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External cervical resorption: why does it occur?

UPDATE

Reabsorciones cervicales externas: ¿por qué se producen?

Reabsorções cervicais externas: ¿por que ocorrem?

Abstract

External cervical resorption (ECR) is a relatively rare but highly aggressive form of dental tissue destruction. Despite numerous reported cases over the decades and various clinical studies investigating this condition, its etiology remains unclear.

This work aims to identify potential predisposing factors associated with the development of ECR through a broad narrative review of the available literature.

Based on current evidence, it is concluded that ECR is a pathological process of multifactorial origin. The combination of predisposing factors appears to increase the likelihood of its development. The factors most frequently linked to ECR development include dental trauma, orthodontic movements involving uncontrolled forces, and internal bleaching treatments using highly concentrated agents combined with heat application and improper chamber preparation.

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Resumen

La reabsorción cervical externa (RCE) es una forma de destrucción del tejido dentario, relativamente poco frecuente pero muy agresiva. A pesar de los casos reportados a lo largo de décadas y de los estudios clínicos que las han investigado, su etiología aún no está clara.

Este trabajo tiene como objetivo identificar los potenciales factores predisponentes relacionados al desarrollo de RCE. Para ello se realizó una revisión amplia de la literatura disponible, de tipo narrativa.

En virtud de la evidencia encontrada se concluye que las RCE son procesos patológicos de etioloagía multifactorial. La combinación de factores predisponentes parece potenciar la posibilidad de que estos procesos se desarrollen. Los traumatismos dentarios, los movimientos ortodóncicos mediante fuerzas no controladas y los tratamientos de blanqueamiento interno con agentes en altas concentraciones combinados con aplicación de calor e incorrecta preparación cameral, parecen ser los factores más frecuentemente relacionados al desarrollo de RCE.

Palabras clave: Reabsorción cervical externa, traumatismo dentario, blanqueamiento dental.

Resumo

A reabsorção cervical externa (RCE) é uma forma relativamente rara, mas muito agressiva, de destruição do tecido dentário. Apesar dos casos relatados ao longo de décadas e dos estudos clínicos que os investigaram, a sua etiologia ainda não é clara.

Este trabalho tem como objetivo identificar potenciais fatores predisponentes relacionados ao desenvolvimento de reabsorções cervicais externas, para o que foi realizada uma ampla revisão da literatura narrativa disponível.

Com base na evidência disponível, conclui-se que os RCE são processos patológicos de causa multifatorial e a combinação de fatores predisponentes parece aumentar a possibilidade de desenvolvimento destes processos. Trauma dentário, movimentações ortodônticas e tratamentos de clareamento interno com agentes em altas concentrações combinados com aplicação de calor e preparo incorreto da câmara, parecem ser os fatores mais frequentemente relacionados ao desenvolvimento de RCE.

Palavras-chave: Reabsorção cervical externa, traumatismo dentário, clareamento dental.

Introduction

External cervical resorption (ECR) is a relatively rare but highly aggressive form of dental tissue destruction. Epidemiological studies estimate its prevalence to range between 0.02% and 2.3%.[1,2] It is often an incidental finding, both clinically and radiographically, though certain clinical signs may be present, such as localized gingival inflammation, bleeding, and sensitivity to thermal changes or percussion.

ECR starts at the cervical region of the root surface and progresses apicocoronally and circumferentially into the dentin. Cone beam computed tomography (CBCT) has significantly improved the diagnosis and management of ECR by providing precise information on its location, extent, and proximity to the pulp space—critical parameters for successful treatment. [3]

Despite decades of reported cases and clinical studies investigating various risk factors for ECR, the relationship between these factors and the onset and progression of the condition is not completely understood.[3]

GENERAL OBJECTIVE

This review aims to identify potential predisposing factors associated with the development of external cervical resorption.

SPECIFIC OBJECTIVES

- Identify the characteristics of different types of cervical resorption.
- Understand the mechanisms that drive the onset and progression of these lesions.
- Provide guidelines for preventing ECR lesions in cases of internal dental bleaching.

Methodology

A broad narrative review of the available literature was conducted to identify studies analyzing the main causes of external cervical resorption. The databases PubMed

(Medline) and SciELO, as well as the resources Timbó and Google Scholar, were consulted.

