INTERACTION BETWEEN CARIOUS LESIONS AND BRUXISM: COMMON PATHOGENETIC MECHANISMS AND CLINICAL IMPLICATIONS – A NARRATIVE REVIEW

Antonia-Theodora Vrabie[1], Yllka Decolli[2]*, Georgiana-Andreea Frumuzache[1], Alice-Tedora Rotaru-Costin[2], Tinela Panaite[3]

[1]Department of Odontology-Periodontology and Fixed Prosthodontics, Faculty of Dental Medicine, Grigore T. Popa University of Medicine and Pharmacy Iasi, 16 Universitatii Street, 700115 Iasi, Romania; PhD student
[2]Department of Odontology-Periodontology and Fixed Prosthodontics, Faculty of Dental Medicine, Grigore T. Popa University of Medicine and Pharmacy Iasi, 16 Universitatii Street, 700115 Iasi, Romania
[3]Department of Surgery, Faculty of Dental Medicine, Grigore T. Popa University of Medicine and Pharmacy Iasi, 16 Universitatii Street, 700115 Iasi, Romania

*Corresponding author: Yllka Decolli e-mail: yllka.decolli@umfiasi.ro

ABSTRACT

Background: Dental caries and bruxism are common conditions of the stomatognathic system, with different etiopathogenic mechanisms, but with the potential to interact at the level of dental structures. **Objectives:** The aim of this narrative review is to analyze current data from the literature regarding the interaction between carious lesions and bruxism, highlighting the involved mechanisms and clinical implications. **Materials and Methods:** A narrative literature analysis was performed by consulting the PubMed, Scopus, and Web of Science databases, using relevant keywords. Studies published in the last 10 years were included, such as experimental studies, clinical studies, and review articles. **Results:** The data suggest a synergistic effect between dental demineralization and parafunctional mechanical loads. Caries-affected tissues become more susceptible to mechanical stress, while bruxism may favor lesion progression and compromise dental restorations. **Conclusions:** The interaction between dental caries and bruxism requires an integrated clinical approach. Further studies are needed to clarify the relationship between the two conditions and to optimize therapeutic strategies.

INTRODUCTION

Dental caries represents one of the most common chronic diseases worldwide. Dental caries affects all age groups and has a major impact on both oral health and quality of life. The etiopathogenic mechanism responsible for the development of carious lesions is the result of a dynamic process involving the imbalance between demineralization and remineralization. This imbalance is determined by the activity of the dental biofilm, the consumption of fermentable carbohydrates, the composition of saliva, and host susceptibility. In the absence of therapeutic interventions, carious lesions progress through the loss of hard dental structure and, in advanced stages, lead to pulpal involvement (1–3).

Repetitive muscular activity of the mandible, characterized by grinding or clenching of the teeth and/or by maintaining a tense position of the mandible, is defined as bruxism. Bruxism may occur both during sleep and during wakefulness. From an etiological perspective, bruxism is considered a phenomenon with multifactorial etiology, involving neurobiological mechanisms and psychological factors. From a clinical point of view, bruxism is associated with tooth wear, enamel microcracks, coronal fractures, as well as with the compromise of dental restorations (4–6).

Most often, dental caries and bruxism are analyzed separately, with dental caries considered a predominantly biochemical process, and bruxism a mechanical phenomenon. Thus, the interaction between them remains insufficiently explored in the scientific literature.

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From a pathological and physiopathological perspective, bruxism can affect the integrity of demineralized enamel, facilitating the progression of carious lesions due to excessive occlusal loads. At the same time, the dental structure compromised by caries becomes more vulnerable in the presence of parafunctional forces, increasing the risk of fracture and restorative failure (7,8).

The correlation of these two destructive mechanisms, namely chemical and mechanical, may generate a negative synergistic effect on hard dental tissues, influencing the progression of dental caries and the prognosis of restorative treatments. Additionally, the presence of common risk factors, such as stress or salivary changes, may contribute to this interrelationship (9).

The aim of the present narrative analysis is to synthesize current data from the literature regarding the interaction between carious lesions and bruxism, highlighting the biological and mechanical mechanisms involved, as well as the clinical and therapeutic implications resulting from the coexistence of the two conditions.

MATERIALS AND METHODS

The present study represents a narrative analysis of the scientific literature, aiming to evaluate the existing data regarding the interaction between carious lesions and bruxism, with a focus on pathogenetic mechanisms and clinical implications.

