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The Hidden Consequences of Bruxism and Periodontitis: A Case Report of Multiple Root Resorption

SUMMARY

Background: *Evidence regarding the effects of bruxism and periodontitis on root resorption is scarce and remains controversial in the literature. This case report aims to investigate the potential relationship between chronic periodontitis, severe bruxism, and aggressive external root resorption affecting multiple teeth in a patient with no systemic predisposition.* ***Materials and Methods:*** *Following clinical and conventional radiographic (panoramic and periapical) examinations, Cone-Beam Computed Tomography (CBCT) was performed for a detailed analysis of the resorptive lesions. An affected tooth with an extraction indication was subjected to histopathological examination. To exclude potential systemic etiologies, a comprehensive laboratory workup was conducted, including differential blood count, erythrocyte sedimentation rate, Hb%, alkaline phosphatase, T3, T4, TSH, serum potassium, and serum calcium levels.* ***Results:*** *Panoramic radiographs revealed cervical resorption in tooth #13 and a radiolucent area in the apical region of tooth #26. CBCT analysis confirmed localized external root resorption in the mesial root of tooth #26, accompanied by apical periodontitis involving all three roots. Histological findings confirmed the diagnosis of ERR, while the radiographic appearance of tooth #13 supported a suspicion of multiple external root resorption. Hematological and biochemical analyses showed no systemic pathology that could increase susceptibility to resorption.****Conclusion:*** *After excluding all other etiological factors, it was concluded that the severity and extent of the root resorption in this case may be associated with the combined effects of the patient's periodontitis and excessive occlusal forces related to severe bruxism.*

Keywords: Periodontitis, Bruxism, Case reports, Histology, Root resorption

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Introduction

The pathological relationship between root resorption and periodontal disease has long been an area that has not been sufficiently elucidated in the literature.[1–3] Studies evaluating the disease according to its severity, report a significant increase in the number of external resorption foci as the periodontitis picture worsens.[4,5] These lesions, thought to involve microtraumas affecting the periodontal ligament and leading to cementum and dentin loss, have been considered as processes that are difficult to detect radiographically and self-limiting.[6–8] The study by Crespo et al., which proved the presence of root resorption in teeth with periodontitis, has given a significant boost to the limited knowledge on this subject.[4] Especially in severe cases, the etiology is multifactorial; In addition to inflammation, excessive occlusal forces and idiopathic factors are also involved in the clinical picture.[1]

Bruxism, one of the most fundamental clinical manifestations of excessive occlusal forces, stands out as

a common oral parafunction that can lead to irreversible damage to the periodontium, such as root resorption.[9,10] When the pathological etiology of root surface resorptions is examined, bruxism is seen to be an important risk factor.[11] The basic mechanism in pathogenesis is thought to be that the severe occlusal trauma that occurs during this parafunction causes a concentration of force in the periodontium and disrupts local homeostasis, eliminating the protective cementoblast layer.[10,12,13] Although occlusal trauma due to bruxism is accepted in the literature as a potential predisposing factor for root resorption, the direct cause-and-effect relationship between harmful oral parafunctions and the risk of resorption is still a controversial issue.[10,11]

Histological data in the literature prove that external inflammatory root resorption develops in the apical region in up to 75% of teeth exhibiting periapical periodontitis.[14] The fact that current clinical diagnostic methods are limited in detecting this high prevalence poses a serious clinical risk; because the progression of resorptive lesions that cannot be diagnosed in the early stages can lead to total destruction of the root and eventual tooth loss.[15]

The aim of this case report is to highlight the potential role of the inflammatory environment created by severe periodontitis and the additional biomechanical stress caused by bruxism in the development of multiple root resorptions, which often have a complex etiology, and to discuss the relationship between these factors in light of current literature.

Case Report

A 60-year-old Turkish male patient applied to Kirikkale University Faculty of Dentistry with complaints of mobile teeth in the upper left posterior region. Patient, who had no systemic or genetic disease, was referred from the oral diagnosis unit to the endodontics clinic for a detailed examination of root resorption.