The search strategy was as follows:

((“Root Resorption”[Mesh]) AND “Tooth Bleaching” [Mesh] AND (y_10[Filter])) OR (root cervical resorption external teeth whitening AND (y_10[Filter]))

(((“Root Resorption”[Mesh]) OR (root cervical resorption external) AND (Traumatisms)) AND (y_10[Filter]))

Exclusion criteria included studies published before 2013 and case reports. A total of 92 records were retrieved. After screening titles and abstracts, 11 records were selected. The search was completed by reviewing and tracking references in those records, gathering 17 additional publications. Eight records predating the exclusion date were included due to their relevance to the study. In total, 29 records were analyzed. A search flow diagram is presented in **Figure 1**.

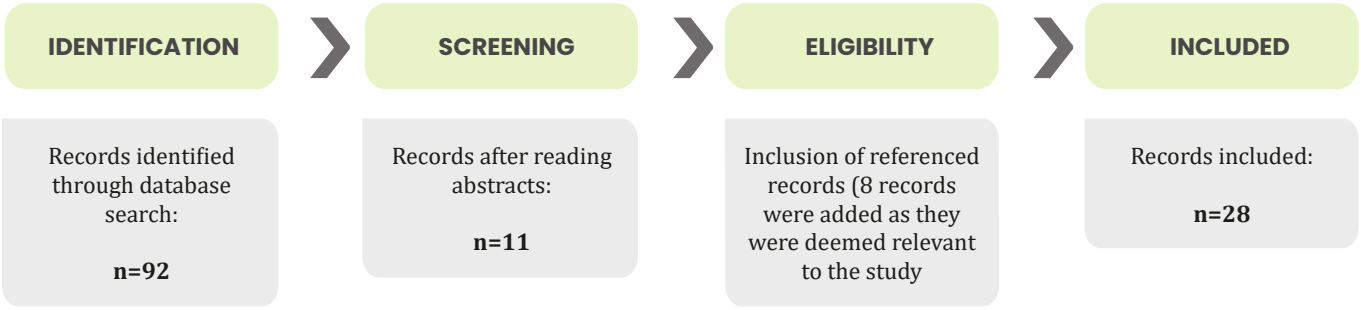

Figure 1 Search flow diagram

Background

Since Andreasen first described the classification of root resorptions in the 1970s, several classifications using varied terminology have been proposed.[4] Physiologic root resorption occurs during the exfoliation of primary teeth and is thought to aid the eruption of permanent teeth. In contrast, pathological resorption affects the permanent dentition and leads to undesirable outcomes.[3]

Pathologic root resorption can be classified as external or internal, depending on the site of the lesion.[3–5] External resorption can be further categorized as:[6,7]

a. External superficial resorption.
b. External cervical resorption.
c. External inflammatory resorption.
d. Replacement resorption.
e. Transient apical rupture.

ECR is primarily an asymptomatic pathological process in the permanent dentition.[4] Traditionally, diagnosis has been made using radiographs. Recent advancements in clinical evaluation methods of the lesions, along with the use of cone beam computed tomography (CBCT) for diagnostic imaging, have provided more detailed and accurate information regarding the nature of ECR.[3,8]

It begins at the cervical third of the root, apical to the junctional epithelium. The defect extends horizontally toward the root canal and vertically, either apically, coronally, or both. During this process, the pulp generally remains intact, except in the final stage, when resorption may reach the root canal space.[9]

Heithersay proposed a classification based on the degree of destruction and severity of ECR.[10] According to Mavridou, this classification has two major limitations: it is based only on two dimensions of the lesion extent and does not account for the reparative nature of ECR, which has been recently reported.[11] Patel suggests a classification that addresses the three-dimensional nature of the lesions and relies on computed tomography analysis.[12] **Table 1**

TABLA 1
Three-dimensional classification of Patel, table modified by the authors.[12]

HEIGHT	CIRCUMFERENTIAL SPREAD	PROXIMITY TO THE ROOT CANAL
1. At cemento-enamel junction level or coronal to the bone crest (supracrestal)	A: ≤90°	d: Lesion confined to dentine
2. Extends into coronal third of the root and apical to the bone crest (subcrestal)	B: >90° a ≤180°	p: Probable pulpal involvement
3. Extends into mid-third of the root	C: >180° a ≤270°	
4. Extends into apical third of the root	D: >270°	

