ORIGINAL RESEARCH PAPER

Dentistry

ROOT RESORPTION

KEY WORDS:

Madhan Kumar

INTRODUCTION

Root resorption (RR) is a term used to describe non-infectious damage brought on by clastic cell activity, which leads to the loss of both hard and soft dental tissue. In permanent dentition, it is seen as a pathologic process that is mainly asymptomatic and physiological during the loss of primary teeth. Unmineralized organic cementoid and predentine shield the roots. Clastic cells cannot adhere to surfaces that are not calcified, which leads to RR. Since Andreasen initially introduced the RR classification in the 1970s, a number of other classifications with various terminologies have been suggested.1

Resorption is a pathology that results in the loss of dentin, cementum, and/or bone through either a pathologic or physiological process.2 After different forms of trauma, such as mechanical, chemical, or thermal injury, root resorption may take place. Internal or external root resorption are the two primary types.

Resorption of hard dental structures initially was narrated in 16th century. Root resorption (RR) in deciduous teeth is a typical physiological response, and resorption of hard dental structures was first noted in the 16th century.3 Though the process appears to be controlled by cytokines and transcription factors that are comparable to those involved in bone remodelling, the beginning mechanisms for physiologic root resorption in primary dentition are not well understood.4, 5 Permanent tooth root resorption does not happen spontaneously, and is almost always inflammatory in origin, unlike bone, which goes through continual physiologic remodelling throughout life. As a result, root resorption in the permanent dentition is a pathologic occurrence that, if left untreated, could cause premature loss of the concerned teeth. This review's objective is to provide a concise explanation of the biology, etiology, epidemiology, classification, and histologic characteristics of root resorption as well as the challenges associated with its diagnosis and treatment planning.

Classification

- Internal Root Resorption
  - Internal root canal inflammatory resorption
  - Internal root canal replacement resorption
- External Root Resorption
  - Progressive inflammatory resorption
  - Cervical resorption
  - Replacement resorption

Internal Root Resorption

Internal resorption is an inflammatory process that starts in the pulp area and results in dentin loss and maybe cementum invasion.6 Internal root canal inflammatory resorption and internal root canal replacement resorption are the two types of internal root resorption that are typically discussed.

Pathogenesis And Histology

A pathological process known as internal root resorption (IRR) is defined by the loss of dentine as a result of clastic cell activity. The blood supply transports the clastic cells into the pulp chamber when there is pulpal inflammation.

(i) In inflammatory resorption, intraradicular dentin resorption proceeds without the concomitant deposition of hard structures at the resorption sites. The condition, which may be seen on routine radiographs as a radio-clear zone centered on the root canal, is linked to the existence of granulation tissues in the resorbed area.

(ii) The resorptive activity in replacement resorption results in defects in the dentin next to the root canal and the concurrent deposition of bone-like tissue in some areas of the defect. It causes the pulp space to expand irregularly, obliterating part or all of the pulp chamber in the process.

Injury and stimulation are required for the two phases of root resorption. Predentin and the layer of odontoblasts, which coat the interior surface of the root canal, are involved in injury. The primary stimulator of IRR is infection. In the early stages of resorption, teeth do not exhibit symptoms. The pulpal source of the resorbing cells is the apical vital region of the pulp.6

Etiology

IRR's etiology is largely unknown. Numerous pathophysiological explanations for the loss of prednisone have been put forth, but trauma seems to receive the most support. In research including 27 individuals, carious lesions (25%) and trauma (43%) are the two most frequent etiological factors.7 The walls of the pulp chamber get colonized by cells that resemble macrophages when bacteria continue to infect the pulp. The main requirement for starting root resorption is the adhesion and dissemination of such cells.8 Although the full etiology and pathology have not yet been fully clarified, it can be said that trauma and pulpal inflammation/infection are the key contributory elements at the beginning of internal resorption.9

