Review Article

A Review of Internal Root Resorption

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ABSTRACT

Root resorption is the term used to describe resorption that occurs in the cementum or dentin of a tooth's root. Resorption is a challenging issue for all dental specialists. The reasons for the rapid erosion of dental tissues are unknown, the diagnosis is educated guesses, and treatment frequently fails to prevent it. Resorption can be observed radiographically even though it cannot be anticipated. Even this diagnostic tool has limits; because resorption on the buccal or lingual surface of the tooth often is not apparent until 20–40% of the tooth structure has been demineralized. Practitioners must be able to diagnose resorption radiographically or clinically, differentiate between internal and external resorption, and initiate the proper treatment to stop the resorption process, because the etiological factors, diagnosis, treatment, and prognosis vary depending on the different types of resorption defects. Thus, the aim of this study is to present etiopathogenesis, a clinical course, and diagnostic peculiarities of internal types of tooth root resorption, enabling practicing dentists to timely diagnose root resorption, and take appropriate measures to avoid further complications.

Key words: Cone-beam computed tomography, internal resorption, mineral trioxide aggregate, root canal treatment

INTRODUCTION

Resorption is described as a condition linked to either a pathologic or a physiological process that causes the loss of dentin, cementum, or bone in the American Association of Endodontists' glossary.

A uncommon pathological disorder called internal resorption begins in the pulpal cavity and spreads to the dentin around it. This dissemination is believed to have started with persistent coronal pulp inflammation brought on by ongoing bacterial stimulation. It is necessary to have live pulp tissue for internal resorption to occur. A thin anti-resorptive barrier protects the tooth roots' internal and external walls. The outer walls of the tooth's dentin are shielded by the pre-cementum layer, while its interior walls are kept safe by pre-dentin and odontoblasts.[1]

In the literature, a number of etiological factors for tooth root resorption are mentioned, with trauma and iatrogenic factors like bleaching and orthodontic treatment appearing out as particularly significant examples.[2,3] Although there are many potential causes, the etiology of some forms of resorptions is still unknown, necessitating additional study.

Based on the clinical and radiological presentation, the diagnosis can be made. Since tooth root resorption frequently exhibits minimal or no clinical symptoms, the patient should have a complete examination.[4] Radiographic information, however, eventually confirms the final diagnosis. Cone-beam computed tomography (CBCT) can be informative in this regard.

This article's aim was to enable practicing dentists to acknowledge root resorption in a timely manner and take the necessary precautions to prevent further complications by presenting the etiology, pathogenesis, clinical course, and diagnostic peculiarities of internal types of tooth root resorption.

Classification

Root resorption is a poorly known and perplexing subject. Many authors have categorized the resorptive area using their own terminology.

Andreasen has classified tooth resorption as internal and external root resorption.[5]

1. Internal resorption
   - Root canal replacement resorption
   - Internal inflammatory resorption.
2. External resorption
   - Surface resorption
   - Inflammatory resorption
   - Replacement resorption
   - Dentoalveolar ankyloses.

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Lindskog also subdivides resorption into three broad groups, namely:[6]

- Trauma induced tooth resorption
  - Surface resorption
  - Transient apical internal resorption
  - Pressure
  - Orthodontic
  - Replacement resorption.
- Infection-induced tooth resorption
  - Internal inflammatory resorption
  - External inflammatory resorption
  - Communicating internal-external (infective) inflammatory resorption.
- Hyperplastic invasive tooth resorption
  - Internal (invasive) invasive replacement resorption
  - Invasive coronal resorption
  - Invasive cervical resorption
  - Invasive radicular resorption.

Etiopathogenesis and Histology

After different types of damage such as mechanical, chemical, or thermal injury, root resorption may take place. After odontoblasts necrosis, internal resorption occurs and is accompanied by chronic partial pulp inflammation and partial pulp necrosis.

It can be brought on by a number of things, including pulpotomy, trauma, cavities, pulp capping with calcium hydroxide, partial pulp removal, severe heat, and a broken tooth. These elements cause the pulp tissue to get stimulated, which starts an inflammatory process. Later, some undifferentiated pulp cells turn into osteoclasts or macrophages, causing dentinal resorption.

The presence of living pulp tissue at or below the resorption area and partially or completely necrotic pulp coronal to the site of resorption, which allows continuous access of bacteria and their antigens into the root canal, is two criteria that define the course of internal resorption. For resorption to last, microbial stimulation is essential.[4]

The degree of development is also influenced by the intensity of the stimuli and the inflammatory response. A healthy blood supply is necessary for the production of clastic cells, and necrotic tissue acts as a catalyst for this process. This explains why, in contrast to external root resorption, IR is considered a rare phenomenon. Hyperemia, which is brought on by vascular changes in the pulp and results in low pH and high oxygen tension, draws a lot of macrophages to the area and starts the resorptive process.

