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Root Resorption and It's Management: a Review Article

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Abstract:
Root resorption is largely pathological and known to be initiated by several factors, including pulpal necrosis, trauma, periodontal treatment, orthodontic treatment and tooth whitening agents. The loss of dental hard tissue results from the activity of osteoclasts and may occur both externally and internally.
Accurate and early diagnosis of root resorption is essential for successful treatment.

Keywords: *Ankylosis, Hyperplastic, Invasive, Osteoclastic activity, Resorption*

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I. Introduction

In the adult dentition root resorption is caused by osteoclast-like multi or occasionally mononucleated cells called odontoclasts. Roots are usually protected against external and internal root resorption by unmineralised organic cementoid and predentin, respectively, and therefore do not undergo resorption in normal circumstances.[1] This is due to the inability of the clastic cells to adhere to unmineralised surfaces.

However, in the primary dentition it is desirable as this physiologic root resorption aids exfoliation of the deciduous tooth and thus facilitates eruption of the permanent successor.

Root resorption occurs in three stages; initiation, resorption and repair.[2]

The process of resorption may be self-limiting and go undetected clinically. Once initiated, if the initial surface resorptive process is sustained, for example by infection and/or pressure, dental hard tissue destruction will continue and tooth tissue loss may occur. This may result in the tooth becoming unsalvageable.

There are some rare tooth resorptions of unknown cause that do not fit into any of the above categories and they are usually labelled "idiopathic".

DIAGNOSIS:

Diagnostic accuracy based on conventional and digital radiographic examination is limited by the fact that the images produced by these techniques only provide a two-dimensional (2D) representation of three-dimensional (3D) objects.[3] This might lead to misdiagnosis and incorrect treatment in the management. A second radiograph taken from a different mesio-distal angle would alter the relationship of the defect to the root canal but not in the case of internal resorption.

The advent of cone beam computed tomography (CBCT) has enhanced radiographic diagnosis providing greater 3D appreciation of the tooth, the resorption lesion, and the adjacent anatomy[4]. The true nature of the lesion might be assessed, including root perforations and whether the lesion is amendable to surgical or non-surgical treatment.

CLASSIFICATIONS:

There are many classifications and terms for different types of resorptions:
[I] Andreasen classification of root resorption- (1970)
1) Internal
a) Inflammatory
b) Replacement

2)External
a) Surface
b) Inflammatory
c) Replacement
Andreasen's original classification does not include other resorptive processes which have been identified over the past two decades.
[II] Clinical related classification of resorption for that assist in diagnosis and treatment of this pathological process: Fuss et al (2003)
1) Pulpal infection root resorption
2) Periodontal infection root resorption
3) Orthodontic pressure root resorption
4) Impacted tooth or tumor pressure root resorption
5) Ankylotic root resorption
[III] Classification proposed by Lindskog (2006)
1) Trauma induced;
2) Infection induced; or
3) Hyperplastic invasive.

In this paper classification given by Lindskog et al will be followed:
(1) Trauma induced
It is induced by non infectiveetiology like:
-Pressure from unerupted or erupting teeth or some neoplasms,
- From biomechanical forces involved in orthodontics,
- Mechanical trauma (luxation and avulsion injuries) ,
- Surgical, thermal or
- Chemical trauma

Trauma induced tooth resorption may be subdivided into:
(1) Surface resorption;
(2) Transient apical internal resorption;
(3) Pressure resorption and orthodontic resorption; and
(4) Replacement resorption.

1)Surface resorption
A shallow resorption of cementum often with involvement of a small amount of underlying dentine. This type of resorption is self-limiting and transient and can follow some traumatic injuries or orthodontic treatment.
Radiographic features: Surface resorption may be difficult to observe radiographically as there may be absence of or only slight changes in the root morphology and the image of the periodontal membrane.

2) Transient apical internal resorption
Identified by Andreasen in 1986[5].
Cause: It follows luxation injuries and may be associated with a transient apical breakdown.
Radiographic features: Recognized by a confined periapical radiolucency which resolves within a few months.
It is considered to be a positive response, with the internal apical resorption allowing ingress of a greater vascular network to aid in the healing of a traumatized pulp.Often there is an associated colour change due to intra-pulpal haemorrhage and this may resolve spontaneously if revasularization to the coronal pulp chamber occurs.
In the longer term, as this is a transient process, the internally resorbed apex will close uneventfully.With the removal of the initiating "trauma", these non-infective resorptions will become inactive and uncomplicated repair will occur.

