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Root Resorption in the Permanent Teeth. A Review

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Review

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ABSTRACT

Root resorption is losing dental hard tissue because of odontoclastic activity. It is undesirable and pathological in permanent teeth. Root resorption may happen within the root canal, called internal root resorption, or on the outer surface of the root, called external root resorption. Regardless of where it occurs, root resorption is irreversible, can cause pain for the patient, necessitates treatment, and in some circumstances, results in the early loss of the affected tooth. It might be challenging to diagnose and treat root resorption precisely. There is limited information within the literature on root resorption; therefore, this review aims to understand the clinical and radiographical characteristics of root resorption and evaluate their effect on the accurate diagnosis and management of root resorption in permanent teeth.

Key words: Root Resorption, External Resorption, Inflammatory Resorption, Internal Resorption, Replacement Resorption.

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Introduction

Root resorption is a destructive procedure, resulting in a gradual degradation and eventual loss of dentine and cement in the tooth root. It might be a normal or pathological occurrence. Root resorption in the primary teeth is normal except when it happens early.[1] However, the resorption of roots in primary dentition is a typical physiologic reaction. On the other hand, it has a pathologic origin in the permanent teeth, and the etiology necessitates two phases: injury and stimulus.[2] Injury can occur to the tissue covering the root's exterior surface (precementum) or inner surface (predentin). The injury might be mechanical due to trauma, surgery, or the compression of an impacted tooth.[3] It can also happen as a result of chemical irritation after using hydrogen peroxide to whiten teeth. Multinucleated cells inhabit denuded mineralized tissue and begin the resorption process.[4] However, under normal circumstances, the permanent tooth is surrounded by alveolar bone with very strong osteoblasts and osteoclasts without being affected by either of these cell types. According to many studies, this immunity is due to healthy cementoblast and odontoblast cell layers.[5] Consequently, after differentiation, an osteoclast's role is to resorb hard tissue (i.e., enamel, cementum, dentine). The process of hard tissue breakdown occurs by dissolving inorganic materials with acids, along with the degradation of organic components.[6]

Root resorption is self-limiting until the clastic cells are stimulated further. If the damage is minor, it can be repaired by cementum-like tissue in 2 to 3 weeks. The active resorption process will continue in the presence of a general stimulating factor of the osteoclastic cells, such as inflammation or pressure. Each form of root resorption has a different origin. As a result, the various forms of root resorption should be distinguished according to the factors that stimulate them. It will be feasible to stop the process by eliminating the etiological aspects once these stimulating factors have been discovered.[7]

This review aims to discuss the etiological factors as well as the radiological and clinical features of root resorption to summarize the current strategies of proper diagnosis and evaluate the effectiveness of any interventions to manage root resorption in permanent teeth.

Diagnosis of root resorption

Early detection is the most important and beneficial aspect of root resorption treatment because the faster treatment begins, the less extreme the resorption effects will be. Because the lack of pathognomonic symptoms makes an accurate diagnosis more complicated, a definitive diagnosis can be reached through a thorough clinical and radiological examination.[8] Because teeth with root resorption are usually asymptomatic, the diagnosis may be made by chance during a radiographic examination, and sensitivity testing may be effective for root resorption detection.[3] The most common method for diagnosis is intraoral radiography, and most clinical investigations utilize radiographs of an irregular root surface contour at various angles to establish which surface is involved.[9] However, using an intraoral radiograph with a panoramic radiograph has been suggested to improve assessment in

the anterior areas.[10] Because the limits of standard dental radiography are well known, numerous case studies have shown that cone-beam computed tomography (CBCT) can help diagnose resorptive lesions early in their progression. The precise visualization provided by CBCT leads to better definitive outcomes.[11,12]

Because several tiny resorptive channels stretch inside the dentine and are trapped beneath the root surface, the resorption cannot be adequately examined intraorally; however, a CBCT scan enables observation of these abnormalities, which is a good alternative for histopathological analysis.[12]

CBCT can provide the following information: the position, size, and form of the lesion, the existence of root perforations and apical bone pathology, the thickness of the root dentine, and the location of specific structures such as the inferior alveolar nerve canal, maxillary sinus, and mental foramen. These findings support the differential diagnosis and enable a prognosis evaluation of the tooth if the condition is treatable.[13] CBCT can also detect the root resorption complexity accurately and aid the clinician in determining the precise amount of the resorptive lesion.[14,15]