Clinically, these lesions may be mistaken for carious lesions. However, they differ in terms of hardness and roughness on probing, while subgingival caries are soft. Additionally, cervical resorption lesions often bleed profusely upon probing due to the high vascularization of the tissue.[6]

Development

PATHOGENESIS

The exact causes and pathogenesis of ECR remain unclear. However, clinical and experimental findings suggest that the resorption process results from the proliferation of periodontal ligament tissues[3]. The inflammatory response theory states that ECR is triggered solely by an inflammatory reaction. It is believed that stimulation of the periodontal ligament (e.g., internal bleaching or abnormal external pressure) induces an immune response, which in turn activates osteoclastogenesis. Based on this, some researchers have suggested that ECR is an aseptic process.[2,3,13]

Conversely, the infection theory argues that microorganisms originating from the gingival sulcus or pulp are responsible for ECR. This theory suggests that resorption may be initiated by endotoxins or as an inflammatory response to microbial infection.[2,3]

Despite differing perspectives on the nature of ECR, the general consensus is that inflammation is a prerequisite for its onset.[3] Histopathological analyses of resorption lesions confirm that the degradation of dental hard tissues occurs following an inflammatory response. However, the precise relationship between inflammation and osteoclastogenesis remains unclear.[3]

Studies have shown that proinflammatory mediators (e.g., interleukin 6 and interleukin 1 beta), which play a key role in ECR, are also elevated in periodontitis. This suggests that osteoclastogenesis in ECR and periodontitis may develop through a similar mechanism.[3]

The initiation of osteoclastogenesis requires two key elements: an injury and a stimulus factor[3,4]. In most cases of ECR, signs of mechanical stress or inflammation have been observed. Both factors can cause significant damage to the periodontal vascular system, leading to microcirculatory dysfunction. Ischemia and hypoxia can disrupt bone metabolism homeostasis, triggering osteoclastogenesis.[3,9]

In external resorption, the pathological process originates in the periodontal ligament, whereas in internal resorption, it arises from the pulp tissue.[3] Dentin is internally lined by the odontoblastic layer and predentin, and externally by the cementoblastic layer and precementum. These layers form a protective barrier against resorption, as odontoclasts are unable to adhere to or degrade the non-mineralized matrix. The inhibitory effect of the organic precementum and predentin makes an intact root resistant to resorption. However, when an initial stimulus such as infection or trauma occurs, mineralized dentin becomes vulnerable. Thus, two conditions are necessary for resorption to take place: loss or disruption of the protective precementum or predentin layer and subsequent damage to the exposed root surface.[4,6,8] This process may be self-limiting and go unnoticed clinically; however, once initiated by an injury and sustained by a stimulus, the destruction of hard dental tissue will continue.[4]

ETIOLOGY

External resorption has multiple causes and is more prevalent than internal resorption, which is relatively rare.[4] Potential etiologic factors that cause damage to the root surface in the cervical area include dental trauma, orthodontic treatment, internal bleaching, periodontal treatment, and idiopathic causes.[3,8,9]

Heboyan et al.[6] list several endogenous and exogenous factors, which are shown in **Table 2**.

TABLE 2
Factors Related to ECR, table modified by the authors.[6]

EXOGENOUS FACTORS	ENDOGENOUS FACTORS (HOST-DERIVED)
Trauma	Age
Caries	Gender
Pulpitis	Ethnicity
Dental preparation for prosthetic purposes	Genetic predisposition
Orthodontic intervention	General diseases (chronic asthma, hormonal disorders, etc.)
Pulpotomy by vital amputation method	Impacted teeth
Direct pulp capping with Ca(OH)	Root anatomy
Root resection	Occlusal relationships
Dental fractures	Habits
Bleaching	

Regarding internal bleaching, Harrington and Natkin were the first to report ECR following treatment in 1979.[14] Since then, numerous studies have examined the association between internal bleaching and cervical resorption.[15–17]