Search Strategy

The identification of relevant studies was carried out by consulting the electronic databases PubMed, Scopus, and Web of Science. The literature search was performed using combinations of keywords such as “dental caries”, “bruxism”, “tooth wear”, “occlusal stress”, “enamel demineralization”, and “dental biomechanics”.

Inclusion and exclusion criteria

Articles published in the last 10 years were included in the analysis to ensure the relevance of the data. Original studies (in vitro and in vivo), clinical studies, observational studies, and review articles relevant to the addressed topic were selected.

Articles published prior to the established period, studies with limited access to the full text, as well as papers that did not present direct relevance to the interaction between dental caries and bruxism were excluded.

LITERATURE REVIEW

The scientific literature frequently addresses both dental caries and bruxism, but most often they are analyzed separately, without addressing a systematic integration of the possible mechanisms of interaction between them.

3.1.1. Dental caries and structural changes of hard tissues

Dental caries is described as a dynamic process, characterized by the progressive loss of minerals from the structure of enamel and dentin. Demineralized enamel shows decreased microhardness and changes in the prismatic structure, thus leading to reduced resistance to mechanical stress (10).

Caries processes progressing in dentin, the alteration of the tubular structure, and the reduction of mineral content lead to a decrease in the biomechanical properties of the tooth. Multiple in vitro studies have shown that the modulus of elasticity and fracture resistance are significantly reduced in demineralized tissues compared to healthy structures (10,11).

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3.1.2. Bruxism and Its Impact on Dental Integrity

Bruxism is associated with excessive, repetitive occlusal loads, which most often exceed normal forces. Studies indicate that bruxism contributes to the occurrence of wear facets, enamel microcracks, coronal fractures, and restorative failure (12–14).

Research on the mechanical fatigue of enamel shows that repeated cyclic loading can lead to the progression of pre-existing microcracks. Additionally, clinical studies have reported an increased incidence of dental fractures and deterioration of restorations in patients with bruxism (5).

3.1.3. Data on the indirect interaction between carious lesions and bruxism

Studies that directly investigate the relationship between dental caries and bruxism are limited, but the literature provides relevant indirect data. Studies conducted on demineralized enamel have demonstrated an increased susceptibility to fracture under mechanical stress. In parallel, studies on tooth wear suggest that the loss of the superficial enamel layer may favor bacterial plaque retention and the exposure of vulnerable structures (15).

Various studies indicate that mechanical deterioration of the dental surface may lead to changes in its topography, influencing the process of bacterial colonization as well as the diffusion of acids into the underlying structure. However, the direct causal relationship between bruxism and the acceleration of carious lesion progression has not yet been fully demonstrated, and the available data are methodologically heterogeneous (15).

Overall, the literature supports the hypothesis of a possible synergistic interaction between the chemical damage caused by caries and the mechanical stress induced by bruxism. Longitudinal clinical studies are needed to clarify the extent and direct clinical significance of this relationship (5,16).

3.2. Mechanism of interaction between carious lesions and bruxism

The interaction between carious lesions and bruxism must be analyzed from the perspective of the overlap between the two types of aggression on the dental structure, namely progressive demineralization of a biochemical nature and repetitive mechanical loading of increased intensity. These two mechanisms, although independent in terms of etiology, may become interdependent in terms of structural consequences (17).

3.2.1. Alteration of the Mechanical Properties of Demineralized Enamel

From the early stages of caries, demineralization affects the superficial layer of enamel, leading to a decrease in microhardness and changes in the arrangement of hydroxyapatite prisms. This modification reduces the ability of enamel to absorb and evenly distribute occlusal forces (10).

In the presence of bruxism, repeated cyclic loading generates mechanical fatigue phenomena. Enamel weakened by mineral loss becomes more prone to the initiation and progression of microcracks, fracture of superficial fragments, leading to structural collapse in demineralized areas (15,18).

Repetitive mechanical stress can accelerate the transformation of an initial non-cavitated carious lesion into a cavitated lesion by compromising the integrity of the superficial layer (19).

3.2.2. Crack propagation and structural amplification effect

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Bruxism is associated with the generation of lateral and axial forces that may exceed normal physiological thresholds. In the presence of demineralized areas, the concentration of stresses at the level of structural defects favors crack propagation (20).

The enamel–dentin interface represents a critical area in stress distribution. When the carious process affects this region, the tooth's ability to absorb mechanical loads decreases significantly. Therefore, microdefects may evolve into macroscopic fractures, especially at the level of posterior cusps (19).