A syncytium of activated macrophage progenitor cells is represented by osteoclasts. The Rank-Rankl/osteoprotegerin (OPG) system is in charge of controlling this stimulation. It functions as an on-off switch for osteoclastic activity, with stimulation of the OPG system and down-regulation of the RANKL system possibly favoring development of new osteoclasts. The less frequent type of root resorption is known as internal root resorption (IRR), which is a resorptive defect of the internal portion of the root. In order for IRR to take place, the pre-dentin and outermost odontoblast layer of the root canal wall must be damaged. This will cause odontoclasts to release hydrolytic enzymes that break down dental hard tissues, demineralizing the apatite crystals in the hard dental tissues through the H+- ATPase enzyme. They use the enzymes cathepsin K and MMP-9 to degrade dentin proteins.[7,8] Clinically, IRR frequently shows no symptoms. IRR, however, will eventually result in necrosis of the pulp tissue apical to the resorptive lesion, which can produce periapical diseases such apical periodontitis or apical abscess if left untreated.

The resorption site represents resorbing granulation tissue interposed between healthy and diseased pulp tissue. The untreated internal resorption can progress into external or *vice versa* which causes fracture of the tooth. Internal resorption is rarely found in permanent dentition.

Male participants are affected by the ailment more commonly than female ones. Although internal resorption is a very uncommon ailment, it has been linked to a higher frequency in teeth that have undergone particular treatment methods like auto-transplantation. Maxillary incisors are the teeth that are most frequently impacted.[4]

In addition, it can occasionally manifest as a pink or reddish spot that is visible on the crown; this is granulation tissue that is poking through the resorbed area. In the IRR case, the pink spot can be seen centered on the clinical crown. Herpes zoster has also been linked to resorption and odontoblast degeneration as a result of systemic viral infection, according to the literature.[9]

Internal resorption may continue if left untreated until the inflammatory connective tissue filling the resorptive defect degenerates, pushing the lesion in an apical direction. If untreated, the pulp tissue apical to the resorptive lesion necrotizes and the bacteria infect the entire root canal system, resulting in apical periodontitis.

PREVALENCE

Although IRR 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. The frequency of IRR was found to be higher by histological studies than by only looking at the X-rays. Depending on how inflamed the pulp is, it has been estimated that between 0.01% and 55% of cases of internal resorption occur.[9]

CLINICAL FEATURES

The clinical characteristics of IRR are mostly determined by the histologic condition of the afflicted pulp, the level of hard tissue destruction induced by the resorptive process, and the location of the resorptive cavity within the root canal space. Bacterial contamination of critical pulpal tissue during the active stages of resorption may trigger an immediate inflammatory reaction, resulting to clinical pulpitis symptoms.

Clinical signs and symptoms of acute or chronic apical periodontitis may emerge with the start of pulpal necrosis and an established bacterial colonization of the root canal space. Sinus tract(s) may develop and be associated with suppuration in the periapical tissues or a perforation of the root canal wall caused

by hard tissue deterioration. Extensive resorption of the coronal pulp can induce granulomatous tissue to spread into and occupy the resorptive defect, resulting in a pink or crimson discoloration visible through the crown of the affected tooth.[10-12]

Although these pink patches are frequently cited as a common clinical sign of the disease, they are uncommon in cases with IRR. They may occur more frequently in cases of ECR, but they are also uncommon with that resorption type. Often, the afflicted tooth is asymptomatic, with no clinical indications.

RADIOGRAPHIC FEATURES AND DIAGNOSIS

The existence of any sort of root resorption is determined by radiographic evidence. The detection and separation of distinct forms of resorption are difficult with two-dimensional radiography. Both IRR and ERR may have comparable characteristics, making distinction and diagnosis difficult from a two-dimensional shadowgraph.

According to Gartner *et al.*, IRR lesions appear radiographically as radiolucency of uniform density with a smooth shape and are symmetrically distributed over the root of the affected tooth. The authors also stated that the outline of the root canal wall should not be traceable through the resorption defect because the root canal wall balloons out.