Classification

Internal (Root Canal) Inflammatory Resorption

Any part of the root canal system could experience this kind of resorption. It is characterized by an oval-shaped expansion within the pulp chamber that can be seen on radiographs (Fig. 3). Until the lesion has substantially deteriorated and caused a perforation or symptoms of acute or chronic apical periodontitis after the complete pulp has undergone necrosis and the pulp space has become infected, the problem may go undetected.10 The pink tooth of Mummery, so named in honor of the 19th-century anatomist James Howard Mummery, who originally documented the occurrence, may appear if resorption affects the coronal region of the tooth.11

Internal root canal inflammatory resorption causes a gradual loss of intraradicular dentin without the addition of hard tissue implantation at the resorption sites (Figs. 1–3). When the lesion progresses to the point that it can be seen on standard radiographs, it is typically linked to chronic pulpal inflammation and bacteria may be found in the granulation tissues.12 (Fig. 3). Even though chronic inflammation is frequently present in pulpal infections, it does not alone create the conditions required for mediating root canal inflammatory resorption. To start the event, additional requirements must be present at the same time.

This likely explains why external inflammatory root resorption rather than root canal inflammatory resorption is seen less commonly (EIRR). For the resorptive lesion to advance and expand, the apical region of the pulp must remain viable because the coronal part of the pulp is typically necrotic (Fig. 1). According to one theory, the apical part of the

infected pulp experiences inflammation as a result of the necrotic coronal region of the infected pulp.

The discovery that osteocytes participate in bone homeostasis by suppressing osteoclastogenesis provides a different explanation.13 The actin rings that distinguish active resorbing cells from osteoclasts are not produced by osteoclasts in the presence of living osteocytes. On the other hand, osteocyte death results in the release of osteoclastogenic cytokines, which cause bone resorption.14,15 Dental pulp cells and odontoblasts go through apoptosis throughout tooth development as well as in reaction to specific kinds of injury, just like osteocytes do.16-19 Therefore, it's likely that odontoblasts or pulpal fibroblasts going through apoptosis due to injury or caries create cytokines that start an internal resorptive response in the pulp's apical region. Only when the predentin surrounding the site of persistent inflammation is removed as a result of trauma or other unidentified etiologic causes can internal resorption take place.20

Internal (Root Canal) Replacement Resorption

Internal root canal replacement resorption is defined by a discontinuity of the usual canal space and an uneven radiographic expansion of the pulp chamber.21 The defect encompasses a portion of the canal space because the resorption process begins inside the root canal; as a result, the original canal's contour seems deformed.

A substance with a fuzzy appearance and a mild to moderate radiodensity appeared to have obliterated the expanded canal space on radiographs (Fig. 4). Unless the resorptive process develops in crown or root rupture, this form of resorption is often asymptomatic, and the affected teeth may respond normally to heat and/or electric pulp testing.22 A minimal inflammation of the pulpal tissues, such as chronic irreversible pulpitis or partial necrosis, is likely to be the primary reason for root canal replacement resorption. The odontoblast layer and the predentin must be obstructed or compromised along a region of the canal wall for the chronic inflammatory process to occur before the resorption component of the condition starts, as odontoblasts must attach to extracellular proteins that contain the RGD amino acid sequence.20

Internal Tunneling Resorption

Internal tunneling resorption is a variation of internal root canal replacement resorption that has been documented in the past.23 This entity can appear after luxation losses but is typically encountered in the coronal region of root fractures. In some areas, bone-like tissues are also deposited as a result of the resorption process, which creates tunnels in the dentin near the root canal. These bone-like tissues resemble cancellous bone as opposed to compact bone (Fig. 5). The process may then come to a halt, and cancellous bone may completely obliterate the canal space.

Regarding the genesis of the metaplastic hard tissues that develop within the canal space, various hypotheses have been put forth. According to the first theory, postnatal dental pulp stem cells found in the apical, essential portion of the root canal form the metaplastic tissues as a reparative reaction to the resorptive insult.24,25 According to the second theory, nonpulpal origins are shared by the granulation tissues and metaplastic hard tissues. Those tissues may have evolved from periodontal or vascular compartment cells that have transmigrated there.26 According to this theory, periodontium-like connective tissues replace the pulpal tissues during internal resorption.