In endodontic examination, it was observed that there was an independent composite filling in the mesial and occlusal areas of tooth number 26, accompanied by mild pain on percussion and palpation, and third-degree mobility. The patient had no current or previous pain history. No sinus tract was detected. A negative response was obtained to the vitality tests.

Comprehensive periodontal examination revealed that full-mouth probing pocket depths generally ranged from 1-3 mm. However, localized pathological pockets reaching 5 mm were detected on the palatal surfaces of teeth with prosthetic restorations in the maxillary region and in the mesio-aproximal region of the right mandibular first molar. Clinical attachment loss ranging from 1 to 3 mm, more pronounced in teeth in the mandibular arch, was recorded even without accompanying pathological pocket depth. Furthermore, bone resorption in the mandibular posterior molars reached the furcation entry, resulting in Class II furcation involvement according to the Glickman classification. Radiographic evaluation revealed that bone loss extended to the middle third of the root, particularly in the anterior region. No bleeding was observed on general mouth probing. In the left maxillary first molar, the patient's primary complaint area, gingival recession of 2-3 mm was observed on the buccal and interproximal surfaces. A localized periodontal pocket 4 mm deep, extending from the mesio-palatal to the mesial interface of the affected tooth, and a concurrent Glickman Class II furcation defect were detected. Clinical examination revealed the presence of dental calculus, particularly in the mandibular anterior region. The patient's history showed heavy smoking (1-2 packs per day), regular brushing habits, but lack of interdental hygiene (Figure 1,2). Consequently, the patient was diagnosed with Stage III, Grade C periodontitis

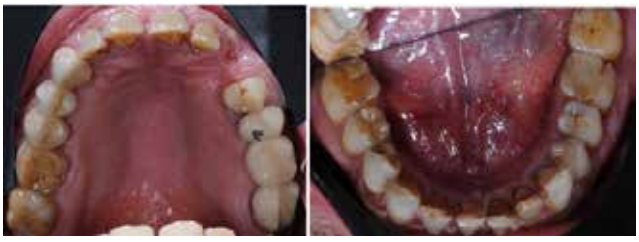

Figure 1. Patient's occlusion, lower and upper jaw occlusal intraoral photographs

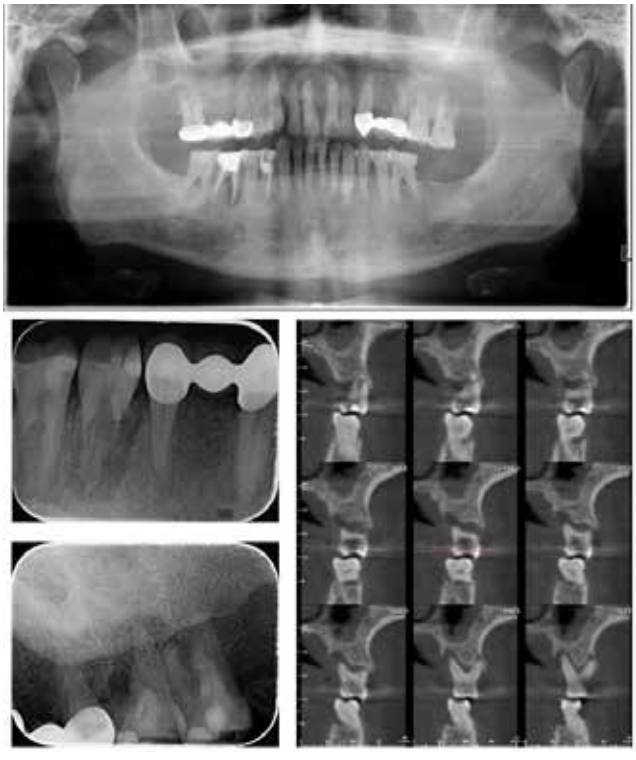

Figure 2: Panoramic X-ray, periapical radiography and CBCT

according to the current classification system, with heavy smoking acting as a significant grade modifier.[16]

In radiographic examination, a lesion was seen in the periapical region of tooth number 26 and it was determined that the buccal roots of the tooth were not evident in the periapical radiograph. Resorption was observed in the mesial root extending to the cervical third, accompanied by a wide apical periodontitis image (Figure 2).