For ECR to develop, the bleaching agent must reach the periodontal tissues through the dentinal tubules.[18] Several factors may increase the diffusion of hydrogen peroxide into the ligament space, including larger dentinal tubules in young patients, anatomical defects at the cementoenamel junction, bleaching agents with low molecular weight and high concentrations, the absence or improper placement of a cervical barrier, and the application of heat during the bleaching procedure.[15,19]

It has been suggested that hydrogen peroxide denatures dentin, triggering an inflammatory immune response. The pH at the root surface drops to approximately 6.5, and some studies propose that this may lead to increased osteoclast activity, potentially causing ECR.[2,15]

Mavridou et al.[20] analyzed endodontically treated teeth with ECR and found that resorption was more intense and aggressive in these teeth compared to those with pulp vitality. This may be explained by the absence of the predentin layer, reduced oxygen content due to the lack of blood supply, and the chemical alterations in dentin tissue caused by endodontic treatment.

Regarding trauma, a retrospective study identified "history of trauma" as the only significant local predisposing factor for ECR.[9] It has been suggested that patients with a history of dental trauma should undergo radiographic monitoring, even in the absence of clinical signs or symptoms, due to the risk of "late" ECR.

A systematic review published in 2020.[21] analyzed the incidence of root resorption in relation to concussion, subluxation, lateral luxation, intrusion, and extrusion. The findings suggest that resorption is most frequently associated with intrusive luxation.

Orthodontic treatment is a commonly identified risk factor not only for ECR but also for other types of external resorption.[1]

Discussion

The first cross-sectional study on potential predisposing factors for ECR was conducted by Heithersay in Australia in 1999.[10] A total of 11 risk factors were reported, the most frequent being:

- Orthodontic treatment
- Trauma
- Internal bleaching (in combination with other factors)
- Restorative treatment

In 16.4% of cases, no risk factors could be identified, leading to their classification as idiopathic ECR.[10] The study highlighted a strong association between ECR and both trauma and orthodontic treatment, while intracoronal bleaching was linked to a lower proportion of cases-though still exceeding 10%. **Figure 2**

In 2017, Mavridou[22] conducted a retrospective study in Europe, identifying the following main factors:

- Orthodontic treatment (45.7%)
- Trauma (28.5%)

This study also identified several novel risk factors, including parafunctional habits, poor oral health, malocclusion, and extraction of adjacent teeth. In most cases (59%), more than one potential predisposing factor was present, reinforcing the idea that ECR is multifactorial rather than idiopathic[22] **Figure 3**

The prevalence of orthodontic treatment as a predisposing factor was higher in this study compared to Heithersay's, which may be attributed, in part, to increased awareness and improved diagnostic capabilities for ECR, as well as a rising incidence of orthodontic treatment.[5]

In 2020, Jeng conducted a cross-sectional study in Asia.[1] The most notable difference from previous studies was the much lower proportion of cases associated with orthodontic treatment (15.87%) compared to trauma (33.33%) and periodontal treatment (26.98%). This was

attributed to differences in craniofacial patterns and aesthetics-related assessments among patients from different regions, which could influence orthodontic treatment modalities.[3]

Restorative treatment (17.46%) and extraction of an adjacent tooth (7.94%) were also identified as major predisposing factors. Notably, none of the cases in this study were classified as idiopathic ECR. [1] **Figure 4**