Prevalence

Although internal root resorption is regarded as being uncommon, it is unknown how frequently internal resorption occurs. The accuracy of the methods used to assess the pathology will substantially influence the results. IRR was found to occur more frequently through histological research than by merely looking at the X-rays. According to the pulp's level of inflammation, estimates for the frequency of internal resorption range from 0.01% to 55%.27 In teeth with pulpitis and pulp necrosis, internal resorption was frequently found, according to more recent histological research. Due to their small size, the lesions are unlikely to be found by standard clinical or radiographic techniques. The growth of the resorption is stopped when the pulp becomes completely necrotic. One other justification for completely irrigating canals with sodium hypochlorite during therapy is provided by the incidence of such lesions (concavities).28

Clinical Diagnosis

Internal resorption is frequently clinically diagnosed with standard full mouth radiographs and is typically asymptomatic. Depending on the pulpal state or root perforation that causes a periodontal lesion, pain may be experienced.29 The position and width of the IRR, however, can affect the clinical symptoms. Clinical evidence of "pink spot" can be seen if the internal resorption is situated in the coronal portion of the canal. The highly vascularized connective tissue next to the resorbing cells is what gives the pink tint. When the pulp turns necrotic, this hue changes to grey or dark grey.30

Thermal and electrical vitality tests produce a good response until the lesion becomes much larger and perforates. Apical periodontitis is brought on by the inflammatory connective tissue that fills the IRR defects degenerating, going through necrosis. After that, the tooth can exhibit symptoms, and periradicular abscesses might develop.

Radiographic Diagnosis

The radiographic appearance of an oval-shaped expansion within the pulp chamber or the root canal is what distinguishes an intraoral X-ray of IRR. However, a typical X-ray examination makes it challenging to make an early diagnosis of the IRR. If IRR is thought to exist, it is advised to take many shots at various incidence angles. But developing a suitable treatment strategy requires a precise diagnosis.

In a few case reports, CBCT has been used successfully to assess the true nature and severity of resorption lesions, indicating that the doctor might more confidently diagnose and treat the issue. ROC Az results of a study comparing the CBCT's accuracy of diagnosis to that of intraoral radiographs were 0.78 and 1.00, respectively, demonstrating the CBCT's greater accuracy.31

The use of CBCT offers axial, coronal, and parasagittal views of the anatomy together with a 3-dimensional understanding of the resorption lesion. The size and position of the resorption are identified in the series of cross-sectional views with good sensitivity and great specificity. CBCT is highly accurate at spotting early-stage root lesions.32

There may occasionally be a deposition of metaplastic hard tissue in the resorption area that resembles bone or cement. This replacement resorption material offers the illusion of expanding the pulp chamber while giving the canal space a fuzzy appearance. All of these factors support the differential diagnosis of external root resorption and enable the evaluation of the tooth's prognosis if the lesion is amenable to therapy.

Differential Diagnosis

The patient may have pulpitis signs if the pulp is still partially vital. However, the patient may eventually experience the signs of apical periodontitis if the resorption is no longer occurring and the entire pulp has turned necrotic. The clinical examination may reveal sinus tracts, which may be a sign of a persistent apical abscess or root perforation. The pink spot of

Mummery has historically been considered a pathognomonic sign of internal root resorption. However, ECR is more frequently linked to these pink patches.33 As a result, pink patches alone cannot be used to make a diagnosis of internal root resorption. The teeth that display internal root resorption are frequently asymptomatic and show no clinical symptoms. Internal root resorption can show clinically in a variety of ways, hence radiographic examination is the primary tool used to diagnose the condition. History and clinical findings are used as supplemental data.34

Management Of Internal Root Resorption

Conservative Dental Treatments Of Resorbed Teeth

Internal root resorption is still best treated with root canals since they cut off the clastic cells' blood supply and granulation tissue. Instrumentation and filling challenges associated with internal root resorption are specific.