3) Pressure resorption and orthodontic resorption [OIIRR]
Causes: Resorption may be induced by the pressure of a crypt of an unerupted/erupting tooth or some neoplasms and more commonly during orthodontic treatment.Orthodontically induced resorption is an iatrogenic consequence of orthodontic treatment.
Radiographic features: The resorption is often extensive and easily observable radiographically.
Treatment:Longer intervals between activations remain strongly recommended[6]. When OIIRR is detected in the six-month periapical radiograph, treatment should be halted for two to three months with passive archwires[7].

4) External replacement resorption (ERR)

The most serious form of trauma induced noninfective root resorption is replacement resorption which, as the name suggests, involves the progressive replacement of tooth structure by alveolar bone and ultimately tooth loss.

Causes: Replacement resorption follows the death of viable periodontal ligament cells due to factors such as compression or drying of the ligament cells as in the case of delayed replantation of an avulsed tooth.

Pathogenesis: After stimulation denuded demineralized area is covered by multinucleated cells. In absence of further stimulation resorption process may stop spontaneously. If this damage is not large enough repair by cementum like tissue will occur in 2-3 weeks. If damaged surface is large then bone cells are able to attach to root and ankylosis occurs.

Clinical features: There is total loss of mobility due to this union of tooth and bone, and the tooth gives a characteristically high percussion sound but otherwise patients with replacement resorption are symptom-free.It has been reported that when ERR covers more than 20% of root surface the tooth will lose its physiologic mobility which may result in a high-pitched metallic sound to percussion.The tooth may appear infra-occluded in developing dentition

Radiographic features: There will be total loss of the image of the periodontal ligament followed by evidence of the progressive replacement of tooth structure by bone – in time the image of the tooth root is lost.

Treatment:

As ERR is external in nature, the tooth should respond to sensibility testing albeit with delay if there is tertiary dentine formation. However, lack of response to sensibility testing in the absence of other clinical signs and symptoms of endodontic infection, is not an indication for endodontic treatment.

If a tooth is in a satisfactory position in a mature dentition, there is no urgency for tooth replacement as often the replacement resorption proceeds at a slow rate – in some instances taking many years to reach a stage where carefully planned intervention is necessary. This provides valuable time for both the clinician to plan elective treatment.

Ankylosis in the developing dentition can severely disrupt arch formation and some form of early clinical intervention is desirable. In some cases of ankylosis and limited replacement resorption, a surgical repositioning procedure can be attempted to restore arch integrity.This procedure can be supplemented with the application of Emdogain (Biora AB Malmo, Sweden) to the affected root area in an attempt to repopulate the denuded surface with cementoblasts.

In cases of ankylosis with advanced replacement resorption a decoronation and submergence procedure is recommended. This allows ongoing alveolar growth both vertically and axially, and it facilitates the uncomplicated transition to implant therapy when appropriate[8].

(2) Infection induced

This may be a consequence of infective endodontic pathosis alone or superimposed on trauma induced resorption.

Inflammatory root resorptionsmay occur as

1) Internal resorptions,
2) External resorptions or
3) Combined internal-external lesions.

1) Internal inflammatory (infective) root resorption

Internal root resorption is the progressive destruction of intraradicular dentin and dentinal tubules along the middle and apical thirds of the canal walls as a result of clasticactivities[10].

In active progressive IIR lesions, the root canal coronal to the resorptive lesion will typically be necrotic, while apically the pulp is vital providing nutrients to the odontoclasts allowing the resorptive lesion to progress. However, if the tooth loses vitality, resorption will cease to progress[1].

Clinical features: Internal root resorption is usually asymptomatic, detected coincidentally through routine radiographs. Pain or discomfort may be the chief complaint if the granulation tissue has been exposed to oral fluids [11].

Radiographic features:

IIR may still be misdiagnosed as external cervical resorption in multi-rooted teeth as the root canal of an unaffected tooth may be superimposed over the affected root, thus giving the appearance of external cervical resorption.

