [31]. According to this classification, nocturnal bruxism is a sleep disorder, included in the parasomnias category.

The hypothesis of a genetic component should be considered for the patient whose case is reported in this paper, because his mother has presented with bruxism. Hublin and Kaprio (2003) [32] reported a similar pattern of bruxism in patients belonging to the same family (parents, children or brothers). Their hypothesis that bruxism could be associated with a familial predisposition has been supported by studies performed on twins.

For the patient presented in this case study, bruxism was correlated with daily consumption of soda drinks containing caffeine. Winocur *et al.* (2003) [33] suggested a connection between the consumption of alcohol, tobacco, some medication and bruxism.

It has also been suggested that bruxism might be associated with some psychiatric or neurological diseases, with cerebral trauma as a triggering cause [34]. The same triggers have been suggested to play a role in temporomandibular joint syndrome [35]. In this context it may be pertinent to note that although our patient suffered major cranial trauma in childhood, it could not be related to his bruxism, because the trauma occurred after his bruxism began.

One of the authors has managed the patient for over 12 years, during which time he has worn several night guards, received topical fluoride applications, been given psychological counselling for stress management and encouraged to stop consuming caffeine and carbonated drinks. This regimen has enabled him to stop bruxing for some periods and the night guards have protected his teeth. However, as reported earlier, when he becomes

stressed he bruxes.

In summary, bruxism is the most serious form of parafunctional activity of the masticatory system. Its main consequence is tooth surface loss (tooth wear). Several factors have been claimed to contribute to its aetiology including local factors (dental occlusion), systemic factors (stress, anxiety, sleep disorders, some medication/drug consumption), traumatic and hereditary factors, many of which could be associated with the current patient's bruxism.

Conclusions

No one factor could be identified as the major trigger of the current patient's bruxism. His treatment has therefore involved the protection of the occlusal surfaces of his teeth with a night guard, fluoride applications, a recommendation to reduce or eliminate his caffeine and carbonated drink consumption, stress counselling, and monitoring.

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Contributions of each author

- VM is main author, and was responsible for clinical treatment of the patient, literature review, and writing the paper.
- MS is co-author and was responsible for the literature review and writing the paper.
- SMP is co-author and was responsible for the literature review, writing the paper.
- MC is co-author and was responsible for the translation into English.
- PM is co-author and was responsible for the translation into English.
- RM is co-author is co-author and was responsible for the photographs

Statement of conflict of interests

As far as the authors are aware, there are no conflicts of interest.

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