Classification of root resorption

1. Internal root resorption

It begins along the root canal surface and may cause the neighboring radicular dentine to deteriorate gradually. This entity occurs inside the canal system.[16]

Etiology

The exact etiological and pathogenic processes are yet unknown. Internal resorption may be linked to traumatized/replanted teeth, teeth with pulpotomy and crown preparations, partial pulp removal, caries, pulp capping with calcium hydroxide, intense heat, and a broken tooth. These agents activate the pulp tissue, triggering inflammatory reactions, which lead to the conversion of specific undifferentiated pulp cells into osteoclasts or macrophages, resulting in dentine resorption.[17,18]

Male patients have a higher prevalence than female individuals. The previous literature rarely discussed the prevalence of internal root resorption. Thoma (1935) showed only one case out of 1000 teeth of internal resorption studied.[19] Another study observed internal resorption in eight out of 28 teeth (28%) following coronal pulp amputation and calcium hydroxide capping.[20] 51.5% of this resorption in auto-transplanted maxillary canines was found in another study.[21] In these studies, the diagnosis of resorption was based on 2-dimensional radiography data, which would have underestimated the exact prevalence of internal root resorption.[22]

Internal root resorption has two sub-categories, as described by Sak et al. (2016):2

a. Internal inflammatory resorption

The pulp chamber or root canal enlarges in an ovoid or fusiform shape. The enlargement usually spreads in apical and lateral directions, as shown in Figure 1 (a). The pulp may be inflamed on a long-term basis. Although persistent inflammation is widespread in pulpal infections, it does not create the conditions for root canal inflammatory resorption to occur.[22]

Figure 1a: Internal inflammatory resorption.

b. Internal replacement root resorption

It is an uncommon type, and the canal space may increase unevenly. This root resorption shows metaplastic changes, and there are diffuse zones of heterogeneous radiopacities and radiolucencies, as shown in Figure 1 (b). It may result in obliterating the canal by cancellous-like bone. The exact cause for this occurrence is unknown. Dental pulp stem cells may produce the osteoid material in reaction to trauma, inflammation, or infection.[22]

Figure 1b: Internal replacement root resorption with metaplasia.[22]

Figure 2: Pink/red spot in the cervical area of a maxillary central incisor with internal resorption.[23]

Clinical features

Clinical features of internal inflammatory and internal replacement resorption are similar. Internal root resorption has different clinical features depending on its development and where it occurs. Most teeth with this resorption have no symptoms; however, when the resorption is actively proceeding, the tooth is at least partly vital and may present with pulpitis-like symptoms. In established internal resorption cases, the pulpal tissue may become necrotic and persistently infected, causing signs and symptoms of acute or chronic apical periodontitis. A pink or reddish discoloration visible through the crown of the concerned tooth may be produced by severe resorption of the coronal pulp, as shown in Figure 2.[23,24]

Treatment

Following the diagnosis of internal root resorption, the dentist must determine if the tooth is restorable. Root canal therapy is necessary if the tooth is judged restorable and has a good prognosis. Instrumentation and disinfecting the root canal with internal resorption presents a few unique problems not found in standard endodontic therapy. Because internal resorption lesions are inaccessible to chemomechanical debridement, activation of irrigants by ultrasonic after the preparation of the root canal system should be considered a necessary step in the cleaning and disinfection of the internal resorption defects to enhance the removal of necrotic tissue and biofilms from inaccessible parts of the root canal when used to agitate the irrigation solution (3% NaOCl, 17% EDTA,) and normal saline used as the final irrigation. An intracanal antimicrobial medicament should be employed to increase the disinfection of root resorption defects. Calcium hydroxide has been used as a dressing between appointments to eliminate microorganisms successfully that persist following chemomechanical instrumentation.[25,26]

Thermoplasticized techniques are recommended to enhance the seal of the internal resorption defect. If the resorption defect has perforated the root canal wall, bioactive hydraulic calcium silicate materials such as Biodentine or Mineral trioxide aggregate (MTA) must be used to repair the resorptive perforation defects.[27] Therefore, combining bioactive materials with thermoplasticized gutta-percha obturation will provide three-dimensional obturation and promote remineralization and healing.[28,29]

Recently, perforated internal root resorption has been treated using regenerative endodontic treatment. First, the entire root canal is debrided, followed by intracanal medicament such as calcium hydroxide for three months before MTA placement over the blood clot. This treatment method had shown encouraging results, as evidenced by the development of hard tissue in the perforation area and an increase in the thickness of the root canal wall after

2–3 years.[29,30] There is little information regarding the long-term result of this resorption and its management because of various treatment techniques that may result from the very few internal root resorption cases described in the literature.[31] Long-term clinical studies with good designs are necessary to evaluate the medium- to long-term results of internal resorption.