Internal resorption may be described as symmetrical or eccentric lesion with sharp, smooth and clearly defined margin, with a uniform density of radiolucency, and the outline of pulp chamber or root canal could not be followed through the lesion[12]

Due to the granulomatous nature of the lesion, profuse bleeding from the root canal is frequently observed upon accessing the canal system. This will cease as soon as the pulp and granulation tissue have been completely removed.
Classified according to location as: (a) apical and (b) intraradicular.

a) Apical
Radiographic features: Apical internal resorption may be difficult to diagnose when the resorptions are of the lower grades.
Treatment: There are two approaches to the endodontic management of apical internal resorption. The first is to extend instrumentation only to the position of the resorption with the expectation that with the removal of micro-organisms followed by root canal filling, hard tissue repair will occur in the resorbed apical region of the tooth. The second approach is to enlarge and prepare the apical region, either with hand or rotary filing techniques, to include the resorbed region and then root fill to the root canal "terminus"[13].
Resorption control can be provided by the use of anti-clastic therapeutic agents such as Ledermix Paste used as intra-canal medicaments. Other materials such as calcium hydroxide or ProRoot MTA (Dentsply Tulsa Dental, Johnson City, Tennessee, USA) can also be used to stimulate hard tissue formation on resorbed root surfaces [9].

b) Intraradicular.

Internal resorption fully contained within an otherwise intact root will be referred to as intraradicular internal inflammatory resorption.

Pathogenesis: A common finding is a large accessory canal communicating from the periodontal ligament to the resorbed area; this may have allowed the passage of a collateral blood supply which probably played an important role in the development and maintainance of the internal resorptive process.

Clinical features: Patient might experience symptoms of pulpitis at the initial stage. At a later stage, the root canal system may become necrotic and patient might eventually develop symptoms of apicalperiodontitis[14]

Treatment: When diagnosed, immediate removal of the causative agent must be considered, aiming to arrest the cellular activity responsible for the resorptive activity[15]. However, the complex irregularities of the root canal system and the inaccessibility of internal resorption defect provide technical difficulties for thorough cleaning and obturation of the root canal system. The persistence of organic debris and bacteria in these areas may jeopardize the long-term success of the endodontic treatment. Therefore, a detailed exploration of the interior of the root canal is necessary for a successful treatment outcome[16].

A combined approach involving both hand instrumentation and antibacterial irrigation can be performed on the involved tooth. Sodium hypochlorite is the most commonly usedirrigant during root canal treatment due to its tissue dissolving and broad antimicrobial properties. Ultrasonic activation of irrigants should be viewed as an essential step in the disinfection of the internal resorption defect.However, even with the use of ultrasonic instruments, bacteria might still remain in confined areas. Chemo mechanical debridement of the root canal space fails to consistently render the root canal system bacteria-free. Thus, an intracanal, antibacterial medicament should be used to improve disinfection of the inaccessible root resorption defects. Calcium hydroxide is antibacterial and has been shown to effectively eradicate bacteria that persist after chemo mechanical instrumentation.Calcium hydroxide has also been shown to have a synergistic effect when used in conjunction with sodium hypochlorite to remove organic debris from the root canal.

The obturation of the canal can be achieved by a variety of techniques including hot vertically condensed gutta-percha, Obtura-delivered hot gutta-percha and more recent innovations such as the Microseal technique.

Due to the irregular nature of IIR and the root canal space, it is desirable to obturate the root canal with a thermoplasticised root filling to ensure optimum adaptation and compaction. In cases where IIR has perforated the root it may be necessary to seal the affected portion of root canal space with a bioactive root filling material, for example MTA or Biodentine.

2) External inflammatory root resorption (ERR)

Causes: Classically, this type of external root resorption occurs when infection is superimposed on a traumatic injury – usually following replantation of an avulsed tooth or a luxation injury. Nevertheless it can also be induced in some cases of endodontic pathosis.

Pathogenesis: A prerequisite for external inflammatory root resorption is damage to the normally protective cementum/cementoid which then initiates surface resorption exposing the underlying dentine to the

passage of bacteria or their metabolites from the root canal to the external root surface. This is a progressive form of root resorption which will ultimately result in tooth loss.

Radiographic features:Lesions caused by ERR may be asymmetrical and have ill-defined borders, with radio density variations in the body of the lesion[3]. The canal wall should be traceable through ERR lesion because ERR superimposes over the root canal.

The roots may appear shorter than normally expected and/or have ragged root ends, and will have apical radiolucencies adjacent to the root end.