The presence of such a mechanism suggests the existence of a structural amplification effect, in which the initial biomechanical weakening is enhanced by repetitive mechanical stress (19).

3.2.3. Vulnerability of carious dentin in the presence of parafunctional loads

The extension of the carious process to the level of dentin significantly alters the biomechanical behavior of the tooth. Demineralized dentin is associated with a decrease in the modulus of elasticity and compressive strength, which reduces the ability to dissipate mechanical energy (21).

In the presence of bruxism, cusp flexure may occur, along with an increase in internal stresses at the level of cavity walls, as well as the occurrence of incomplete or complete fractures (19,20).

Posterior teeth with significant structural loss are particularly vulnerable. In these situations, parafunctional loads may transform a restoratively treated carious lesion into a structural failure (22).

3.2.4. Modification of dental topography and biofilm retention

Bruxism modifies the dental surface through occlusal wear and microfractures, increasing roughness and forming micro-retentive areas. Irregular surfaces may favor biofilm accumulation and may hinder effective oral hygiene (9,23).

Additionally, the removal of the superficial enamel layer, often more highly mineralized, may expose more porous layers that are more susceptible to acid attack. Thus, mechanical degradation may facilitate the biochemical progression of caries (10).

3.2.5. Implications for restorations and the risk of secondary caries

In the case of restored teeth, the remaining structure is often reduced. In the presence of bruxism, increased occlusal loads may lead to marginal microleakage, poor adaptation of the restoration, fracture of the restorative material, as well as the recurrence of the carious process at the tooth–restoration interface (14).

Repeated mechanical fatigue may compromise adhesive integrity, facilitating bacterial penetration and the development of secondary caries. Thus, the interaction between caries and bruxism may negatively influence the long-term restorative prognosis (24).

3.2.6. Possible indirect mechanisms and common factors

Psychological stress, frequently associated with bruxism, may influence dietary behavior and oral hygiene, increasing the risk of caries. Additionally, reduced salivary flow during sleep limits the buffering capacity of acids, while episodes of bruxism generate additional mechanical loads (25).

Therefore, the relationship between the two conditions may go beyond a simple mechano-structural interaction, with the possibility of a common pathogenic framework that favors their coexistence and the mutual enhancement of their destructive effects (25).

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3.3. Clinical implications

The interaction between carious lesions and bruxism leads to complex clinical consequences, influencing both the progression of dental pathology and the success of restorative treatments. In this context, the therapeutic approach must be integrated, taking into account the overlap of mechanical and biochemical factors (23,26).

3.3.1. Risk assessment and early diagnosis

Patients with bruxism present an increased risk of accelerated deterioration of dental structures, especially when early carious lesions are present. Excessive occlusal forces may promote the collapse of demineralized enamel structure, leading to the rapid transition from non-cavitated lesions to cavitated lesions (13).

In this regard, the early identification of clinical signs of bruxism (wear facets, hypersensitivity, muscle hypertrophy) and their correlation with the presence of carious lesions are essential. Caries risk assessment should also include the analysis of parafunctional factors, not only biochemical ones (13).

3.3.2. Preventive strategies and risk factor control

Preventive management must be adapted for patients who present both caries and bruxism. Preventive measures include the use of remineralizing agents (fluorides, CPP-ACP), dietary control, and the optimization of oral hygiene (27).

In addition, the control of bruxism plays an essential role. Interventions may include stress reduction, behavioral therapies, and the use of occlusal splints. These contribute to reducing mechanical loads on the teeth and, consequently, to reducing the progression of carious lesions (28).

3.3.3. Particularities in restorative treatment

The presence of bruxism significantly alters the prognosis of dental restorations. Teeth affected by caries and simultaneously subjected to parafunctional loads present an increased risk of fracture, marginal microleakage, and restorative failure (24).

In these cases, the choice of restorative material must take into account mechanical resistance and the ability to evenly distribute occlusal forces. Materials with superior adhesive properties and increased wear resistance are preferred. A

Additionally, cavity design should minimize stress concentration (14).

The protection of restorations through nocturnal occlusal splints is frequently recommended, contributing to increased longevity (29).

3.3.4.Management of Complications and Restorative Failures

The interaction between caries and bruxism increases the risk of complications such as dental fractures, decementation of restorations, or the development of secondary caries. Repeated mechanical fatigue may compromise the adhesive interface and facilitate bacterial infiltration (30).