The severe wear on the occlusal morphology of the teeth was questioned and it was learned that the patient had bruxism, which was especially severe at night and felt continued during the day (Figure 1). The patient stated that there was no pain, that his teeth gained mobility without any previous dental treatment and extracted after a while.

It was reported that tooth number 13 had been extracted due to severe mobility in faculty's surgical clinic. When the patient's panoramic radiograph was evaluated, the radiolucent structure observed on the cervical third surface of the root of tooth number 13, which had been extracted, was noteworthy; however, since the tooth was extracted before the patient applied to the Endodontics clinic, a detailed examination could not be performed (Figure 2). The patient, who did not attend routine check-ups and applied to our faculty recently, could not access his previous examination records.

Extraction of tooth number 26 was planned due to severe mobility, but the patient was informed about the need for a detailed pre-extraction examination. The inability to fully examine the root structure of tooth number 27 on both panoramic and periapical radiographs, along with root resorption in the mesial root of tooth number 26 and the cervical root of tooth number 23, increased suspicion of multiple root resorption. A CBCT scan was requested beforehand, and it was planned that if the indication for extraction of tooth number 26 is confirmed, the tooth to be extracted will be sent to a histology laboratory for evaluation.

A CBCT was performed for the patient for detailed examination, and except for the presence of an periapical lesion in the mesial root of tooth number 26, no apical periodontitis or root resorption was observed in the left posterior teeth. When examined in nine sections with 40 mm width and 0.50 intervals, apical periodontitis covering all three roots and more widespread in the palatal root was observed in tooth number 26. In the sections, resorption was observed from mesial to distal, up to the middle third of the mesial root, and small resorption areas were observed in the distal and palatal roots (Figure 2).

The patient had no history of medication; differential count, erythrocyte sedimentation rate, Hb%, alkaline phosphatase, T3, T4, TSH, serum potassium, and serum calcium values were also within normal values. The tooth was extracted due to severe mobility and sent to the histology laboratory (Figure 3).

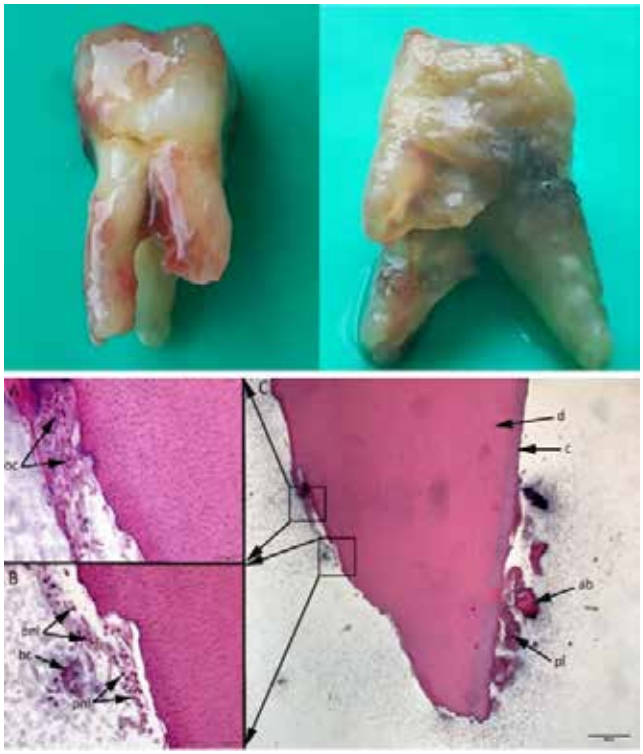

Figure 3: Histopathological photograph of a section of the resorbed tooth root taken from a depth of approximately 1300 µm. A) High-magnification image of the area in the upper square in C, B) High-magnification image of the area in the lower square in C, C) General image of the resorbed root at low magnification. In A, it represents oc) odontoclasts, in B, it represents pnl) polymorphonuclear leukocytes, bc) bacterial colony, in C, it represents ab) alveolar bone, c) cementum, d) dentin, pl) periodontal ligament. Hematoxylin-eosin staining, scale bar; A-B: 25 µm, C: 250 µm.