As with other forms of infection induced resorption, treatment involves the thorough debridement and preparation of the root canal system. Irrigation is a most important component of this debridement process and the sequential use of 17% EDTA, 1% sodium hypochlorite and a final rinse with EDTA solution has been shown to be a most effective regimen resulting in a dentine surface devoid of smear layer.

An alternative approach involves the use of Ledermix paste as the initial intra-canal medicament to act as an anti-clastic agent. This root canal medication is replaced at six-weekly intervals for a period of approximately three months and then if there has been radiographic signs of resorption control (like no further increase in resorptive defect), calcium hydroxide can be used to influence hard tissue deposition on resorbed root surfaces.

Such dentine preparation using EDTA facilitates the diffusion of medicaments such as Ledermix paste through dentine to the external root surface where the corticosteroid and antibiotic components of the paste can exert a positive effect on the clastic cells responsible for the resorptive processes[17].

Prevention of inflammatory root resorption: A tooth with complete root development which has been subjected to avulsion, intrusion or a severe luxation injury should, after replantation or repositioning, have the pulp removed as soon as possible and the canal dressed with Ledermix paste so that its anti-clastic effect can be exerted in the early phases of the healing process[18].

3) Communicating internal-external inflammatory resorption

Where resorption has extended from an internal inflammatory resorption to involve the external surface a communicating lesion is created.

Radiographic features: This can be recognized radiographically by a radiolucency within the tooth structure extending to the exterior surface and the surrounding bone.

Ttreatment; Topical application of 90% aqueous trichloracetic acid to the resorptive tissue following endodontic preparation to the level of the resorptive defect.Trichloracetic acid is applied for 1–2 minutes on a mini-applicator or a small cotton pellet attached to an endodontic file. This will induce a sterile coagulation necrosis of the resorptive tissue which can act as a nidus for calcification – a process labelled by the author as "scaffolding"

Surgically after reflection of flap resorptive defect can be filled using MTA or biodentine.

(3) Hyperplastic invasive

The hyperplastic invasive tooth resorptions pose considerable challenges in management due to the complexity and aggressive nature of the resorptive process.

In these cases, resorbing tissue invades the hard tissues of the tooth in a destructive, and apparently uncontrolled fashion.

Unlike the first two types of resorption, simple elimination of the cause of the lesion is ineffective in arresting their progress. Total removal or inactivation of the resorptive tissue is essential if recurrence is to be avoided.

Subdivided into

a) Internal replacement (invasive) resorption (IRR)

Histological appearance of IRR defects reveals the presence of metaplastic hard tissue replacing the resorbed dentine at the periphery of the defect, which is suggestive of active and simultaneous resorption and replacement. This metaplastic hard tissue resembles cementum or osteoid-like tissues

Clinical features: Appear clinically as a pink area in the crown of the affected tooth.The location of the pink spot is more likely to be entirely within the crown of the tooth.

Radiographic features:The resorptive defect and the adjacent root wall usually have a cloudy and/or mottled appearance due to the radiopaque inclusions of hard tissue. The outline of the canal appears distorted and expanded.

Treatment: Management consisting of pulpectomy, curettage of the resorptive defect and obturation will generally control the resorptive process.

b) Invasive coronal resorption

Causes: Develops in erupting teeth where a localized coronal enamel defect allows the invasion of aggressive hyperplastic resorptive tissue. Invasive coronal resorption has also been observed in teeth which have been injured by the intrusion of a primary tooth.

Clinical features: In this case a pink resorptive defect can be observed in an area of hypomineralization.

Radiographic features: The image of the resorptive defect is generally irregular in outline and, depending on its extent, the radiolucency may extend both coronally and into the radicular tooth structure.

Treatment: Is directed towards the total removal or inactivation of all resorptive tissue and the restoration of the coronal defect. This may be achieved by physical curettage of the defect with round burs and hand instruments, but is more conveniently and effectively treated by the topical application of 90% aqueous trichloracetic acid, curettage, endodontic therapy if there is pulp involvement, and restoration of the defect with a glass ionomer cement. Orthodontic extrusion to render the defect into a supragingival position may supplement treatment if the resorption extends deeply.

c) Invasive cervical resorption(ICR) or peripheral inflammatory root resorption (PIRR) or External cervical resorption (ECR)

Invasive cervical resorption (ICR) is a type of external resorption that can involve the coronal, middle, and apical parts of the root in its advanced stages. The diagnosis and treatment of ICR depend on the extent of the resorption into the dentin. The treatment of advanced ICR is challenging, and these teeth have poor prognosis.