To protect the dental structure and prevent additional deterioration of the already weakened tooth, the access cavity preparation must be as conservative as feasible. In teeth with active resorbing lesions, brisk bleeding may make it difficult to see until the apical pulp tissue has been severed and eliminated. Direct mechanical instrumentation is typically unable to access the resorption defect because of its form.35

In the event of resorptive perforation, the working length determination with an apex locator is not feasible. The chemical breakdown of the viable and necrotic pulp tissue with sodium hypochlorite must receive a lot of attention. The irrigation solution of hypochlorite is activated and made easier to penetrate to all parts of the root canal system when using ultrasonic devices.36 EndoActivator's non-traumatic plastic tips are especially recommended for achieving a thorough chemomechanical debridement of the root canal. When used as an interappointment dressing, calcium hydroxide increases the effectiveness of cleaning techniques, aids in controlling bleeding, and necrotizes leftover pulp tissue.

To seal the resorptive defect in the root canal filling, the material must be flowable. When the canal walls are respected, thermoplastic gutta-percha procedures appear to produce the best outcomes. MTA is the material of choice to seal a perforation in the rootwall because it is biocompatible, bioactive, and well-tolerated by periradicular tissues.37 If the substance begins to harden while being used, the addition of water can be used to modify the working duration.

Complete Root Canal Filling Using Warm Gutta Percha

The best long-term prognosis is provided by this option, which is for IRR without perforation of the canal walls. Two sessions are needed to complete the treatment. During the initial appointment, the root canal length is measured manually, the canal and resorption lacuna are cleaned and disinfected with sodium hypochlorite, calcium hydroxide is filled into the canal and lacuna as an interappointment dressing to finish the cleaning of the canal space, and glass ionomer cement is used to temporarily seal the access cavity.
In the second session, the root length is measured, the gutta-percha master cone is fitted, the last irrigation is performed, the apical third of the root is obturated with warm gutta-percha, the resorption lacunae are filled with gutta percha thermocompaction, and the access cavity is waterproofed with a GIC.

Sealing Internal Root Resorption using Bioactive Cement like MTA

This choice is suggested if the canal walls have been breached, allowing communication between the periapical tissue and the root canal system. The prognosis of the tooth in this clinical scenario is more predictable with the smaller the perforation size. The canal can be filled with MTA due to the position of the resorption and the short length of the root (Figure 3). If not, gutta-percha will be used to fill the canal's healthy portion.

Surgical Treatment

When access to the lesion through the canal is not possible, surgery is required. After orthograde treatment (or retreatment) has been completed and the coronal portion of the canal has been filled, surgery should always be done with a second intention. In these situations, the surgical method enables direct access to the lesion and mechanical cleansing of the resorbed defect due to the morphology of the lesion. There is a mucoperiosteal flap raised after local anesthetic. Access to the root area is made possible by removing the cortical bone plate. With the help of an operating microscope, the intraradicular dentin cavity is cleansed, dried, and the soft tissue lesion is curetted. On its outside, the filling materials (such as MTA or Biodentine) are positioned and smoothed. After the surgery, the area around the wound is painstakingly cleaned. Repositioning and suturing the flap (Figure 5).

External Root Resorption

The process of external inflammatory root resorption begins with mild lesions of the periodontal ligament and/or cementum brought on by trauma or bacterial contamination, which results in tiny root resorption cavities that eventually reach the dentinal tubules and the root canal. 1 At first, the pulp tissue is not involved. 2 rapid damage (trauma upon reimplantation) or long-term persistence are other potential causes (orthodontic force).39

Progressive External Inflammatory Resorption

The main causes of this kind of resorption are endodontic or orthodontic issues. Approximately 5–10 years after the end of orthodontic treatment, a radiologic investigation of 20–24-year-old patients indicated that 42.3% of the maxillary central incisors, 38.5% of the maxillary lateral incisors, and 17.4% of the mandibular incisors had suffered apical resorption.40

The blood vessels in the apical foramina are damaged as a result of the teeth' movement, which causes ischemic pulp necrosis. In most cases, bacteria will then enter the root canal through enamel-dentin fissures and exposed dentinal tubules, and infection will develop within two to three weeks. The root resorption brought on by the denuded portions of the root surface may have exposed tubular root dentin at this point. Through the dentinal tubules, bacteria from the infected root canal will subsequently go to the resorptive lacunae on the root surface and support the root's resorption.