2. External root resorption

a. External inflammatory root resorption

Etiology

It is a resorptive defect that develops after a trauma, periodontal disease, or orthodontic therapy that causes an inflammatory reaction inside the periodontal ligament. This type of resorption is not a self-limited process and may result in massive damage.[32]

Clinical and radiographic features

The tooth is usually non-vital and may be either symptomatic or asymptomatic. It is characterized by abnormal mobility and the existence of a sinus tract. The tooth may be tender to percussion and palpation.[32] External inflammatory resorption is widespread in teeth with diseased necrotic root canal systems, and traumatic dental accidents do not necessarily cause it.[33] The usual radiographic appearance of these defects is scooping out radiolucency with an adjacent radiolucency in bone. In the resorption region, there is a total loss of the lamina dura, as shown in Figure 3 (a).[34,35]

Treatment

For teeth with extrinsic inflammatory resorption, the interceptive management strategy is to obtain access to the root canal if appropriate for treatment, explore the root canal, determine the working length, and do chemomechanical cleaning, then thoroughly irrigate and dry the canal. A Corticosteroid antibiotic paste, such as Ledermix paste, must be used as a root canal dressing and replace the Corticosteroid antibiotic paste intracanal dressing after six weeks. After that, obtain periapical radiography after another six weeks, and if the inflammatory resorption has not advanced, use a 50 - 50 combination of corticosteroid antibiotic paste and Ca (OH)2 to make a fresh root canal dressing. Then, obtain periapical radiography after three months and apply a Ca (OH)2 root canal dressing to stimulate hard tissue healing if the inflammatory resorption has not proceeded. The Ca (OH)2 root canal dressing must be replaced every three months till the resorptive lesion's hard tissue healing is visible in the periapical radiography. Once a hard tissue has healed, apply the gutta-percha and cement as root canal filling. Plan a six-month assessment, followed by a review for at least five years.[35]

If gutta-percha is utilized when this resorption occurs in the apex, extreme caution is necessary to ensure a proper cone fit because accidental over-extension of the root filling is likely. Root filling with calcium silicate bioactive materials may be advantageous due to their excellent biocompatibility, sealing ability, the potential to repair the periodontal ligament and cementum, the ability to improve the conditions for hard tissue repair, and the suppression of clastic activity.[16,36] However, Long-term evidence on this therapy approach is inadequate.

b. External replacement resorption or ankylosis external replacement resorption.

Etiology

The root substance is eventually replaced by bone, resulting in a direct connection between bone and root tissue. The most common reason is a traumatic injury, including severe luxation, such as lateral luxation, intrusion, and avulsed tooth replantation. In cases of the avulsion, replacement resorption was more frequently observed (87.2%), followed by intrusive luxation (57.1%), according to Soares et al. (2015) retrospective study.[37]

Clinical and radiographic features

On percussion, the tooth looks to be very hard in its socket, producing a high metallic sound. There is no evidence of tooth mobility, and the tooth is asymptomatic. The resorption area is filled with bone, and the periodontal ligament spaces disappear. So, there is no radiolucency, and the whole root may be substituted by bone, as shown in Figure 3 (b).[38,3]

Treatment

Replacement resorption, which tends to be persistent until the root is substituted by bone, currently has no therapy available. The resorptive process is delayed (but not prevented) in teeth when immersed in fluoride before replantation.[39]

A case series showed the use of regenerative endodontic treatment to manage three external replacement resorption cases. As a scaffold to encourage stem cell differentiation, they selected platelet-rich fibrin rather than an artificial blood clot, and then they added Biodentine on top of the scaffold. The resorption was arrested and, in some cases, reversed after three years of follow-up.[40]

c. Cervical root resorption

Etiology

The etiology is incompletely understood, the conjunction of damage to the periodontal ligament and the cementum, as well as other factors, has been suggested as a cause of cervical root resorption start. The most commonly reported risk factors were orthodontic treatments and a history of injury. The teeth most typically involved are the maxillary incisors, canine, first molar, and mandibular first molar.[41] It was found that when 35 percent hydrogen peroxide was utilized for whitening methods, these resorptions were detected in 6% - 8% of instances and 18% - 25% of patients whenever the hydrogen peroxide was thermally activated.[42] When 30 % hydrogen peroxide was placed in the pulp chamber of teeth that lost part of the cementum layer, 82 percent of the bleaching agent diffused to the periodontal tissues around the root.[43,44]