Hithersay suggested that ECR lesions are aseptic and only become secondarily invaded with bacteria. However, Tronstad1 stated that ECR lesions were stimulated and sustained by microorganisms in the dentinal tubules and gingival sulcus and therefore were inflammatory in nature. A molecular study on the expression of bacteria-induced toll-like receptors in ECR lesions appears to support a bacterial aetiology.

Causes: Various predisposing factors basically include either physical (orthodontic treatment, segmental orthognathic surgery, transplanted teeth, trauma, bruxism, guided tissue regeneration) or chemical trauma (intracoronal bleaching, bone grafting, tetracycline conditioning of root) and periodontal treatment (including deep root scaling and planning have also been cited [19].

Clinical features: The condition is usually painless unless there is superimposed secondary infection when pulpal or periodontal symptoms may arise. Invasive cervical resorption has been and continues to be, misdiagnosed as a form of internal resorption.

Heithersay G.S.[20] has proposed a clinical classification of invasive cervical resorption depending on the amount of destruction:

Class 1; A small invasive resorptive lesion near the cervical area with shallow penetration into dentin,

Class 2; A well-defined invasive resorptive lesion that has penetrated close to the coronal pulp chamber but shows little or no extension into the radicular dentin,

Class 3; A deeper invasion of dentin by resorbing tissue, not only involving the coronal dentin but also extending into the coronal third of the root and

Class 4; A large, invasive resorptive process that has extended beyond the coronal third of the root

In the most advanced type of lesion (class 4 lesions), additional resorption channels are created. The channels burrow deep into the dentin and later interconnect more apically with the periodontal ligament through communicating channels.

ICR expands first coronally and thereafter apically, encircling the root canal. The pulp usually remains unperforated and healthy (uninflamed) because of the presence of a non-mineralized predentin layer. However, in long standing lesions, the root canal may be perforated by the advancing resorptive lesion (21)

Radiographic features: Range from well-delineated to irregularly bordered mottled radiolucencies, which can be confused with dental caries.

Treatment: If an invasive cervical resorption has been diagnosed at a Class 1 or Class 2 stage, the resorptive tissue is fibro-vascular in character and the pulp is walled off by a protective pre-dentine and dentine barrier. Traditional methods of treatment consist of curetting the active tissue from the resorption cavity and restoring the defect with a suitable restorative material. An alternative method, which utilizes the topical application of 90% aqueous trichloracetic acid, curettage and restoration. TCA is a very aggressive acid, and unplanned contact with healthy soft tissue during treatment causes severe chemical burns. When used on the skin, 50% TCA was found to be a highly errating and unpredictable agent, producing hypertrophic scarring and full-thickness skin loss [23].

When invasive cervical resorptions are diagnosed at the Class 3 and Class 4 stage of development, treatment poses far greater challenges due to the infiltrative and fibro-osseous characteristics of the resorptive lesion. Ectopic bone-like deposits can be observed both within the lesion and at the interface with resorbed dentine.

In long standing advanced ICR lesions (class3 or 4), it is very difficult to remove all the resorptive tissue because of the circumferential and coronal-apical extension. In addition, a significant amount of bone must be sacrificed, which endangers the periodontal prognosis.

Surgical management: Surgical treatment of varying degrees or invasive cervical treatment has generally involved periodontal flap reflection, curettage, restoration of the defect with amalgam, composite resin or glass-ionomer cement and repositioning the flap to its original position[22]

Heithersay reported a 100% success rate in the surgical treatment of classes 1 and 2 ICR lesions, whereas the success rate was 77.8% for class 3 lesions and only 12.5% for class 4 lesions. He concluded that classes 1–3 were treatable, whereas Clss 4 lesions were not amenable to treatment. He suggested that class 4 cases would benefit from alternative treatment options such as extraction and replacement with an implant.

d) Invasive radicular resorption.

II. Conclusion

Early diagnosis, correct case selection, an appropriate restorative/regenerative material and proper treatment are essential for long-term retention of the tooth with root resorption.

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