Thus, mechanical damage in luxated teeth causes root resorption, which removes cementoblasts, precementum, and occasionally cementum from portions of the root surface. The microbial stimuli from the infected root canal then continue to stimulate the resorbing cells as needed, maintaining the resorptive process. The problem can be identified radiographically after a few weeks as periradicular radiolucent regions, which typically include portions of the root and the nearby alveolar bone.

Cervical Resorption

A common yet underappreciated form of increasing external inflammatory resorption is cervical resorption. Clinically and radiographically, it appears as a solitary resorption lacuna in the tooth's cervical region.41,42 It seems to be related to damage to the cervical attachment system, specifically to the precementum that covers a portion of the cervical root surface below the epithelial attachment. Hard tissue-resorbing cells then colonize the injured area of the root surface, and in most cases, the cervical resorption is temporary, meaning that cemental healing will take place in 2-3 weeks without therapy.

However, bacterial products from the gingival sulcus and the tooth's surface, rather than the root canal, may supply the

essential stimulation of the resorbing activity for extended periods via the tubules of the cervical dentin. Cervical resorption may manifest as ankylosis and replacement resorption if the local traumas cause the periodontal ligament tissue nearby to become necrotic.

Dento-alveolar Ankylosis And Replacement Resorption

Following significant necrosis of the periodontal ligament and the development of bone over a denuded portion of the root surface, dentoalveolar ankylosis develops. Specifically, in avulsed teeth that have been out of the mouth for long enough for the cells on the root surface to dry out and die, the condition is most frequently encountered clinically as a consequence of luxation injuries. Ankylosis may reverse itself if less than 20% of the root surface is affected.43,44 If not, ankylosed teeth become a part of the bone's natural remodeling process and are absorbed into the alveolar bone. The phrase "replacement resorption" came because they will gradually resorb and be replaced by bone.45,46

The osteoclasts typically involved in bone remodeling are the resorbing cells in replacement resorption. Therefore, replacement resorption shouldn't be considered a disease process even though it results in total tooth disintegration. Because the cells involved in bone remodeling are unable to distinguish between bone, dentin, and root cementum, it happens "by accident." The osteoblasts that are unable to create dentin or enamel will then replace the resorbed portions of the root with bone. The osteoclasts will resorb the dental tissues just as readily as they resorb the bone.

Clinically, ankylosed teeth's immobility makes dentoalveolar ankylosis apparent.44 These teeth will eventually be in infra-occlusion and produce a distinctive metallic percussive sound. Lack of a periodontal ligament space on radiographs is a sign of dentoalveolar ankylosis. The tooth will also have a distinctive moth-eaten appearance due to replacement resorption and bone ingrowth into the dental tissues.

Prevalence

External root resorption typically affects people between the ages of 21 and 30 more frequently (28.40%) than other age groups, and it affects females more frequently (59.04%) than males (Opacic 2004). Numerous aetiological causes have been identified, including trauma, prior periodontal surgery, pressure from neighboring unerupted teeth, pathological diseases including tumors, and tooth reimplantation (Opacic 2004; St George 2006). When the forces used to cause tooth movement are not under control, orthodontic tooth movement may also contribute to ERR. In these circumstances, the resorption typically takes place in the apical third of the root (Abuabara 2007).

In addition to radiation therapy, root resorption can develop from endocrine problems and systemic diseases such as hyperparathyroidism, Paget's disease, calcinosis, Gaucher's disease, and Turner's syndrome (Carrotte 2004). However, it is well acknowledged that for root resorption to begin, two factors—injury and stimulation—must coexist (Fuss 2003).

