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Invasive cervical Resorption: A Review

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Abstract
Invasive cervical resorption (ICR) is loss of dental hard tissue due to odontoclastic action and usually begins at the cervical region of the root. The management of ICR is challenging due to its complexity and aggressive nature. Accurate diagnosis and appropriate treatment are critical to effective management. This article reviews the etiology, predisposing factors, diagnosis, and management of ICR.

Keywords: hyperplastic tooth resorption, invasive cervical resorption

Introduction
Root resorption is the loss of hard dental tissue (i.e., cementum and dentin) as a result of odontoclastic action [1]. According to Lindskog, resorption may be classified as trauma-induced tooth resorption, infection-induced tooth resorption and hyperplastic invasive resorption [2]. Hyperplastic invasive resorption is characterized by invasive nature of resorptive tissue and interconnections exist between these and the periodontal tissues through small infiltrative channels in dentin. Therefore such resorptions present a therapeutic challenge as complete elimination of the resorptive tissue is essential, else the lesion may recur. Hyperplastic resorptions may have a pulpal (internal) or a periodontal origin (external) and may be subdivided into internal replacement (invasive), invasive coronal, invasive cervical and invasive radicular resorptions [3].

Invasive cervical resorption (ICR) is an aggressive form of external root resorption, termed so by Heithersay due to its cervical location and invasive nature, which may occur in any tooth in the permanent dentition [4]. Other terms used to describe ICR include odontoclastoma [5], peripheral cervical resorption [6], extracanal invasive resorption [7], supraosseous extracanal invasive resorption [8], peripheral inflammatory root resorption [9], subepithelial external root resorption [1], external cervical resorption [1], idiopathic external resorption, late cervical resorption, cervical external resorption and periodontal infection resorption [10].

ICR usually begins at, or below the epithelial attachment and above the ridge crest. The lesion is therefore not always located at the cervical area, but may be present anywhere along the root, related to the level of marginal tissues and pocket depth [1, 2]. Accurate diagnosis and prompt treatment is necessary as the lesion is aggressive leading to a considerable loss of tooth structure.

Etiology
The etiology of ICR is poorly understood. It is periodontally derived, the pulp has no role in its etiology [2, 3]. The pulp chamber and root canals may get involved as the lesion progresses. ICR occurs when the protective pre cementum layer is damaged or deficient below the epithelial attachment, which exposes the denuded root areas to osteoclasts that progressively resorb the root [2, 3]. Several factors have been suggested to predispose to the occurrence of this lesion. These include orthodontic treatment, trauma, intracoronal bleaching, surgical procedures, periodontal therapy, bruxism, intracoronal restorations, developmental defects, delayed eruption, interproximal stripping and systemic diseases [1, 11]. Of these, orthodontic treatment, trauma, intracoronal bleaching are the major contributory factors [11]. In some cases a combination of the above may be present and yet in several cases, the etiology may be unknown.

Maxillary canines, maxillary central incisors and mandibular first molars are the teeth commonly affected by ICR following orthodontic treatment. Excessive orthodontic forces at the cervical region can result in necrosis adjacent to exposed dentin of root which might stimulate differentiation of odontoclasts that resorb the exposed root dentin [11].

Bleaching agents such as 30% hydrogen peroxide used intracoronally may penetrate dentin, alter root surface and irritate the periodontal ligament and surrounding tissues. Sealing the coronal portion of the root canal orifice with a barrier of glass ionomer cement or intermediate restorative material can help prevent cervical leakage of the bleaching agent. Use of sodium perborate alone rather than in combination with hydrogen peroxide, and dressing the access cavity with calcium hydroxide for a week following bleaching, seem to decrease the possibility of resorption [1, 12, 13].

Dental trauma, especially luxation and avulsion injuries, are major predisposing factor for cervical resorption [14]. Trauma can cause ICR indirectly. Developmental defects can be found in cervical region of unerupted permanent successors as a result of intrusion of primary teeth. Splints applied following trauma, especially interdental wiring, might also potentially damage cementoenamel junction (CEJ). Repositioning of luxated teeth must be done with utmost care to minimize damage to CEJ [1, 14].

Surgical procedures and periodontal therapy may damage cervical area of teeth, thus predisposing to ICR. Removal of adjacent impacted or erupted third molars or supernumerary

teeth, transplantation of canine, surgical exposure of unerupted teeth, periodontal surgery for root amputation etc. are potentially damaging [1]. Cases of ICR in patients previously treated with tetracycline root-conditioning and guided tissue regeneration have been reported [15, 16].