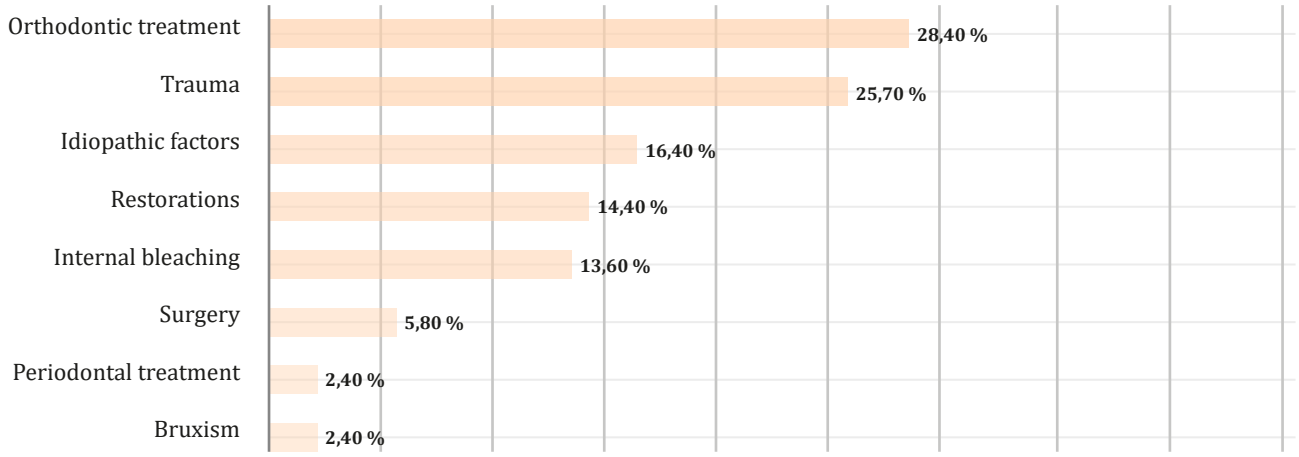

Figure 2 FECR-associated factors in the study by Heithersay et al. The high percentage linked to trauma and orthodontic treatment is highlighted. Graph modified by the authors.[3]

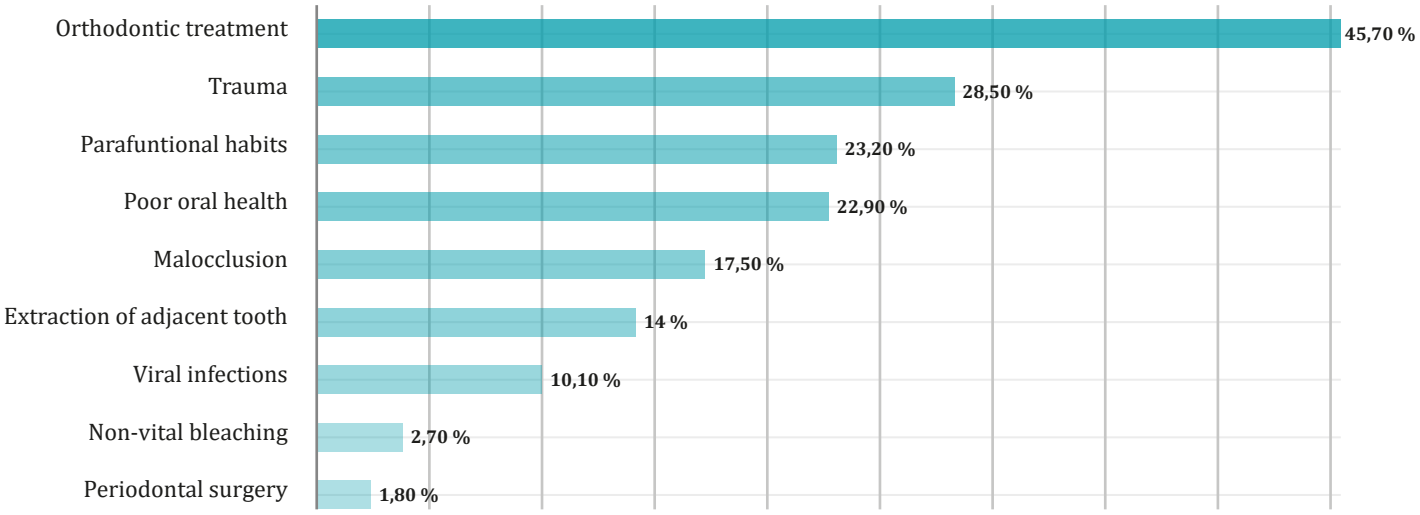

Figura 3 ECR-associated factors in the study by Mavridou et al. The high percentage related to trauma, orthodontic treatment, parafunctional habits, and poor oral hygiene is highlighted. Graph modified by the authors.[3]