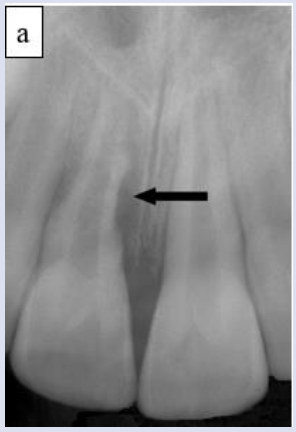

Figure 3a:External Inflammatory resorption.[35]

Figure 3b: Ankylosis external replacement resorption.[3]

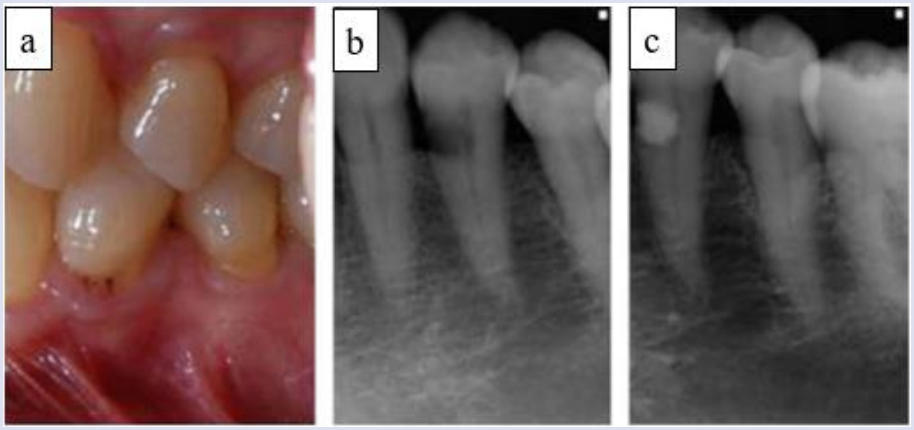

Figure 4: a) Cervical root resorption on the lower left first premolar. b) A radiolucent defect having ill-defined edges. c) After the cervical root resorption cavity was repaired using glass ionomer cement.[45]

Clinical and radiographic features

The symptoms of cervical root resorption vary widely and are influenced by many factors, including the position and the stage of advancement. Until there is a superimposed infection late in the resorptive procedure, the afflicted tooth will usually stay asymptomatic unless there are pulpal or periodontal complaints. Teeth without a pulp infection typically respond well to vitality tests. There is a lot of spontaneous and copious bleeding on probing, and around the resorptive cavity, there is the sharp, thinned-out margin. It has a wide range of radiographic appearances, which are determined by the size and type of the lesions. It usually shows an uneven, asymmetrical radiolucent area with a discernible root canal outline, as shown in Figure 4.[45,46]

Treatment

Treatment depends on the intensity of the lesion, its location and if it is perforated to the root canals, and the tooth's restorability. Many therapy regimens have been recommended depending on the form of the resorption defect. Therapy includes removing all resorptive tissues and filling the resultant defect with a tooth-colored plastic restoration. In situations when the cervical root resorption defect has perforated the root canal system, endodontic therapy may be necessary. Internal repair is suggested when the resorption is near or has already perforated the root canal, and a surgical approach is not feasible because of improper accessibility or if surgical access will result in the removal of an excessive amount of sound tooth structure. After endodontic therapy, the access cavity is restored. Under a dental microscope, long shank burs and ultrasonic tips help to eliminate the resorptive lesion. Then, Biodentine may be used to restore resorbed dentine, and its high pH may aid in stopping the osteoclastic action of any remaining osteoclast.[47,48]

Conclusions

Early discovery of root resorption and accurate documentation of the patient's history are essential steps for successful management, prognosis, outcome of root resorption, and treatment at the appropriate time, which will prevent tooth loss. With the current advancement of sophisticated imaging methods, such as cone-beam computed tomography, which is an effective screening method for confirming the existence of root resorption, and bioceramic-based endodontic materials, which allows for the extension of the limits for tooth conservation, the treatment of root resorption has become more predictable and successful.