A relatively recent method of imaging in three dimensions, CBCT, uses a lot less radiation than traditional computed tomography. When using standard computed tomography, the patient is slowly moved through the equipment, while a narrow fan-shaped X-ray beam performs a series of spins around their head.[13]

Tomographic images are then created by reconstructing the raw data from each rotation. In contrast to traditional computed tomography imaging, CBCT acquires the entire volume of data with a single scanner sweep. Analysis options include counting the number of root surfaces, axial, transverse, and tangent slices, and real root resorption extension.

Light microscopy reveals various degrees of pulpal tissue inflammation with the infiltration of lymphocytes, macrophages, and some leukocytes as well as dilated blood vessels and multinucleated dentinoclasts on the pulpal-dentin surface.[8]

The pulpal-dentin wall is visible under an electron microscope without odontoblasts. Numerous philopod-equipped dentinoclasts with a size of 50 m are oriented toward the surface of the dentin and attached to it.[14]

MANAGEMENT OF RESORPTION

Root canal treatment remains the treatment of choice of IRR as it removes the granulation tissue and blood supply of the clastic cells.

IRR requires specific precautions during root canal treatment.

- To prevent further weakening of the already weakened tooth and to preserve tooth structure, the access cavity preparation must be as conservative as feasible
- The morphology of the resorption defect usually makes it inaccessible to direct mechanical instrumentation
- A brisk bleeding could impede visibility in teeth with active resorbing lesions until the apical pulp tissue has been cutoff and eliminated
- In the event of resorptive perforation, the working length determination with an apex locator is not feasible. A great emphasis must be placed on the chemical dissolution of the vital and necrotic pulp tissue with sodium hypochlorite
- The irrigation solution of hypochlorite is activated and made easier to penetrate to all parts of the root canal system when ultrasonic devices are used
- 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 seem to produce the greatest outcomes
- As MTA is biocompatible, bioactive, and well-tolerated by periradicular tissues, it is the material of choice to seal perforations in the root wall. If the substance begins to harden while being used, the addition of water can be used to modify the working duration.

IRR without perforation

The treatment is performed in two stages:

First stage	Second stage
After administration of local anesthesia, rubber dam should be placed then access cavity preparation should be made along with determination of the root canal length with manual instruments and shaping of the canal	After the placement of rubber dam temporary filling has to be remove along with copious irrigation of root canal activated with sonic tips to removal the calcium hydroxide.
For disinfection of the canal and resorption lacuna sodium hypochlorite should be used with activation of the solution with ultrasonic tips to clean the resorptive area.	Master gutta-percha cone should be verified radiographically to assess the good fit. Then final irrigation and drying of the root canal has to be done with sterile paper tips along with obturation of the apical third of the root with warm gutta-percha[15] (closed apex), or in case of the open apex obturation of root canal and resorption lacuna should be done with MTA under visual control with an operative microscope.[15]
Calcium hydroxide as an inter-appointment dressing will be used to complete the disinfection of the canal space followed by temporary sealing of the access cavity with glass ionomer cement (GIC).	Radiographic assessment has to be done for proper obturation followed by placement of a water-moistened cotton pellet directly over the material and provisional sealing of the access cavity with a GIC.

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. Due to the morphology of the lesion in these situations, surgical intervention enables direct access to the lesion and mechanical cleansing of the resorbed defect.

DISCUSSION

Internal resorption entails a gradual loss of intra-radicular dentin without the concomitant deposition of hard structures close to the resorptive sites. When the lesion progresses to the point where it can be seen on standard radiographs, it is typically accompanied by chronic pulpal inflammation, and bacteria may be found in the granulation tissues.[16]

It is unintentionally discovered through normal radiographs, because it has no symptoms. Only when the predentin surrounding the area of persistent inflammation is removed as a result of trauma or some unidentified etiologic event can internal resorption take place.

The success or failure of therapy should be monitored clinically as well as through radiological control. Naturally, if resorption is stopped and does not progress, we believe that our treatment is effective. We were able to save a tooth, and our therapy goal was met. The treatment of teeth with IRR is mostly determined by the magnitude of the lesion. Large lesions reduce the tooth's resistance to shear stresses, which can lead to tooth breakage. As a result, root canal therapy must begin as soon as feasible after the discovery of an inflammatory resorptive lesion in order to stop additional loss of hard tissue and eventual root rupture.[17]

CONCLUSION

Early diagnosis, source removal, and appropriate root resorption treatment are necessary for a satisfactory treatment outcome. Internal resorption is an uncommon form of tooth resorption that starts in the root canal and damages the tooth's surrounding tissue. By shutting off the blood supply to the resorbing tissues during conventional root canal therapy, it is easy to control the course of IRR. Regular recall is necessary to monitor the progress of healing and the overall prognosis of the tooth.

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