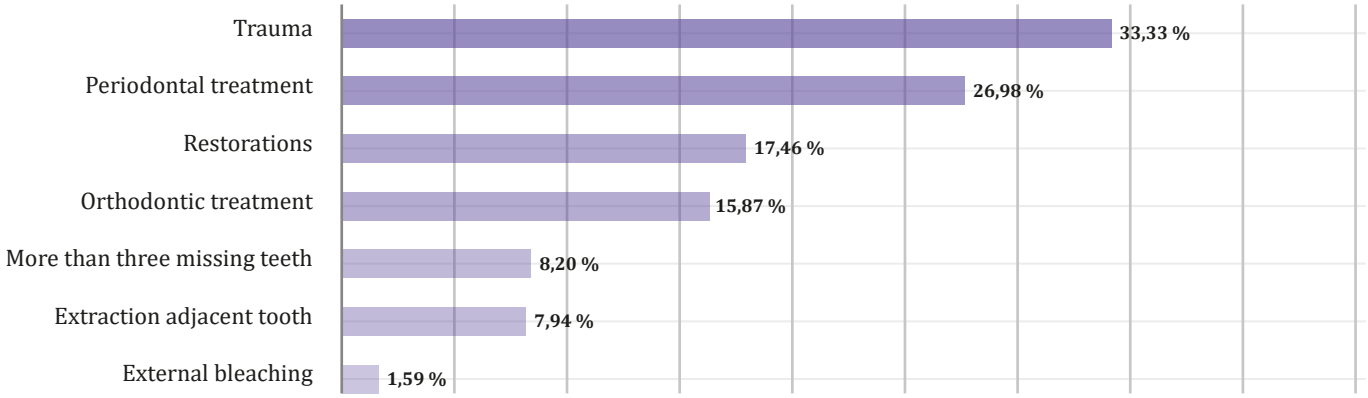

Figura 4 ECR-associated factors in the study by Jeng et al. In this case, trauma remains the most prevalent factor, while the association with orthodontic treatment decreases considerably. Graph modified by the authors.[3]

Interestingly, internal bleaching was not identified as a major risk factor in the studies by Mavridou and Jeng. This could be attributed to modifications in bleaching treatments, including the type and concentration of bleaching agents as well as in techniques, which have significantly reduced the risk of ECR.[5] Since 2011, the European Union has prohibited bleaching agents containing more than 6% hydrogen peroxide,[23] and the use of light- or heat-activated bleaching techniques is discouraged.[15,16,19] Dahl et al.[19] reviewed the adverse effects of internal bleaching and reported a 7% incidence of ECR over an 8-year follow-up in teeth treated with high concentrations of hydrogen peroxide combined with thermocatalytic techniques.

Heithersay et al.[24] investigated the incidence of ECR in teeth that had undergone internal bleaching, finding that 78% of cases had a history of trauma and all had received high-concentration bleaching combined with heat. Among these, 1.96% developed ECR, all with a history of trauma. It is important to consider that dental discoloration is often a consequence of trauma; therefore, cases of ECR observed in previously bleached teeth may be attributable to trauma rather than the bleaching treatment itself.[16]

Abbot and Heah[25] analyzed 255 internally bleached teeth from 203 patients over a 5-year follow-up and found no cases of ECR. Bleaching agents based on carbamide peroxide have demonstrated lower extraradicular diffusion, making them the preferred choice for internal bleaching treatments. These agents break down to produce ammonia, which increases the pH, making it more alkaline. Additionally, they diffuse more slowly through dentin, allowing more time for deionization and reducing the concentration of peroxide that reaches the root surface without reacting.[15,17]

A key aspect to consider is the proper application of a cervical barrier, as this helps prevent diffusion into the cervical region. A 2022 in vitro study[26] analyzing the penetration of bleaching agents into the cervical area using different barrier materials confirmed that an adequate cervical barrier, along with the use of low-concentration products, effectively reduces the passage of agents into the cervical region, thereby lowering the risk of ECR.

A 2023 systematic review and meta-analysis[27] compared the microleakage associated with different materials used as barriers in chamber preparation for internal bleaching procedures. Although the results are limited to in vitro studies, they indicate that the placement of glass ionomer, composite resin, Cavit, or MTA is effective in preventing coronal microleakage.