Diagnosis

The care of ERR requires early diagnosis since the earlier treatment is started, the less severe the long-term effects of resorption will be (Da Silveira 2007). A mix of radiographic and clinical examinations should be used to make the diagnosis. Radiographs taken from various angles may be helpful to identify which surface is affected since intraoral radiographs of the lesion typically exhibit an inconsistent root surface shape (Bergmans 2002). The type of ERR can also be determined through vitality tests (Fuss 2003; Nance 2000).

Due to its better sensitivity and specificity, computerized tomography may be a valuable diagnostic technique, especially in identifying minor and less accessible root resorption (Da Silveira 2007). Additionally, the diagnosis should attempt to distinguish between internal root resorption (IRR) and ERR (Carrotte 2004).

Management

Depending on the kind and degree of resorption, treatment options may include symptomatic alleviation for pain and swelling as well as, if necessary, stabilizing any movable teeth (Trope 2000).

Endodontic therapy, surgery to remove the granulation tissue, and filling of the resorptive defect may be necessary if there is pulpal involvement (Fuss 2003). To stop the resorptive process and seal the tooth's apex, root canal medicines and intracanal cement like MTA have also been applied (Gulsahi 2007).

The removal of the tooth or pressure will typically stop additional root resorption if it resulted from pressure from an erupted tooth, erupting teeth, or orthodontic therapy and there is no sign of infection (Heithersay 2007). However, splinting might be necessary if teeth are still very movable after orthodontic treatment is finished.

Due to the invasive nature of hyperplastic invasive cervical resorption, it is crucial to completely remove or inactivate the resorbing tissue via a chemical method or surgical techniques (Heithersay 2007).

The course of treatment for replacement resorption (ankylosis) will depend on the stage of tooth development, the seriousness of the damage, and the degree of necrosis of the periodontal ligament. Younger individuals are more likely to experience early tooth loss followed by ridge resorption, thus the doctor should think about timely and proper management of the resorptive process. This may entail regenerative therapies, closing orthodontic spaces, or ultimately extracting the ankylosed tooth and adding bone (Sapir 2008).

There is currently no agreement on how to address the various types of external root resorption (Fuss 2003; Majorana 2003).

CONCLUSION

As can be seen from the examples above, root resorption in dentistry is a multidisciplinary issue. However, with the knowledge, we currently have about the pathophysiology, etiology, and clinical manifestations of the resorptive processes, our therapeutic approaches can be more targeted and successful than ever. Only the replacement resorption that takes place in teeth with dentoalveolar ankylosis is still beyond the scope of our treatment expertise. This is a significant clinical issue in dental traumatology, and it is important to conduct rigorous research to determine how to regenerate the periodontal ligament in luxated teeth.

A specific type of pulp disease called internal inflammatory root resorption can be identified through clinical and radiographic evaluation of the patient's teeth. Three-dimensional imaging has a considerable impact on internal root resorption diagnosis nowadays. Additionally, because of the CBCT's greater diagnosis accuracy, resorptive abnormalities were better managed and conservative treatment for teeth with internal resorption had better results. Internally resorbed teeth should be treated with root canals using contemporary endodontic techniques such as optical assistance, ultrasonic enhancement of chemical debridement, and thermoplastic filling techniques. Alternative materials, such as calcium silicate cement, present fresh possibilities for restoring resorbed teeth. Even when the root walls are pierced in these circumstances, the prognosis for treating internal resorptions is favorable.

