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Root Resorption: A Comprehensive Review of Etiology, Classification, Diagnosis, and Management Strategies

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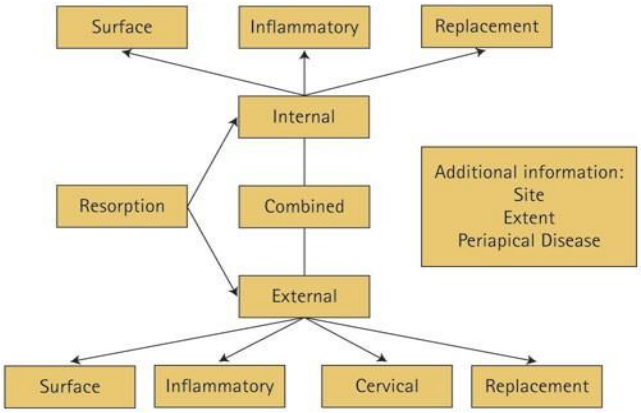
Abstract
Rootresorptionisacomplexpathologicalprocesscharac terizedbytheloss of dental hard tissues—cementum, dentin, and occasionally bone—due to the activity of clastic cells. It can be broadly classified into internal and externaltypes,eachwithdistinctetiologies,histopatholo gicalfeatures,and clinical implications. While internal root resorption (IRR) originates from the pulp and typically requires a partially vital pulp to sustain the process, external root resorption (ERR) arises from the periodontium and is commonly associated with trauma, orthodontic forces, inflammation, or systemic factors.
Accurate diagnosis, often relying on cone-beam computed tomography (CBCT),iscrucialfordifferentiatingIRRfromERRandf ordetermining the extent and severity of lesions. Timely and appropriate intervention is essential to arrest the resorptive process and preserve the affected tooth. Treatment approaches vary depending on the type and severity of resorption, ranging from conservative endodontic therapy and the use of bioactive materials like mineral trioxide aggregate (MTA), to surgical repair and, in extreme cases, extraction.
This review consolidates current literature on the etiology, classification, diagnosticmodalities,andevidence-basedmanagementofrootresorption, highlighting advances in biomaterials and imaging. Understanding the biological mechanisms and clinical presentations of root resorption enhances diagnostic precision and improves therapeutic outcomes, ensuring better long-term prognosis for affected teeth.

Keywords: Root resorption, internal root resorption, external root resorption, MTA, CBCT, clasticactivity, dentaltrauma, perforation repair

I. Introduction

Root resorption is a significant issue for endodontists. Timely diagnosis, addressing the underlying cause when possible, appropriate treatment, and strengthening the affected root are essential for achieving success. Resorption can be defined as either a physiological or a pathological condition that results inalossofsubstancefromatissue[1].Resorptionmayresult inthelossofdentin, cementum and or bone.Root resorption refers to the loss of hard dental tissues (dentin or cementum, and even alveolar bone) due to the action of clastic cells (often called odontoclasts, analogous to osteoclasts)[2]. In primary (deciduous) teeth, root resorption is a normal physiological process allowing exfoliation.
However,inpermanentteethanysignificantresorptionis considered pathological.

Classification



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SchematicclassificationbaseduponAndreasen'sdescriptionsofresorption[3]

Internaltoothresorption	Externaltoothresorption
Internalsurfaceresorption	Externalsurfaceresorption
Internalinflammatoryresorption	Externalinflammatoryresorption Apical and Lateral
Internalreplacementresorption	Externalreplacementresorption External invasive resorption External pressure resorption Orthodontic resorption Physiological resorption Idiopathicresorption

Etiology

Root resorption is multifactorial, and its etiology can be understood by consideringtheinitiatingfactorsthatremovethe"protective"layersofthetooth andtriggerclasticcellactivity.Undernormalconditions, twoprotectivelayers– predentin internally (unmineralized dentin lining the pulp chamber) and precementumexternally(unmineralizedcementummatrixontherootsurface) – prevent clastic cells from adhering to mineralized tissues [4].Any factor that damages or alters these protective layers can initiate resorption by allowing odontoclasts to bind to dentin or cementum.

Keyetiologicalfactorsinclude:

1. Dentaltrauma
2. PulpalInfectionandInflammation
3. OrthodonticForces
4. PeriodontalDisease
5. SystemicandGeneticFactors
6. PhysiologicalResorptionofPrimaryTeeth

MechanismofAction

Root resorption is a cell-mediated process involving odontoclasts (and osteoclasts), similar to bone resorption. These multinucleated cells resorb mineralized tissues under the influence of RANKL (ReceptorActivator of NuclearfactorKappa-BLigand)andothercytokineslikeTNF-α,IL-1β,andM-CSF[5].