In 2018, the European Society of Endodontics published a statement by a committee of experts on ECR, affirming that a history of dental trauma and/or orthodontic treatment are the factors most frequently associated with ECR.[28]

A retrospective study conducted between 2009 and 2019[1] in the Taiwanese population reported that the most significant predisposing factors were trauma, periodontal treatment, and orthodontic treatment. Dental bleaching was associated with only a small percentage of cases—only one case involved external bleaching—likely due to improvements in bleaching procedures. Approximately 10% of cases presented exposed dentin in the cervical area due to a lack of bonding between enamel and cementum.

Another 2023 retrospective study[29] found that 1%

of endodontically treated teeth exhibited ECR. Among these cases, patients with a history of trauma or orthodontic treatment had the most severe presentations. Additionally, parafunctional habits such as bruxism were identified as predisposing factors for ECR.

Regarding the location of the lesions, most cases of ECR have been observed in the maxilla, primarily affecting the maxillary anterior teeth.[3,29] This has been attributed to the greater movement these teeth undergo during orthodontic treatment and their increased susceptibility to trauma.[3] Recent retrospective analyses indicated that anterior teeth account for half of all ECR cases.[1,9]

Conclusions

ECR is a pathological process with a multifactorial etiology, and the combination of predisposing factors appears to increase the likelihood of its development. Dental trauma, orthodontic movements, and internal bleaching treatments using high concentrations of bleaching agents combined with heat application and improper chamber preparation are among the factors most frequently associated with ECR.

Among traumatic lesions, luxations and avulsions seem to be responsible for the most severe cases of ECR. Preventive measures against dental trauma, such as the use of mouthguards in high-risk sports, are recommended.

A thorough patient history, along with detailed clinical and radiographic examinations, is essential before indicating bleaching treatment in endodontically treated teeth to identify predisposing factors such as a history of trauma or orthodontic treatment. Internal bleaching should be performed with proper chamber preparation and low-concentration bleaching agents. Patients should be informed about the potential risk of ECR, particularly in younger individuals, as their dentinal tubules have a larger diameter, increasing the likelihood of diffusion to the cervical area.

To date, the etiology of ECR remains unclear, and further studies are needed to better understand its predisposing factors and etiopathogenesis.

REFERENCES

1. Jeng PY, Lin LD, Chang SH, Lee YL, Wang CY, Jeng JH, et al. Invasive Cervical Resorption—Distribution, Potential Predisposing Factors, and Clinical Characteristics. J Endod [Internet]. 2020;46(4):475–82. Available from: https://doi.org/10.1016/j.joen.2020.01.011

2. Rotondi O, Waldon PA, Kim SG. The disease process, diagnosis and treatment of invasive cervical resorption: A review. Dent J (Basel). 2020;8(3).

3. Chen Y, Huang Y, Deng X. A Review of External Cervical Resorption. J Endod [Internet]. 2021;47(6):883–94. Available from: https://doi.org/10.1016/j.joen.2021.03.004

4. Aidos H, Diogo P, Santos JM. Root resorption classifications: A narrative review and a clinical aid proposal for routine assessment. Eur Endod J. 2018;3(3):134–45.

5. Patel S, Mavridou AM, Lambrechts P, Saberi N. External cervical resorption-part 1: histopathology, distribution and presentation. Int Endod J. 2018;51(11):1205–23.

6. Heboyan A, Avetisyan A, Karobari MI, Marya A, Khurshid Z, Rokaya D, et al. Tooth root resorption: A review. Sci Prog. 2022;105(3):1–29.

7. Patel S, Krastl G, Weiger R, Lambrechts P, Tjäderhane L, Gambarini G, et al. ESE position statement on root resorption. Int Endod J. 2023;56(7):792–801.

8. Patel S, Ford TP. Is the resorption external or internal? Dent Update. 2007;34(4):219–29.

9. Irinakis E, Aleksejuniene J, Shen Y, Haapasalo M. External Cervical Resorption: A Retrospective Case-Control Study. J Endod [Internet]. 2020;46(10):1420–7. Available from: https://doi.org/10.1016/j.joen.2020.05.021

10. Heithersay GS. Invasive cervical resorption: an analysis of potential predisposing factors. Quintessence Int. 1999;30:83–95.

