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Review Article

Tooth Resorption: A Comprehensive Review of Pathophysiology, Classification, Diagnosis and Management

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ABSTRACT

Tooth resorption refers to the loss of dental hard tissues, including dentin, cementum, or bone, due to physiological or pathological processes. Although initially observed in the 16th century, its etiologies, classifications, and treatments have been significantly elucidated in recent years. This review provides a detailed exploration of the biological basis, types, histopathological features, and current clinical management strategies for tooth resorption. Both internal and external forms are examined, with emphasis on diagnostic features and therapeutic approaches. Early recognition and intervention remain pivotal to prevent irreversible damage and tooth loss.

Keywords: Ankylosis, External Resorption, Internal Resorption, Odontoclasts, Orthodontic Resorption, Inflammatory Root Resorption.

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INTRODUCTION

Tooth resorption involves the progressive loss of dental hard tissues-dentin, cementum, or alveolar bone-due to physiological or pathological processes. While bone remodels continuously, permanent teeth typically resist resorption unless subjected to abnormal stimuli such as trauma, infection, or mechanical stress.[1,2]

The process is mediated by odontoclasts, multinucleated cells derived from hematopoietic precursors, which resemble osteoclasts in their ability to degrade mineralized tissues. These cells become active when protective layers like the pre-cementum or odontoblast layer are compromised. Pro-inflammatory cytokines, enzymes, and growth factors in the local environment modulate their resorptive activity.[3,4]

Tooth resorption can be physiological, as seen in the shedding of primary teeth, or pathological, affecting permanent teeth. Pathological types are classified as internal or external. Internal resorption originates within the pulp, often due to trauma or chronic inflammation. External resorption is more prevalent and includes surface, inflammatory, replacement (ankylosis), and cervical invasive types. It may arise from infections, orthodontic forces, trauma, or pressure from impacted teeth or cysts. Early resorption is typically asymptomatic and detected incidentally via radiographs. Clinical signs may include a pinkish discoloration in cervical resorption. Advanced imaging, particularly Cone-Beam Computed Tomography (CBCT), enhances diagnostic accuracy.[5,6]

Management depends on the resorption type and severity. Root canal therapy is effective for internal and inflammatory resorptions, while surgical repair may be needed for cervical invasive cases. Replacement resorption is generally irreversible.[6]

Preventive strategies include managing trauma, minimizing excessive orthodontic forces, and addressing infections early. Understanding the cellular and molecular basis of resorption is crucial for timely diagnosis and effective treatment planning, ultimately aiding in the preservation of dental structures.

Historical Overview of Root Resorption

Root resorption, defined as the pathological loss of dentin and/or cementum, has been recognized since the 16th century, with early mention in the Artzney Buchlein. Bell described internal resorption in 1830, and Fothergill introduced the "pink spot" sign in 1900. Mummery later provided a detailed anatomical study in 1920.[5]

Ketcham (1929) reported apical root resorption in orthodontic patients, and Munch (1937) emphasized radiography's diagnostic role. Rudolph (1940) linked extended orthodontic treatment to increased resorption risk, while Steadman (1942) noted higher

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susceptibility in root-filled teeth. Henry and Weinnman (1951) suggested uncalcified cementum resists resorption, and Weiss (1969) found no significant difference in resorption between vital and non-vital teeth under force.[5-7]

Andreasen's 1985 classification remains foundational, distinguishing internal and external resorption types. Harrington and Natkin (1979) linked cervical resorption to internal bleaching, supported by Heithersay (1999), who associated it with bleaching, trauma, and orthodontics . Cvek *et al*., (1991) demonstrated effective healing using calcium hydroxide in luxated teeth.[7]

Later, Trope and Friedman highlighted the superiority of HBSS and Viaspan in preserving avulsed teeth. Linskog and Hammarström (1988) detailed morphological changes in dentinoclasts during resorption.[7,8]

Pathophysiology and Cellular Mechanisms

Tooth and bone resorption involve the breakdown of mineralized tissues-bone, dentin, and cementum-by specialized clastic cells. While the tissues differ, the resorptive mechanisms are fundamentally similar. The key cells include osteoclasts (bone), odontoclasts/dentinoclasts (dentin), and cementoclasts (cementum), all derived from the monocyte/macrophage lineage.[9,10]

Osteoclasts are the most extensively studied and are essential in bone remodeling and pathological resorption. They form via fusion of mononuclear precursors and resorb bone by creating an isolated compartment where they secrete hydrogen ions and enzymes like cathepsin K and Tartrate-Resistant Acid Phosphatase (TRAP), leading to mineral dissolution and collagen degradation. The resulting resorption lacunae are known as Howship's lacunae.[10-12]