Clinical studies are necessary to understand the etiology and pathogenesis of the various root resorption types. In addition, a deeper understanding of this area is essential since root resorption diagnosis and management can be difficult for clinicians and result in misdiagnosis.

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None

Conflicts of Interest

None

References

1. Harokopakis-Hajishengallis E. Physiologic root resorption in primary teeth: molecular and histological events. J Oral Sci 2007;49:1-2.
2. Sak M, Radecka M, Karpiński TM, Wędrychowicz-Welman A, Szkaradkiewicz AK. Tooth root resorption: etiopathogenesis and classification. MicroMedicine 2016;4:21-31.
3. Fuss Z, Tsesis I, Lin S. Root resorption–diagnosis, classification and treatment choices based on stimulation factors. Dent Traumatol 2003;19:175-82.
4. MARINESCU IR, BĂNICĂ AC, MERCUŢ V, GHEORGHE AG, DRĂGHICI EC, COJOCARU MO, SCRIECIU M, POPESCU SM. Root resorption diagnostic: Role of digital panoramic radiography. Curr Health Sci J 2019;45:156.
5. Trope M. Root resorption of dental and traumatic origin: classification based on etiology. Pract Periodontics Aesthet Dent 1998;10:515-522.
6. Andreasen JO. Experimental dental traumatology: development of a model for external root resorption. Dent Traumatol 1987;3:269-287.
7. Aidos H, Diogo P, Santos JM. Root resorption classifications: a narrative review and a clinical aid proposal for routine assessment. Eur Endod J 2018;3:134.
8. Da Silveira HL, Silveira HE, Liedke GS, Lermen CA, Dos Santos RB, De Figueiredo JA. Diagnostic ability of computed tomography to evaluate external root resorption in vitro. Dentomaxillofac Radiol 2007;36:393-396.
9. Bergmans L, Van Cleynenbreugel J, Verbeken E, Wevers M, Van Meerbeek B, Lambrechts P. Cervical external root resorption in vital teeth: X-ray microfocus-tomographical and histopathological case study. J Clin Periodontol 2002;29:580-585.
10. Laux M, Abbott PV, Pajarola G, Nair PN. Apical inflammatory root resorption: a correlative radiographic and histological assessment. Int Endod J 2000;33:483-493.
11. Patel S, Dawood A, Ford TP, Whaites E. The potential applications of cone beam computed tomography in the management of endodontic problems. Int Endod J 2007;40:818-830.
12. Patel K, Mannocci F, Patel S. The assessment and management of external cervical resorption with periapical radiographs and cone-beam computed tomography: a clinical study. J Endod 2016;42:1435-1440.
13. Nilsson E, Bonte E, Bayet F, Lasfargues JJ. Management of internal root resorption on permanent teeth. Int J Dent 2013;2013.
14. Liu DG, Zhang WL, Zhang ZY, Wu YT, Ma XC. Localization of impacted maxillary canines and observation of adjacent incisor resorption with cone-beam computed tomography. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;105:91-98.
15. Jaju PP, Jaju SP. Clinical utility of dental cone-beam computed tomography: current perspectives. Clin Cosmet Investig Dent 2014;6:29.
16. Patel S, Saberi N, Pimental T, Teng PH. Present status and future directions: Root resorption. Int Endod J 2022;55:892-921.
17. Darcey J, Qualtrough A. Resorption: part 1. Pathology, classification and aetiology. Br Dent J 2013;214:439-51.
18. Umashetty G, Hoshing U, Patil S, Ajgaonkar N. Management of inflammatory internal root resorption with Biodentine and thermoplasticised Gutta-Percha. Case Rep Dent 2015;2015.
19. Thoma KH. Central osteoclastic resorption of dentine and complete repair with osteo-dentine in the permanent tooth of an adult. Dent Items Interest 1935;57:28.
20. Cabrini RL, Maisto OA, Manfredi EE. Internal resorption of dentine: histopathologic control of eight cases after pulp amputation and capping with calcium hydroxide. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1957;10:90-96.
21. Ahlberg K, Bystedt H, Eliasson S, Odenrick L. Long-term evaluation of autotransplanted maxillary canines with completed root formation. ACTA ODONTOL 1983;41:23-31.

22. Patel S, Ricucci D, Durak C, Tay F. Internal root resorption: a review. J Endod 2010;36:1107-1121.

23. Haapasalo M, Endal U. Internal inflammatory root resorption: the unknown resorption of the tooth. Endod Topics 2006;14:60-79.