REFERENCES

1. Aidos HFA, Diogo P, Santos JM. Root Resorption Classifications: A Narrative Review and a Clinical Aid Proposal for Routine Assessment. Eur Endod J 2018; 3:134-45
2. American Association of Endodontists, "Glossary of endodontic terms," 2012
3. Sak M, Radecka M, Karpiski T, Wdrychowicz-Welman A, Szkaradkiewicz A. Tooth root resorption: etiopathogenesis and classification. MicroMedicine 2016; 4(1):21–31.
4. Trope M. Root Resorption due to dental trauma. Endodontic Topics 2002; 1 (1):79–100.
5. Patel S, Saberi N. The ins and outs of root resorption. Br Dent J 2018; 224(9):691–699.
6. Z. Fuss, I. Tsesis, and S. Lin, "Root resorption—diagnosis, classification and treatment choices based on stimulation factors," Dental Traumatology, vol. 19, no. 4, pp. 175–182, 2003.
7. M. K. Calis¸kan and M. T¨urk¨un, "Prognosis of permanent teeth with internal resorption: a clinical review," Dental Traumatology, vol. 13, no. 2, pp. 75–81, 1997.
8. C. Wedenberg and S. Lindskog, "Experimental internal resorption in monkey teeth," Endodontics&Dental Traumatology, vol. 1, no. 6, pp. 221–227, 1985.
9. S. Patel, D. Ricucci, C. Durak, and F. Tay, "Internal root resorption: a review," Journal of Endodontics, vol. 36, no. 7, pp. 1107–1121, 2010.
10. Frank AL, Weine FS. Nonsurgical therapy for the perforative defect of internal resorption. J Am Dent Assoc 1973;87:863–8.
11. Mummery JH. The pathology of pink spots on teeth. Br Dent J 1920;41:30111.
12. Wedenberg C, Lindskog S. Experimental internal resorption in monkey teeth. Endod Dent Traumatol 1985;1:221–7.
13. Heino TJ, Hentunen TA, Va¨a¨na¨nen HK. Osteocytes inhibit osteoclastic bone resorption through transforming growth factor-beta: enhancement by estrogen. J Cell Biochem 2002;85:185–97.
14. Gu G, Mulari M, Peng Z, Hentunen TA, Va¨a¨na¨nen HK. Death of osteocytes turns off the inhibition of osteoclasts and triggers local bone resorption. Biochem Biophys Res Commun 2005;335:1095–101.
15. Cardoso L, Herman BC, Verborgt O, Laudier D, Majeska RJ, Schaffler MB. Osteocyte apoptosis controls activation of intracortical resorption in response to bone fatigue. J Bone Miner Res 2009;24:597–605.
16. Franquin JC, Remusat M, Abou Hashieh I, Dejou J. Immunocytochemical detection of apoptosis in human odontoblasts. Eur J Oral Sci 1998;106(Suppl 1):384–7.
17. Bronckers AL, Goei SW, Dumont E, et al. In situ detection of apoptosis in dental and periodontal tissues of the adult mouse using annexin-V-biotin. Histochem Cell Biol 2000;113:293–301.
18. Kitamura C, Ogawa Y, Morotomi T, Terashita M. Differential induction of apoptosis by capping agents during pulp wound healing. J Endod 2003;29:41–3.
19. Mitsiadis TA, De Bari C, About I. Apoptosis in developmental and repair-related human tooth remodeling: a view from the inside. Exp Cell Res 2008;314:869–77.
20. Wedenberg C, Zetterqvist L. Internal resorption in human teeth: a histological, scanning electron microscopic and enzyme histochemical study. J Endod 1987;6:255–9.
21. Oehlers FAC. A case of internal resorption following injury. Br Dent J 1951;90: 13–6.
22. Ne RF, Witherspoon DE, Gutmann JL. Tooth resorption. Quintessence Int 1999;30:9-25.
23. Andreasen FM, Andreasen JO. Resorption and mineralization processes following root fracture of permanent incisors. Endod Dent Traumatol 1988;4:202–14.
24. Gronthos S, Brahim J, Li W, et al. Stem cell properties of human dental pulp stem cells. J Dent Res 2002;81:531–5.
25. Huang GT, Gronthos S, Shi S. Mesenchymal stem cells derived from dental tissues vs. those from other sources: their biology and role in regenerative medicine. J Dent Res 2009;88:792–806.