Following extraction, the tooth was placed in 10% neutral formalin for fixation. After 48 hours of fixation, the tooth was kept in Morse solution (aqueous solution containing 22.5% formic acid and 10% sodium citrate) for decalcification for 4 weeks, and the decalcification solution was changed once a week. After dehydration, the tooth sample was taken into xylene and then embedded in molten paraffin, subjected to vacuuming, and blocked in paraffin. 5 µm sections were taken from the prepared paraffin blocks, the sectioning process was carried out sagittally starting from the outer surface of the mesial root where resorption was seen, and sections belonging to the complete tooth root were obtained from the entire tooth root with serial sections taken at 100 µm intervals. The sections obtained were stained with hematoxylin-eosin and Masson's trichrome stain. The stained sections were examined under a light microscope.

It was observed that the direction of resorption started from the apical root tip and progressed towards the cervical, and the middle and apical roots were completely resorbed in mesiobuccal root (Figure 3) Polymorphonuclear leukocytes accompanied the bacterial colonies in the cervical region (Figure 4). It was observed that resorption was slower on

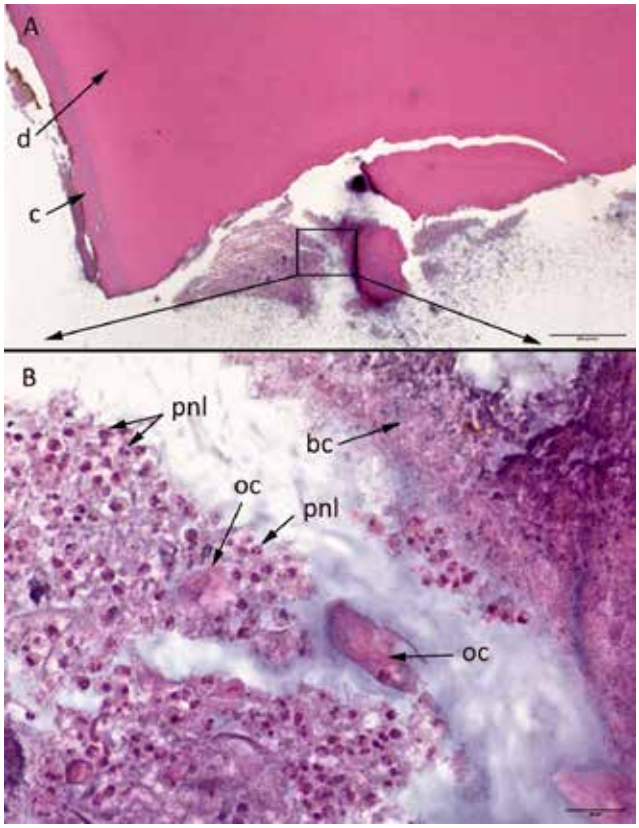

Figure 4: Histopathological photograph of a section of the resorbed tooth root taken from depth of approximately 2300 µm. A) General view of the resorbed root at low magnification, B) High magnification view of the area in the square in A. In A, it represents c)cementum, d) dentin, in B, oc) odontoclasts, bc) bacterial colony, pnl) polymorphonuclear leukocytes. Hematoxylin-eosin staining, scale bar; A: 250 µm, B: 25 µm.