24. Lyroudia KM, Dourou VI, Pantelidou OC, Labrianidis T, Pitas IK. Internal root resorption studied by radiography, stereomicroscope, scanning electron microscope and computerized 3D reconstructive method. Dent Traumatol 2002;18:148-152.

25. Adham AH, Ali AH, Mannocci F. Continuous Chelation Concept in Endodontics. J Baghdad Coll Dent 2022;34:59-69.

26. Siqueira Jr JF, Rôças IN, Santos SR, Lima KC, Magalhães FA, de Uzeda M. Efficacy of instrumentation techniques and irrigation regimens in reducing the bacterial population within root canals. J Endod 2002;28:181-184.

27. Priyalakshmi S, Ranjan M. Review on Biodentine-a bioactive dentin substitute. J Dent Med Sci 2014;13:51-57.

28. Ali A, Banerjee A, Mannocci F. Effect of adhesive materials on shear bond strength of a mineral trioxide aggregate. Am J Dent 2016;29:46-50.

29. Arnold M. Reparative endodontic treatment of a perforating internal inflammatory root resorption: a case report. J Endod 2021;47:146-155.

30. Kaval ME, Güneri P, Çalışkan MK. Regenerative endodontic treatment of perforated internal root resorption: a case report. Int Endod J 2018;51:128-137.

31. Haapasalo M, Endal U. Internal inflammatory root resorption: the unknown resorption of the tooth. Endod Topics 2006;14:60-79.

32. Nagne M, Ikhar A. A Review on Internal Resorption. J Pharm Res Int 2021;33:3458-3465.

33. Patel S, Dawood A, Whaites E, Pitt Ford T. New dimensions in endodontic imaging: part 1. Conventional and alternative radiographic systems. Int Endod J 2009;42:447-462.

34. Bhuva B, Barnes JJ, Patel S. The use of limited cone beam computed tomography in the diagnosis and management of a case of perforating internal root resorption. Int Endod J 2011;44:777-786.

35. Abbott PV. Prevention and management of external inflammatory resorption following trauma to teeth. Aust Dent J 2016;61:82-94.

36. Alhashimi R. Effects of heating on the physical properties of bio ceramic root canal sealer (in vitro comparative). J Popul 2023;30:1-8.

37. Soares AJ, Souza GA, Pereira AC, Vargas-Neto J, Zaia AA, Silva EJ. Frequency of root resorption following trauma to permanent teeth. J Oral Sci 2015;57:73-78.

38. Andreasen JO, Borum MK, Jacobsen HL, Andreasen FM. Replantation of 400 avulsed permanent incisors. 4. Factors related to periodontal ligament healing. Dent Traumatol 1995;11:76-89.

39. Trope M. Clinical management of the avulsed tooth: present strategies and future directions. Dent Traumatol 2002;18:1-1.

40. Yoshpe M, Einy S, Ruparel N, Lin S, Kaufman AY. Regenerative endodontics: a potential solution for external root resorption (case series). J Endod 2020;46:192-199.

41. Patel S, Mavridou AM, Lambrechts P, Saberi N. External cervical resorption-part 1: histopathology, distribution and presentation. Int Endod J 2018;51:1205-1223.

42. Cvek M, Lindvall AM. External root resorption following bleaching of pulpless teeth with oxygen peroxide. Dent Traumatol 1985;1:56-60.

43. Rotstein I, Torek Y, Misgav R. Effect of cementum defects on radicular penetration of 30% H2O2 during intracoronal bleaching. J Endod 1991;17:230-233.

44. Irinakis E, Aleksejuniene J, Shen Y, Haapasalo M. External cervical resorption: A retrospective case-control study. J Endod 2020;46:1420-1427.

45. Patel S, Kanagasingam S, Ford TP. External cervical resorption: a review. J Endod 2009;35:616-25.

46. Patel S, Foschi F, Mannocci F, Patel K. External cervical resorption: a three-dimensional classification. Int Endod J 2018;51:206-214.

47. Patel S, Foschi F, Condon R, Pimentel T, Bhuva B. External cervical resorption: part 2–management. Int Endod J 2018;51:1224-1238.

48. Al-Hashimi RA, Habeeb MA, Al-Kinani AA. A Comparative Evaluation of Apical Seal Associated with Ultrasonic Retrograde Cavities Filled with Bioactive Material (In Vitro Study). J Baghdad Coll Dent 2016;28:15-21.