Apart from these local factors, systemic conditions may also play a role in ICR. Conditions like hyperoxaluria, oxalosis [17], normocalcemic hypercalciuria[18] and nephrolithiasis have been suggested to cause root resorption. Certain individuals might have a genetic predisposition to ICR [19].

Classification

According to extent of lesion within the tooth, ICR has been classified by Heithersay [3] as:

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 radicular dentin

Class 3: A deeper invasion of dentin by resorbing tissue, not only involving the coronal dentin but also extending at least to the coronal third of the root

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

Diagnosis

Clinical Features

ICR is an insidious resorptive process that may occur in any permanent tooth [2]. In the absence of treatment, there is progressive destruction of tooth structure. Initially it is painless, but the patient may become symptomatic when pulpal or periodontal tissues are secondarily infected. Resorption of coronal dentine and enamel creates an appearance of 'pink tooth' in the cervical area as the vascular resorptive tissue becomes visible through thin enamel [2, 4]. Sometimes there may be no clinical signs and the lesion may be detected by routine radiographs. The involved tooth usually responds positively to vitality tests until the lesion has advanced sufficiently to cause pulpal involvement. Profuse bleeding may be present spontaneously or on probing. The edges around the resorptive cavity are sharp and thinned out [4].

Radiographic features

ICR is usually detected as chance radiographic finding as the tooth is usually asymptomatic. The appearance may vary from asymmetrically located radiolucency with irregular margins in cervical or proximal areas to uniformly round radiolucency centered over the root. Early lesions are usually radiolucent, however in advanced cases a mottled appearance may be seen owing to fibro-osseous nature of the lesion [1, 4]. As the lesion is external, root canal outline is generally visible and intact. Advanced imaging modalities such as cone beam computed tomography (CBCT) prove to be valuable in diagnosis and treatment planning [1].

Differential diagnosis

ICR may be mistaken for subgingival caries, which usually feels sticky on probing, in contrast with the hard feel of a resorptive defect [1]. Root caries is predominantly seen on the root surface of teeth that are exposed to oral cavity and is more prevalent in people above 60 years or with xerostomia [20]. Root caries generally does not appear as a 'pink spot'. Further, probing the resorptive lesion and the periodontal pocket will cause profuse bleeding.

ICR can be confused with internal resorption because teeth with internal resorption in coronal area may present with 'pink spot'. Internal resorption originates from pulp tissue and distorts or widens the root canal outline. In contrast, the outline of root canal is usually intact in an external resorptive lesion[2]. For distinguishing between the two through conventional radiography, tube shift technique may be used. When the angulation of the radiograph is changed, internal resorption remains centered on the root canal system regardless of the angle of the radiograph exposure, whereas an external defect will either move in the same (lingual/ palatal) or in the opposite (labial) direction of the x-ray tube [1, 2]. CBCT is invaluable in such conditions.

External inflammatory root resorption

EIRR is another condition to be differentiated from ICR. Radiographically, EIRR presents with a bowl like radiolucency on bone and root surfaces. EIRR, being of pulpal origin, becomes arrested following root canal treatment; however, ICR is of periodontal origin[2].

Cervical burnout

Has to be considered in the differential diagnosis. Cervical burnout is a radiographic illusion that appears on periapical radiographs due to the cervical root configuration and exposure parameters. It is seen as a radiolucent band around neck of the tooth with diffuse inner borders but with intact tooth edges. Taking a second radiograph at different angle helps to rule out this phenomenon [21].

Management

There are generally three choices for treatment of a case of ICR: no treatment at present and extraction when the tooth becomes symptomatic; immediate extraction; or access, debridement, and restoration of the resorptive lesion [22]. Treatment depends on severity of the lesion, location, pulpal involvement, and restorability of the tooth. Several treatment regimes have been suggested: intentional replantation, forced orthodontic eruption, guided tissue regeneration, and treatment by internal approach [1]. Treatment objectives include arresting the resorptive process, restoring damaged root surface, preventing further resorption and improving esthetics of the tooth. Heithersay has recommended treatment of lesions categorized as classes 1–3. Class 4 has been reported to have a higher rate of failure and extraction may be the only viable option [23].