Odontoclasts and dentinoclasts, typically smaller with fewer nuclei, operate similarly in deciduous tooth exfoliation and pathological root resorption. They exhibit high acid phosphatase activity and require both hydroxyapatite and collagen to form a functional ruffled border.[12-14]

Cementoclasts, less frequently observed, are often mononuclear and appear in response to trauma or disease, resorbing cementum from lacunae on root surfaces. In inflammatory conditions like periodontitis, monocytes may transform into osteoclast-like cells. RANKL–OPG signalling from osteoblasts and osteocytes regulates this process. Disruption of protective layers such as intermediate cementum exposes dentinal tubules to microbial toxins, exacerbating resorption.[13-16]

Classification of Tooth Resorption

Tooth resorption is broadly classified into internal and external types:[30]

- Internal Resorption: Originates within the pulp, leading to progressive loss of dentin. Subtypes include internal inflammatory and internal replacement resorption.
- External Resorption: Initiates from the periodontal ligament or surrounding alveolar bone. Types include:

- **Surface resorption:** Self-limiting and usually heals spontaneously.
- **Inflammatory resorption:** Due to infection or mechanical trauma.
- **Replacement resorption (ankylosis):** Bone replaces the root surface.
- **Apical resorption:** Common in apical periodontitis or following orthodontic therapy.
- **Lateral resorption:** Affects the mid-root surface.
- **Invasive Cervical Resorption (ICR):** Begins below the epithelial attachment.
- **Extra-canal Invasive Resorption (EIR):** Invades dentin from the external root surface without involving the pulp.

Histopathology of Tooth Resorption

Tooth resorption involves the breakdown of cementum, dentin, and occasionally alveolar bone, and is microscopically characterized by scalloped depressions known as Howship's lacunae, where multinucleated clastic cells-osteoclasts or dentinoclasts-are found. These cells attach to mineralized surfaces via a clear zone and ruffled border, forming a sealed acidic microenvironment that dissolves the inorganic matrix. The exposed organic matrix is subsequently degraded by enzymes such as acid phosphatase and cathepsin B.[17-19]

Cementum resorption appears as bay-like concavities with adjacent clastic cells. In some cases, the process may extend into dentin or pulp tissue, though it often remains asymptomatic. Repair of cementum involves new cementum deposition, identified by reversal lines. Successful repair requires viable connective tissue, while epithelial proliferation may hinder the regenerative process.[18-20]

Various factors contribute to the initiation and progression of resorption, including enzymes (e.g., collagenase, acid hydrolases, carbonic anhydrase), hormones (e.g., parathyroid hormone, vitamin D), and local inflammatory mediators such as prostaglandins and bacterial endotoxins. Prostaglandins enhance osteoclastic activity by increasing calcium influx, while endotoxins stimulate resorption under inflammatory conditions. Understanding the cellular and molecular basis of tooth resorption is essential for early diagnosis, targeted intervention, and effective management of resorptive dental conditions.[21-23]

Internal Root Resorption

Internal root resorption is a progressive, often asymptomatic loss of dentin originating within the pulp chamber or root canal. It is typically associated with chronic pulp inflammation or trauma but may also occur idiopathically. Initiated by injury to the pulp, the condition involves multinucleated dentinoclasts that resorb dentin, often replacing pulp tissue with granulation tissue. Common triggers include trauma, caries, periodontal infection, orthodontic forces, bruxism, dental procedures, and systemic or viral conditions (e.g., herpes zoster). The process requires at least partially vital pulp to persist and frequently goes unnoticed until detected radiographically.[24-26]

Histologically, resorption is marked by dentinoclastic activity and inflammatory granulation tissue. Radiographically, it appears as a well-defined, symmetrical radiolucency aligned with the root canal. Clinically, a "pink tooth" may be observed if granulation tissue shines through the enamel.[23-25]

Prompt endodontic therapy is essential. Treatment includes pulpectomy, irrigation with sodium hypochlorite, calcium hydroxide dressing, and obturation with thermoplastic gutta-percha. In cases of perforation, repair with biocompatible materials or surgical management-such as curettage, root resection, or intentional replantation-may be required. Early diagnosis and intervention are critical to halt progression, preserve tooth structure, and prevent complications such as perforation, infection, or tooth loss.[14-16]

External Root Resorption (ERR)