- Protectivecementumtypicallypreventsclastic attachment;itsdamage initiates resorption.
- Pulpalinflammationincreasesbloodflowandcytokinerelease,attracting clastic cells.
- Inreplacementresorption,PDLisdestroyedanddbonereplacestheroot surface directly.

Types of tooth resorptionInternalToothResorption

InternalresorptionwasfirstreportedbyBellin1830.PinktoothofMummery (1920), so called due to the presence of apink discoloration on the crown, is named after the anatomist Mummery. The process is initiated by a variety of stimuli such as trauma, pulpotomy,extreme heat produced during cutting of dentin , chronic inflammation of the pulp following caries perpetuated bybacterial factors, cracked tooth, tooth transplantation, andorthodontic treatment[6].

1. InternalSurfaceResorption

Internal surface resorption is seen in regions where revascularisation takes place, such as the apical portion of a luxated tooth's root canal undergoing treatment and the fracture lines of root fractures[7]. Radiographically, there appearstobeatemporarywideningoftherootcanal.Notreatmentisrequired except periodic observation.

2. InternalInflammatoryResorption

Internalinflammatoryresorptionisdefinedasaninflammatoryprocesswithina section of the pulp/root canal that results in loss of dentin commencing at the root canal wall and progressing towards the cementum[1].Internal inflammatory resorptioncommencefollowingastimulus(inflammation)withtheelimination of inhibitory mechanism, that is, the loss or alteration of the protective (predentin/odontoblastic) layer and pulpal invasion by multi-nuclear giant cells/odontoclasts/dentinoclasts . Histologically, internal dental resorption is characterizedbyresorptionlacunaecontainingmultinucleatedcellsand surrounding reparative connective tissue[8].

Clinically, in such teeth one will find pulp necrosis in the pulp chamber and possibly in the root canal to a level somewhere coronally to the resorption lacuna.Theresorptiveareaandtherootcanalapicaltothisareawillcontain vital tissue.Aresorptive process may occasionally lead to communication between the root canal and the periodontal ligament[9].

The presentation of symptoms depends on the stage of resorptive process.Secondarytosymptomsfollowedbyapicalperiodontitis,patientstend to visit the dentist . Radiographically , a characteristic oval shaped increase in the size of pulp chamber is observed. There is an evident area of resorption or the presence of resorptive lacunae . The communication could also be an extension of external resorption.

3. InternalReplacementResorption

Replacement type of internal resorption results from a low grade irritation of pulpal tissues such as chronic irreversible pulpitis or partial necrosis

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usually localizedtoasmallareaintherootcanal.Thepulpchamber showsanirregular enlargement,accompaniedbyadisruptioninthetypicalcanalspaceseenonX- rays. Typically asymptomatic, affected teeth generally react normally to pulp testing unless the resorptive process leads to perforation in the crown or root.
Mostly secondary to irreversible pulpitis or partial necrosis. low grade chronic infection produces more of a reactive lesion bringing about deposition of metaplastictissueresemblingboneorcementumresembling cancellousbone[6].
Clinically,normalappearanceoftoothbutitmaybediscoloredorpresentwith slight pink hue occurring only if resorptive process has extended into pulp chamber.
Radiographically,anirregularenlargementofpulpspace withextensioninto dentin towards cementum.
Managementofinternalresorption

Non-PerforatingIRR	PerforatingIRR
AccessandDebridement:Removepulptissue; use ultrasonic irrigation withNaOCl.	InitialCleaning:Cautiousirrigationto avoid extrusion. PerforationRepair:

Intracanal Medicament: Calciumhydroxidefor1–2weekstohaltclasticactivity. Obturation:Thermoplasticizedgutta-percha or MTA; warm verticalcompaction preferred for irregulardefects. Restoration:Cuspalcoveragetoprevent fracture.	Orthograde repair: WithMTA/Biodentineusingaresorbablecollagen matrix. Surgicalrepair:Indicatedwhendefect is large, inaccessible, orcrestal. Follow-up:Radiographs/CBCTtoconfirm healing; monitor forperiodontal health.

ExternalToothResorption
1. ExternalSurfaceResorption
Externalsurfaceresorptionisatransientphenomenonin whichtherootsurface undergoesspontaneousdestructionandrepair.Itisfound inallteeth,invarying degrees, and is likely to be a normal physiologic response.It is the least destructive form of external root resorption and is a selflimiting process[10].
Itoccursasaresponsetoindirectphysicalinjury,causedbyphysiologic function,tolocalizedareasofperiodontalligamentorcementumonroot surface.
Clinically,nosignificantsignsofexternalsurfaceresorptionaredetectableon the supragingival portion of the tooth.
Radiographically,itisusuallynotvisibleonradiographs becauseofitssmall size with normal lamina dura and PDL space[11].
2. Externalinflammatoryresorption
EIR is the most aggressive and destructive type of traumatic resorptions. EIR is associatedwiththeinfectedpulpandperiodontium.Itmoreoftendevelopsafter severe trauma and commonly starts with a vestibular surface of the root[4].
Iftheblood supplytotheaffectedtoothispreserved,osteoclastactivitytargets theexternalrootsurface,resultinginself-limitingresorptionakintotemporary apical breakdown. However, if the root canal is infected, microbial toxins can reach the resorption area through dentinal tubules, leading to inflammatory resorption. Resorptive lesions can be superficial or extend into deeper dentin, with odontoclasts and granulation tissue present at the site of resorption.