Thorough curettage of the granulation tissue in the resorptive defect is essential as remnants may lead to recurrence [23]. Persistence of bleeding after curettage indicates blood supply apical or lateral to the cavity. Advanced and long-standing lesions may contain fibro-osseous tissue. Magnification and illumination are essential to distinguish the fibro-osseous tissue from the underlying dentin and its complete removal is critical to prevent recurrence. Topical application of 90% aqueous solution of trichloroacetic acid (TCA) has been recommended for removal of resorptive tissue. It causes coagulation necrosis of the resorptive tissue and infiltrates small channels and recesses that may be inaccessible by mechanical instrumentation [1, 23].

Endodontic treatment is necessary only if the resorption invades the pulp space or is very close to it, i.e., in Class 2 and 3 cases. Use of corticosteroid/antibiotic paste such as Ledermix paste as an intracanal medicament has been recommended due to its anti-clastic activity [2, 23]. Dressing root canals with calcitonin has been suggested to prevent further progression of resorption. Calcitonin has inhibitory action on osteoclasts and odontoclast activity and suppresses inflammation [24]. Iontophoretic delivery of calcitonin to the resorptive lesion via dentinal tubules is a novel technique that could make conservative management possible. Osteoprotegerin and bisphosphonates are other potential medicaments that could be used with this technique [25].

An internal treatment approach is preferable and possible when the external surface of the tooth remains grossly intact and the lesion is accessible. It is not possible to totally eliminate the resorptive tissue from inside the tooth; therefore careful mechanical and chemical debridement is necessary to arrest the resorptive process. Use of rubber dam provides better isolation and visualization [22].

Orthodontic extrusion can be employed in some Class 3 cases to improve access to the defect and to establish a supragingival margin for the restoration. Further it provides a more ideal bony and gingival architecture. (12) The tooth is extruded using light wire technique, over a period of 4–6 weeks, followed by splinting, pericision, gingivoplasty and restoration [2].

A surgical approach involving a full thickness periosteal flap, curettage and restoration is often necessary for access and complete debridement of the lesion [2]. When materials like amalgam, composite resin or glass ionomer cement are used, periodontal re-attachment cannot be expected. But it might be possible with mineral trioxide aggregate (MTA) [2]. Alternatively the flap can be apically positioned to the base of the resorption repair. Orthodontic extrusion can be done to improve the gingival contour. Application of membranes such as Goretek membrane, Emdogain or bone graft materials may promote regeneration of localized periodontal and bone lesions [10]. The successful use of platelet rich fibrin (PRF) and hydroxyapatite in periodontal defect associated with ICR has been reported [26].

Several materials have been used to restore the resorptive defect after curettage, and many materials continue to be tested for use in such conditions. Amalgam, glass ionomer cement (GIC) and composite resins have been traditionally used for restoration. However dentin that has been treated with TCA is severely demineralized and therefore not suitable for bonded restorations like GIC or composite [22]. The dentin must be refreshed with a bur before bonding procedures. Resin modified glass ionomer cement (RMGIC) and composite resin have been used because they are stronger, bond to tooth structure, and are relatively stable in oral cavity [22].

Newer bioactive materials have been successfully used to treat cases of ICR. MTA has been recommended because of its biocompatibility, good sealing ability, moisture tolerance, and the environment it creates for favorable hard-tissue healing [27]. However, surface of MTA is rough and development of subgingival plaque could be a problem. MTA lacks strength and hardness; therefore it cannot reinforce the tooth structure and may be partially scraped off during mechanical instrumentation of root surface [28]. Biodentine has been used in ICR because it acts as a substitute for dentin, has excellent biocompatibility, sealing ability, favors mineralization, induces repair of the periodontium and new cementum formation over the material [29]. Calcium enriched mixture (CEM cement), a novel bioactive material, has shown favorable result in repair of resorptive defect as is able to induce hard tissue formation, has shorter setting time than MTA, good handling characteristics, and produces no tooth staining [30].

Combinations of different materials have also been used. A sandwich technique involving use of MTA, GIC and composite has been reported [27]. A 'reverse sandwich technique' has been used in another case where a layer of microfilled composite is placed in the interior of the lesion, and RMGIC was used to build-up rest of the defect[28]. Geristore is another resin modified glass ionomer that has been used in the management of ICR [20].

Conclusion

Invasive cervical resorption is an aggressive form of tooth resorption. Its etiology is poorly understood. Several predisposing factors have been identified which contribute to development of the lesion. Prompt diagnosis and appropriate treatment are critical for long-term prognosis.

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