11. Mavridou AM, Hauben E, Wevers M, Schepers E, Bergmans L, Lambrechts P. Understanding External Cervical Resorption in Vital Teeth. J Endod [Internet]. 2016;42(12):1737–51. Available from: http://dx.doi.org/10.1016/j.joen.2016.06.007

12. Patel S, Foschi F, Mannocci F, Patel K. External cervical resorption: a three-dimensional classification. Int Endod J. 2018;51(2):206–14.

13. Heithersay GS. Invasive cervical resorption. Endod Topics. 2004;7(1):73–92.

14. Harrington GW NE. External Resorption Associated With Bleaching of a Non-Vital Tooth. J Endod. 1979;5(1):344–8.

15. Newton R, Hayes J. The association of external cervical resorption with modern internal bleaching protocols: what is the current evidence? Br Dent J. 2020;228(5):333–7.

16. Kahler B. Present status and future directions – Managing discoloured teeth. Int Endod J. 2022;55(S4):922–50.

17. Nathan KB, Nadig RR, Job TV, Nithin PV, Karthik R, Choudary S. Radicular peroxide penetration from different concentrations of Carbamide Peroxide Gel during intracoronal bleaching-an in vitro study. Journal of Contemporary Dental Practice. 2019;20(5):587–92.

18. Lou EK, Cathro P, Marino V, Damiani F, Heithersay GS. Evaluation of hydroxyl radical diffusion and acidified thiourea as a scavenger during intracoronal bleaching. J Endod [Internet]. 2016;42(7):1126–30. Available from: http://dx.doi.org/10.1016/j.joen.2016.04.010

19. Dahl JE, Pallesen U. Tooth bleaching - A critical review of the biological aspects. Critical Reviews in Oral Biology and Medicine. 2003;14(4):292–304.

20. Mavridou, A. M., Hauben, E., Wevers, M., Schepers, E., Bergmans, L., Lambrechts P. Understanding external cervical resorption patterns in endodontically treated teeth. Int Endod J. 2017;50(12):1116–33.

21. de Souza BDM, Dutra KL, Reyes-Carmona J, Bortoluzzi EA, Kuntze MM, Teixeira CS, et al. Incidence of root resorption after concussion, subluxation, lateral luxation, intrusion, and extrusion: a systematic review. Clin Oral Investig. 2020;24(3):1101–11.

22. Mavridou AM, Bergmans L, Barendregt D, Lambrechts P. Descriptive Analysis of Factors Associated with External Cervical Resorption. J Endod [Internet]. 2017;43(10):1602–10. Available from: http://dx.doi.org/10.1016/j.joen.2017.05.026

23. European Union. EU Council Directive 2011/84/EU of 20 September 2011. Official Journal of the European Union [Internet]. 2011;(7):36–8. Available from: https://eur-lex.europa.eu/%0AriServ/LexUriServ.do?uri=OJ:L:2011:283:0036:0038:en:PDF

24. Heithersay GS, Dahlstrom SW, Marin PD. Incidence of invasive cervical resorption in bleached root-filled teeth. Aust Dent J. 1994;39(2):82–7.

25. Abbott P, Heah SYS. Internal bleaching of teeth: An analysis of 255 teeth. Aust Dent J. 2009;54(4):326–33.

26. Sakalli B, Basmaci F, Dalmizrak O. Evaluation of the penetration of intracoronal bleaching agents into the cervical region using different intraorifice barriers. BMC Oral Health [Internet]. 2022;22(1):1–8. Available from: https://doi.org/10.1186/s12903-022-02300-4

27. Chen P, Chen Z, Teoh YY, Peters OA, Peters CI. Orifice barriers to prevent coronal microleakage after root canal treatment: systematic review and meta-analysis. Aust Dent J. 2023;68(2):78–91.

28. Patel S, Lambrechts P, Shemesh H, Mavridou A. European Society of Endodontology position statement: External Cervical Resorption. Int Endod J. 2018;51(12):1323–6.

29. Huang J, Walsh RM, Witherspoon DE, Khalighinejad N, Tam EC, Jalali P, et al. The prevalence, characteristics, and risk factors of external cervical resorption: a retrospective practice-based study. Clin Oral Investig [Internet]. 2023;27(9):5595–604. Available from: https://doi.org/10.1007/s00784-023-05180-2

Conflict of Interest

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