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3.3.5. Importance of oral hygiene and biofilm control

Changes in dental topography induced by bruxism, such as increased roughness and microcracks, favor the

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retention of bacterial biofilm. This aspect may accelerate the progression of carious lesions (23).

Patients should be instructed in adapted oral hygiene techniques, including the use of auxiliary means (dental floss, interdental brushes, mouthwash). Periodic check-ups should be performed more frequently to ensure effective biofilm control (15).

3.3.6. Multidisciplinary approach

Optimal management of patients with caries and bruxism requires a multidisciplinary approach. In addition to dental treatment, interventions from the fields of psychology (stress management), physiotherapy, or sleep medicine may be necessary (28).

This integrated approach contributes to reducing the etiological factors of bruxism and to improving the long-term prognosis (25).

3.3.7. Prognosis and long-term monitoring

Patients with this pathological association require an individualized monitoring plan. The frequency of check-ups should be increased, and the evaluation should include both caries status and signs of tooth wear, as well as the integrity of restorations (12).

By integrating preventive, restorative, and bruxism control measures, dental prognosis can be significantly improved and the risk of long-term complications can be reduced (23).

DISCUSSION

Although dental caries and bruxism are studied as distinct pathologies, there are mechanical and biological arguments that support a complex interaction between them, as also highlighted by the results of this narrative review. Current scientific literature indicates a possible synergism in which tissue demineralization and repeated mechanical overload potentiate each other, leading to the accelerated deterioration of hard dental structures (5,15,17,22).

An important aspect of this interaction is represented by the way in which the biomechanical properties of enamel and dentin are altered in the carious context. Thus, the decrease in microhardness and fracture resistance leads to increased vulnerability to occlusal loading, especially in the presence of parafunctional activities (10,11,19,21). The role of mechanical factors in the progression of dental caries is also suggested by the transformation of initial carious lesions into major structural defects under the influence of parafunctional forces (5,12,22).

On the other hand, the analyzed data show that bruxism is not only an aggravating factor, but may also indirectly contribute to the initiation or acceleration of the carious process. Changes in dental topography, microcracks, and increased surface roughness favor bacterial biofilm retention and contribute positively to cariogenic activity (9,15,16). However, it should be noted that the direct causal relationship between the incidence of dental caries and bruxism has not been fully demonstrated, and the available studies show methodological heterogeneity (15,16).

The literature also highlights the impact of parafunctional activities on restorative treatments. Teeth affected simultaneously by caries and bruxism present a much higher rate of restorative failure, through mechanisms such as marginal microleakage, the occurrence of structural fractures, or fatigue of the materials used. This aspect emphasizes the need to adapt therapeutic strategies and restorative protocols, primarily through the implementation of occlusal protection measures (14,24,30).

Additionally, the existence of common risk factors such as changes in

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salivary flow or psychological stress suggests that the interaction between the two pathological conditions may be part of a broader clinical context, requiring multidisciplinary approaches in patient management (14,29).

LIMITATIONS OF THE ANALYSIS

The limitations of the present analysis arise from the narrative nature of the study and the lack of longitudinal clinical studies that directly investigate the relationship between bruxism and carious lesions. Most of the available data come from observational or in vitro studies, which makes the extrapolation of results to the clinical level difficult. Therefore, future research is needed to focus on this interaction under controlled clinical conditions, following a well-established protocol and using a standardized methodology.

CONCLUSIONS

The interaction between bruxism and carious lesions represents an emerging topic in dentistry, with significant implications for the management of carious diseases.

Current data suggest that there is a synergistic effect between parafunctional mechanical loads and the demineralization of hard dental structures, leading to accelerated deterioration of tissues. Both enamel and dentin affected by caries become more susceptible to mechanical stress, while bruxism is considered to favor lesion progression by altering structural integrity and dental morphology.

From a clinical point of view, this interaction implies the need for a multidisciplinary approach to the patient, and the efficient management of such cases requires the combination of preventive strategies, control of parafunctional activity, and restorative treatments adapted to specific biomechanical conditions.

In conclusion, the coexistence of bruxism and dental caries requires an individualized and complex clinical approach, focused not only on the treatment of existing lesions but also on the management of the underlying etiological factors. The development of future clinical studies is essential to clarify the causal relationship between them and to optimize clinical and therapeutic protocols.

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