External root resorption is a pathological process originating in the periodontium, involving the progressive destruction of the root surface. It may affect apical, lateral, or cervical regions, often leading to significant structural loss and eventual tooth loss if untreated. The condition arises when protective barriers like cementum are compromised, allowing communication between bacterial toxins and pulp or periodontal tissues, triggering clastic cell activity.[20,23,26]

ERR is classified by Andreasen and Hjorting-Hansen into surface, inflammatory, and replacement (ankylosis) types. Frank's classification adds anatomical subtypes like apical, lateral, and cervical resorption. Etiologies include trauma, orthodontic forces, bleaching, periodontal procedures, systemic conditions (e.g., hyperparathyroidism), and idiopathic causes.[20]

- Surface Resorption is self-limiting and resolves with cementum repair.
- Inflammatory Resorption is driven by infection or irritation, often after trauma or necrotic pulp, and progresses rapidly.
- Replacement Resorption results in ankylosis, where bone replaces the tooth root.

Apical ERR is linked to apical periodontitis or over-instrumentation. Radiographically, it shows irregular root apex loss; treatment focuses on endodontic disinfection and sealing.[19] Lateral ERR occurs from trauma or concussions, with radiographic "moth-eaten" defects; management includes antibiotics and timely root canal therapy.Invasive Cervical Resorption (ICR) begins near the CEJ and progresses internally; often asymptomatic, it may present as a pink spot. Treatment involves surgical debridement and restoration.[20] Extra-Canal Invasive Resorption (EIR) affects dentin surrounding the canal without pulpal involvement and may require surgical or replantation approaches.Dentoalveolar Ankylosis, a severe sequela of trauma, leads to direct bone-to-root fusion and eventual tooth replacement by bone.[15,20,27]

Early diagnosis using radiographs, CBCT, and pulp testing is critical. Management depends on the type and extent of resorption, aiming to eliminate causative factors and preserve the tooth.

Dentoalveolar Ankylosis and Orthodontic Root Resorption

Dentoalveolar ankylosis refers to the pathological fusion of alveolar bone directly to the root surface of a tooth, resulting in the loss of the Periodontal Ligament (PDL). It occurs when the protective cementum layer is damaged, allowing osteoblasts to deposit bone directly onto the root dentin.[28,29] This initiates replacement resorption, a process by which root structure is gradually replaced by bone. Ankylosis commonly follows severe traumatic injuries such as avulsion, intrusive luxation, or prolonged extraoral dry time during replantation, which leads to necrosis of PDL cells over large root surface areas.[30,31]

Clinically, ankylosed teeth present with lack of mobility and a characteristic high-pitched metallic sound upon percussion. Over time, especially in growing patients, they become infraoccluded due to the continued eruption of adjacent teeth.[32,33] Radiographically, the diagnosis can be challenging but is marked by loss of PDL space, irregular root contours, and direct continuity between root and bone. Histologically, ankylosis demonstrates direct bone-to-root contact with osteoclasts resorbing dentin and osteoblasts depositing bone in a remodeling-like process.[34,35]

No treatment currently exists to reverse replacement resorption. Preventive strategies focus on minimizing PDL injury-particularly by reducing extraoral dry time, using biologically compatible storage media (e.g., HBSS), and timely replantation. Root canal therapy can prevent inflammatory resorption but does not halt the progression of ankylosis. Management is therefore palliative, aiming to retain the tooth as long as possible.[35-38,39]

Orthodontic Root Resorption (ORR) is a common iatrogenic effect of orthodontic treatment, involving progressive loss of root structure due to mechanical forces. The biological response

begins with PDL compression, leading to disruption of cementum and precementum. This damage exposes the underlying dentin, allowing multinucleated clastic cells to initiate resorption, particularly at the apex.[40-43]

The etiology of ORR is multifactorial, involving host, local, and treatment-related factors. Host factors include genetic predisposition, age, sex, and systemic conditions. Genetic studies indicate a heritable component, with some individuals more susceptible to External Apical Root Resorption (EARR). While adults may exhibit greater vulnerability due to denser bone and thicker cementum, age-related differences in resorption are not always significant. Gender influence is inconsistent across studies.[20,23,27,28]

Local factors such as tooth type and root morphology also affect susceptibility. Maxillary incisors are most commonly involved due to their root shape and position, which concentrates forces at the apex.[31,32] Teeth with short, blunt, or pipette-shaped roots are especially prone to resorption. Parafunctional habits like tongue thrusting or nail biting may further exacerbate risk.[30,32,33,37,44]

Treatment-related factors include force magnitude, direction, duration, appliance type, and root proximity to cortical bone. Prolonged treatment time and excessive or continuous forces increase the likelihood of root resorption.[44]

Prevention and management of ORR involve applying light, intermittent forces, limiting treatment duration, and conducting periodic radiographic monitoring. Early identification of high-risk individuals enables tailored treatment plans to minimize damage while achieving orthodontic objectives.[41,42]

Understanding the complex mechanisms of ankylosis and orthodontic resorption is essential for preserving dental structures and ensuring long-term treatment success.