Externalinflammatoryresorptioncanoccurintwolocations—eitherattheapex of the tooth root or at any other location along the length of the tooth root.
Thesearedesignatedaseither:
(a)“ExternalApicalInflammatoryResorption”,or(b)“ExternalLateral Inflammatory Resorption”.
Clinically,Externalinflammatoryresorptionrequiresthefollowingtwothings to occur:EIR is primarily asymptomatic, while rarely observed clinical manifestationscanbesimilartothesignsofthepulpallesion,Whenresorption leads to additional pulpal necrosis, the crown of the tooth may become discolored, raising aesthetic issues for patients[12]. The clinical appearance can resemblethatofperiapicalperiodontitis,characterizedbyalackofresponseto sensitivity tests and tenderness when palpated or percussed.
Radiographically,externalinflammatoryrootresorptionincreasesthewidthof the periodontal area, resulting in the loss of tooth structure and the adjacent lamina dura, leaving the mesial and distal edges poorly defined.
3. Externalreplacementresorption
External replacement resorption is the process where cementum and dentin are resorbedandreplacedbybone.typicallyoccursfollowinganinjurytothePDL and/orexternalrootsurface(i.e.thecementum).Itisverycommonaftersevere luxation injuries such as avulsion and intrusion.
4. Externalinvasiveresorption
Fibrovasculartissueinfiltrationintothetooth'scervicalr

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egionidentifiesit,and it originates from cells in the periodontal ligament. Dentin, enamel, and cementum are progressively reabsorbed by this tissue.

theprimaryobjectiveremainsthecompleteremovalofresorptivetissueandthe restoration of the affected area
Managementofexternalrootresorptionandsubtypes

Surface resorption	Inflammatory Resorption	Cervical Resorption	Replacement Resorption (Ankylosis)
Management: Notreatment usuallyrequired; monitor radiographically	Cause:Trauma, infection,orthodontic treatment Management:Endodontic therapy(toremove necrotic/infectedpulp)	Class1Surgical ornon-surgical removalof resorptivetissue	Cause:Severe trauma,avulsion withprolongeddry time Management:No definitive
Prognosis: Excellent	Use of calcium hydroxide as an intracanalmedicamentto inhibit osteoclastic activity Final obturation with thermoplastictechniques Prognosis Good if diagnosed early	Restorationwith MTA, GIC, or composite resin Class 3–4: Complex; may need surgical intervention or extraction in severe cases Adjuncts:Useof trichloroacetic acid (TCA) for devitalization of resorptive tissue Prognosis: Depends on extent; guardedinadvancedcases	treatment to stop the process Decoronation may be considered in growingpatientsto preserve alveolar bone Prosthetic planning or implantsinlong term Prognosis:Poor; progressive and irreversible

Recenttrendsinpreventionandmanagementofrootresorption:

- ArtificialIntelligenceinCBCTforautomaticlesiondetection.
- Photobiomodulationtherapytoregulateinflammatorypathwaysinroot resorption.
- 3Dprintingandguidedsurgeryforflapdesignandaccessinapical resorption cases.
- Researchonresorption-specificbiomarkers(e.g.,dentinmatrixprotein1 -DMP1)forearlydetection.

II. Conclusion

Root resorption represents a multifaceted pathological condition that can compromise tooth structure, function, and longevity if not diagnosed and managedpromptly.Itscomplexetiology—rangingfromtraumaandinfectionto orthodontic forces and systemic influences—demands a nuanced understanding of the biological and mechanical factors involved.Accurate classification into internal and external types, alongside appropriate diagnostic modalities such as CBCT, is critical in guiding timely and effective treatment strategies.

Management must be tailored to the type and extent of resorption, with a focus onarrestingtheresorptiveprocess,preservingtoothvitalitywherepossible,and ensuring long-term prognosis through biocompatible materials like MTAand Biodentine.Advances in digital imaging, artificial intelligence, guided surgery, and biomaterial sciences continue to enhance early detection and therapeutic precision.

Ultimately, clinical vigilance, interdisciplinary coordination, and a patient-specificapproachremaincentraltosuccessfuloutcomes. Continuedresearch and innovation are essential to further improve diagnostic accuracy, develop targeted therapies, and mitigate the long-term impacts of this often silent but progressive dental pathology.

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