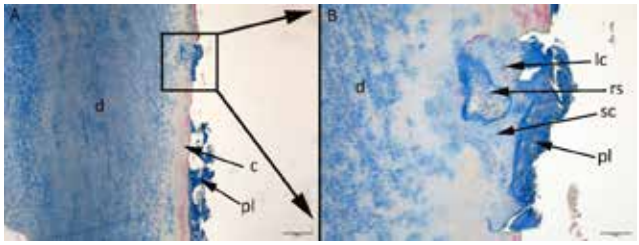

Figure 5: Histopathological photograph of a section of the resorbed tooth root taken from a depth of approximately 4000 µm. A) General view of the resorbed root at low magnification, B) High magnification view of the area in the square in A. In A, it represents c) cement, d) dentin, pl) periodontal ligament, in B, it represents d) dentin, lc) cementoblasts in lacunae, rs) resorption area, sc) secondary cementum, pl) periodontal ligament. Masson's trichrome staining, scale bar; A: 250 µm, B: 25 µm.

the alveolar bone surface and faster on the contralateral side. Because the apical root was completely resorbed, the crown pulp was examined to assess the progression of resorption. The activity of odontoclasts in the resorption region and the presence of bacterial colonies in the immediate vicinity, as well as polymorphonuclear leukocytes, are noteworthy.

In the area where the periodontal ligament is observed, the epithelial cell rests of Malassez indicates homeostasis of the losses due to resorption, but the inflammatory cells seen in the area where the periodontal ligament is located indicate that inflammation continues. The inflammatory response and odontoclast activity due to the severe bacterial infection seem to have caused the resorption to progress aggressively. In the area where the periodontal ligament is observed, resorption areas were observed in the cementum. At the same time, it was observed that the resorption areas in the areas where resorption was observed were filled with new tissue similar to cementum (more like bone) or collagen (Figure 5). As a result of histological evaluations, it was concluded that there was external inflammatory root resorption.

As a result of all evaluations, the patient was thoroughly informed at our clinic. Information was provided about the importance of routine check-ups, bruxism, and the possible consequences of oral care. Following the examination and tests, the patient who had a tooth extracted stated that they thought tooth loss was genetic and that no matter how much attention they paid to oral health, they could not prevent it, and that they were unaware that bruxism and periodontal health could play a role in pathologies. At the request of the patient, who did not want to experience tooth loss again, they were referred to the relevant clinics for treatment, and a treatment and monitoring program was established.

Discussion

The cause of external root resorption, which can be seen as a result of mechanical stimuli, infection, inflammatory and neoplastic processes, systemic diseases, and trauma, is unknown in some cases and can also be defined as idiopathic.[17] Periapical inflammation in chronic apical periodontitis could be caused resorption, and the amount of resorption could be increased as the severity increases.[4] Pulp necrosis can also occur due to the interruption of apical blood supply due to chronic trauma. Although it does not always result in inflammation in the apical region, in the presence of infection, the inability of defense cells to reach the tissue can result in chronic inflammation in the periapical region.[18–20] Root resorption is three times more common in teeth affected by periodontal disease compared to unaffected teeth.[4] This case report examines and interprets in detail the potential factors that may be associated with root resorption; it also evaluates the possible role of severe bruxism and periodontitis in root resorption pathology.

The consequences of occlusal trauma on the pulp have been experimentally proven.[21] It is known that bruxism causes masticatory muscle pain, temporomandibular joint pain, headache, mechanical tooth wear, prosthesis complications and cracked teeth. In the study conducted by Moreira-Souza et al., it was showed that bruxism and increased masticatory muscle activity were associated

with root resorption in second molars adjacent to impacted lower third molars.[22] Apical periodontitis due to occlusal trauma may be observed in patients with severe bruxism. It should not be forgotten that severe apical periodontitis may cause root resorption. Since bruxism may play a role in the etiology of apical periodontitis, it should be questioned with a detailed anamnesis in the presence of unexplained pain.[23] The current literature indicates that parafunctional habits, especially bruxism, are a potential predisposing factor in patients diagnosed with external cervical root resorption.[24,25] The severity and varying nature of the root resorption observed in teeth 13 and 26 in this patient were noteworthy. After excluding most systemic and local predisposing factors, the presence of bruxism and periodontitis emerged as possible contributing factors. However, due to the absence of the patient's previous dental records, a clear timeline cannot be established. This precludes us from definitively determining whether these factors directly initiated the resorptive process or to what extent they exacerbated the severity of the condition. Although a definitive causal relationship cannot be established, in light of literature findings, it is suggested that these occlusal and inflammatory factors may be associated with the resorption and could have played an aggravating role in the pathology.