CONCLUSION

In conclusion, tooth resorption is a complex and multifactorial process involving both physiological and pathological mechanisms that can significantly impact dental health. Its unpredictable nature and varied presentations-ranging from internal to external types pose diagnostic and therapeutic challenges.

Understanding the biological interplay between resorptive cells and hard tissue is crucial for effective management. Early detection, appropriate endodontic intervention, and preventive strategies following trauma are essential to limit progression and preserve tooth structure. Continued research, especially into genetic and mechanical factors, holds promise for improved prevention and treatment approaches, ultimately enhancing patient outcomes in managing root resorption.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

CBCT: Cone beam computed tomography; **CEJ:** Cemento enamel Junction; **ERR:** External Root resorption; **EIR:** External invasive resorption; **ICR:** Invasive cervical resorption; **ORR:** Orthodontic root resorption; **PDL:** Periodontal ligament.

REFERENCES

1. Fuss Z, Tsesis I, Lin S. Root resorption: Diagnosis, classification and treatment choices based on stimulation factors. Dent Traumatol. 2003; 19(4): 175-82.
2. Haapasalo M, Endal U, Zandi H, Coil J. Eradication of endodontic infection by instrumentation and irrigation solutions. Endod Topics. 2005; 10(3): 77-89.
3. Lindskog AB, Bott PV, Pajarola G, Nair PN. Apical inflammatory root resorption: a correlative radiographic and histological assessment. Int Endod J. 2000 ;33(6): 483-93.
4. Lopatiene K, Dumbravaite A. Risk factors of root resorption after orthodontic treatment. Baltic Dent Maxillofac J. 2008; 10: 89-95.
5. Somerlott GS, Lawrence C, Walsh W. Differential diagnosis of internal and external root resorption. J Endod. 1976 ;11(7): 329-34.
6. Shearwood R, Steadman MS. Resume of the literature on root resorption. Angle Soc Orthod. 1941; 28: 28-38.
7. Andreasen JO. External root resorption: its implications in dental traumatology, paedodontics, periodontics, orthodontics and endodontics. Int Endod J. 1985 ;8(3): 109-18.
8. Moskow BS. Periodontal manifestations of hyperoxaluria and oxalosis. J Clin Periodontol. 1989; 60(5): 271-8.
9. Heithersay GS. Clinical, radiographic, and histopathological features of invasive cervical resorption. Quintessence Int. 1999 ;30(1): 27-37.
10. Vier FV, Figueiredo JA. Internal apical resorption and its correlation with the type of apical lesion. Int Endod J. 2004 ;37(9): 730-7.
11. Barclay CW. Root resorption: Aetiology, classification and clinical management. Dent Update. 1993; 20(6): 248-52.
12. Garg N, Garg A. Tooth resorption. In: Textbook of Endodontics. 2nd ed. Elsevier; 2009; 118-32.
13. Wesselink PR. Tooth resorption. In: Gulabivala Textbook of Endodontics. 3rd ed. Elsevier; 2004; 261-8.
14. Andreasen JO, Bakland LK. Pathologic tooth resorption. In: Ingle's Endodontics. 6th ed. Elsevier; 2008; 1358-82.
15. Levin L, Trope M. Root resorption. In: Seltzer and Bender's Dental Pulp. 2nd ed. Quintessence; 2002; 425-48.
16. Trope M, Blanco L, Chivian N. Role of endodontics after dental traumatic injuries. In: Cohen's Pathways of the Pulp. 9th ed. Elsevier; 2006; 610-49.
17. Harokopakis HE. Physiologic root resorption in primary teeth: molecular and histological events. J Oral Sci. 2007; 49(1): 1-12.
18. Patel S, Dent MC, Ricucci D, Durak C, Tay F. Internal root resorption: A review. J Endod. 2010; 36(7): 1107-21.
19. Blayney JR. Problem solving the diagnosis, identification, and management of tooth resorption. In: Guttmann Problem Solving in Endodontics. 4th ed. Elsevier; 2006; 311-36.
20. Andreasen JO. External root resorption: its implication in dental traumatology, paedodontics, periodontics, orthodontics and endodontics. Int Endod J. 1985; 18(3): 109-18.
21. Levander E, Malmgren O, Bajka R. Early radiographic diagnosis of apical root resorption during orthodontic treatment: a study of maxillary incisors. Eur J Orthod. 1998; 20(1): 57-63.
22. Marques LS, Júnior AM, Jorge MLR, Paiva SM. Root resorption in orthodontics: An evidence-based approach. In: Textbook of Orthodontics – Basic Aspects and Clinical Considerations. 4th ed. Elsevier; 2002; 430-46.
23. Chan E, Darendeliler MA. Physical properties of root cementum: part 7. Extent of root resorption under areas of compression and tension. Am J Orthod Dentofacial Orthop. 2006; 129(4): 504-10.
24. Harris EF, Baker WC. Loss of root length and crestal bone height before and during treatment in adolescent and adult orthodontic patients. Am J Orthod Dentofacial Orthop. 2006; 98(5): 463-9.
25. Mavreas D, Athanasiou AE. Factors affecting the duration of orthodontic treatment: a systematic review. Eur J Orthod. 2008; 30(4): 386-95.
26. Tronstad L. Root resorption: etiology, terminology and clinical manifestations. Dent Traumatol. 1988; 4(4): 241-52.
27. Weine. Traumatic injuries. In: Weine Endodontic Therapy. 5th ed. Mosby; 1996; 640-75.
28. Santos BODA, Mendonça DSD, Sousa DL, Neto JJSM. Root resorption after dental traumas: classification and clinical, radiographic and histologic aspects: A literature review. RSBO. 2011; 8(4): 439-45.