26. Stanley HR. Diseases of the dental pulp. In: Tieck RW, ed. Oral pathology. New York: McGraw Hill; 1965.
27. M. Haapasalo and U. Endal, "Internal inflammatory root resorption: the unknown resorption of the tooth," Endodontic Topics, vol. 14, pp. 60–79, 2006.
28. C. Gabor, E. Tam, Y. Shen, and M. Haapasalo, "Prevalence of internal inflammatory root resorption," Journal of Endodontics, vol. 38, no. 1, pp. 24–27, 2012.
29. M. K. Calis¸kan and M. T¨urk¨un, "Prognosis of permanent teeth with internal resorption: a clinical review," Dental Traumatology, vol. 13, no. 2, pp. 75–81, 1997.
30. F. F. Silveira, E. Nunes, J. A. Soares, C. L. Ferreira, and I. Rotstein, "Double "pink tooth" associated with extensive internal root resorption after orthodontic treatment: a case report," Dental Traumatology, vol. 25, no. 3, pp. e43–e47, 2009.
31. S. Patel, A. Dawood, R. Wilson, K. Horner, and F. Mannocci, "The detection and management of root resorption lesions using intraoral radiography and cone beam computed tomography— an in vivo investigation," International Endodontic Journal, vol. 42, no. 9, pp. 831–838, 2009.
32. C. Estrela, M. R. Bueno, A. H. G. de Alencar et al., "Method to evaluate inflammatory root resorption by using cone beam computed tomography," Journal of Endodontics, vol. 35, no. 11, pp. 1491–1497, 2009.
33. Patel S, Kanagasingham S, Pitt Ford T. External cervical resorption: a review. J Endod 2009;35:616–25.
34. Gulabivala K, Searson LJ. Clinical diagnosis of internal resorption: an exception to the rule. Int Endod J 1995;28:255–60.
35. Frank AL. External-internal progressive resorption and its nonsurgical correction. J Endod 1981;7:473–6.
36. 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;18:148–52.
37. Ahlberg K, Bystedt H, Eliasson S, Odenrick L. Long term evaluation of autotransplanted maxillary canines with completed root formation. Acta Odontol Scand 1983;41:23–31.
38. Consolaro A. The four mechanisms of dental resorption initiation. Dental Press J Orthod. 2013;18(3):7-9.
39. William Barnabé, João Batista de Souza, Lawrence Gonzaga Lopes, et al. Abordagem Interdisciplinar no Tratamento de Reabsorção Cervical Externa: Relato de Caso. Revista Odontológica do Brasil Central. 2011;20(55):359-365.
40. Barbin EL, Saquy PC, Guedes DFC, et al. Determination of para-chloroaniline and reactive oxygen species in chlorhexidine and chlorhexidine associated with calcium hydroxide. J Endod. 2008;34(12):1508-1514.
41. Yilmaz Hg, Kalender A, Cengiz E. Use of mineral trioxide aggregate in the treatment of invasive cervical resorption: a case report. J Endod. 2010;36(1):160-163.
42. Silva EJNL, Carvalho NK, Carvalho FR, et al. Abordagem Endodôntica e Visão Ortodôntica da Reabsorção Cervical Externa: Relato de Caso. Rev Odontol Bras Central. 2011;20(52):94-98.
43. Heward S, Sedgley CM. Effects of Intracanal Mineral Trioxide Aggregate and Calcium Hydroxide During Four Weeks on pH Changes in Simulated Root Surface Resorption Defects: An In Vitro Study Using Matched Pairs of Human Teeth. J Endod. 2011;37(1):40-44.
44. Vasconcelos, KF, Nejaim Y, Haiter Neto F, et al. Diagnosis of invasive cervical resorption by using cone beam computed tomography: Report of two cases. Braz Dental J. 2012;23(5):602-607.
45. Camargo SEA, Moraes MEL, Moraes LC, et al. Principais características clínicas e radiográficas das reabsorções radiculares internas e externas. Revista de Odontologia da Universidade Cidade de São Paulo. 2008;20(2):195 203.
46. Baranwal AK. Management of external invasive cervical resorption of tooth with Biodentine: A case report. J Conserv Dent. 2012;19(3):296–299.