The incidence of root resorption increases in the presence of periodontal disease. It has been observed that the more severe the periodontal infection, the greater the number of resorptions. The relationship between resorptions localized in different areas of the root surface and periodontal damage has been investigated, but the data have not been found to be consistent.[4,26] Mahajan, Aaditi et al. reported that the severity of resorption increased with increasing severity of periodontal disease compared to a healthy control group; the most affected area by resorption was the apical third.[27] Zisis et al. reported two case reports in which all possible non-periodontitis causes were ruled out, bringing the relationship between chronic periodontitis and root resorption back into question.[28] In the present case, while both endodontic and periodontal pathologies coexisted, it is crucial to delineate their relative and distinct contributions to the resorptive process. Apical periodontitis, driven by intrapulpal bacterial toxins and infection, predominantly stimulates odontoclastic activity in the periapical region.[18,19,21] Conversely, external root resorption associated with periodontal disease operates through a different biological mechanism; chronic inflammation from severe periodontitis facilitates the release of pro-inflammatory cytokines (such as IL-1β, TNF-α, and PGE2) in the cervical and middle thirds of the root, initiating cementum breakdown from the marginal periodontium.[29] In tooth 26, the convergence of these distinct entities, coronal-apical progression from periodontal pocketing and apical-coronal progression from endodontic infection, likely created a synergistic inflammatory environment that accelerated the extensive multiple root resorption. Moreover, the patient's heavy smoking habit (1-2 packs per day) likely contributed to this pathogenesis, as the literature demonstrates that smoking can create an ischemic environment that impairs tissue repair by suppressing the immune response and causing peripheral vasoconstriction.[30] In conclusion, it is considered that the patient's severe periodontitis, combined with poor oral hygiene and chronic smoking, may have established a cumulative inflammatory and ischemic environment. The interplay of all these factors likely contributed to the eventual formation of pathological pockets and the extensive root resorption diagnosed in tooth 26.

Root resorption is multifactorial; it is quite difficult to understand the etiology and pathogenesis and to make a diagnosis. The reliability of routinely used radiographs is controversial. It has been reported that most of the resorptions in teeth with apical periodontitis cannot be diagnosed with routine radiographic techniques and that there is more apical root resorption than diagnosed with histological examinations.[15] In SEM examinations, external root resorption areas were observed at different levels in teeth with apical periodontitis.[31] In our histological examination, the presence of bacterial colonies and polymorphonuclear leukocytes, together with increased osteoclastic activity in the resorption area, was observed. Histological examinations can be performed after the tooth is extracted and do not provide any clinical benefit. In our case, histological examination was performed to support the diagnosis.

Various methods such as intracanal dressing with calcium hydroxide, surgical cleaning and restoration of the resorption area, and stopping resorption with regenerative endodontic treatment are tried in the treatment of external root resorption and case reports are presented.[32,33] In our case, tooth number 26 was indicated for extraction because it had severe mobility and treatment could not be applied.

The patient's failure to attend routine check-ups and long intervals between dental examinations prevent a clear conclusion from being reached regarding how bruxism and periodontitis affect the pathology. Furthermore, there are three different conditions that can occur in a tooth exposed to chronic occlusal stress due to trauma: root resorption, periapical inflammation, and tooth mobility. Given that these three conditions may be related, bruxism cannot be directly linked to any one of them, but it can be said to contribute to the process.

The main limitations of this case report include its single-case design, the lack of previous dental records, and the consequent inability to establish a precise timeline of the disease progression. Furthermore, it is important to acknowledge that the diagnosis of bruxism was primarily

based on clinical findings and patient history rather than objective diagnostic methods.

Conclusion

External inflammatory root resorption has a complex and multifactorial etiology. The findings of this case support a possible association rather than direct causation between excessive occlusal forces and periodontal inflammation. The conditions that cause resorption should not be evaluated individually; it should be kept in mind that they may be interrelated.

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