29. Finucane D, Kinirons MJ. External inflammatory and replacement resorption of luxated and avulsed replanted permanent incisors: a review and case presentation. Dent Traumatol. 2003; 19(4): 170-4.
30. Heithersay GS. Management of tooth resorption. Aust Dent J. 2007; 52(3): 105-21.
31. Patel S, Kanagasingam S, Pitt Ford T. External cervical resorption: A review. J Endod. 2009; 35(5): 616-25.
32. Anderson L, Blomlof L, Lindskog S, Feiglin B, Hammarstrom L. Tooth ankylosis: clinical, radiographic and histological assessments. Int J Oral Surg. 1984; 13(5): 423-31.
33. Tronstad L. Root resorption: etiology, terminology and clinical manifestations. Dent Traumatol. 1988; 4(4): 241-52.
34. White SC, Pharaoh MJ. Systemic diseases manifested in jaws. In: White and Pharaoh Principles and Interpretation Oral Radiology. 4th ed. Mosby; 2000; 472-92.
35. Nikhil V, Arora V, Padmnabh JHA, Verma M. Non-surgical management of trauma induced external root resorption at two different sites in a single tooth with Biodentine: A case report. J Endodontol. 2005; 150-5.
36. Herrera H, Leonardo MR, Silva FWD, Silva LAB, Salvadar S, Salvador EJ, Ribeiroa. Treatment of external inflammatory root resorption after autogenous tooth transplantation: case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2006; 102(1): 51-4.
37. Neely AL, Gordon SC. A familial pattern of multiple idiopathic cervical root resorption in a father and son: A 22-year follow-up. J Periodontol. 2007; 78(2): 367-71.
38. Nikolidakis D, Nikou G, Gert J, Meijer M, John A. Cervical external root resorption: 3-year follow-up of a case. J Oral Sci. 2008; 50(4): 487-99.
39. Altundasar E, Demir B. Management of a perforating internal resorptive defect with mineral trioxide aggregate: A case report. J Endod. 2009; 35(10): 1441-4.
40. Jacobovitz M, Lima DE. Treatment of inflammatory internal root resorption with mineral trioxide aggregate: a case report. Int Endod J. 2010; 41(9): 905-12.
41. Khojastepour L, Bronoosh P, Azar M. Multiple idiopathic apical root resorption: A case report. J Dent. 2010; 7(3): 223-8.
42. Sodawala J, Reddy R. Root resorption in orthodontics: A review of literature. Ind J Dent Res. 2011; 8: 215-5.
43. Patel S, Foschi F, Mannocci F, Patel K. External cervical resorption: a three-dimensional classification. Int Endod J. 2018; 51(2): 206-14.
44. Carney K, Colloc T, Kilgariff J. Management of rarely seen internal tunneling root resorption associated with a maxillary permanent incisor. Br Dent J. 2024; 236(17): 955-61. doi: 10.1038/s41415-024-7504-7.

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