Clinical and scanning electron microscopic features of invasive cervical resorption in a maxillary molar

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This case report presents clinical and morphological features of severe invasive cervical resorption (ICR) involving a maxillary first molar. Surface morphology of the ICR defect that extended into enamel was studied using a scanning electron microscope. The topography and morphology of resorbed enamel and pulpal surfaces are described. The pulpal space was characterized by proliferation of fibrovascular tissue invaded by a number of ≥15-μm-large dentinoclastlike cells. The vertical orientation and hexagonal geometric outlines of enamel rods indicated that the resorptive defect had extended into the outer surface of enamel. Preferential odontoclastic dissolution of interprismatic enamel was noted. Deposits of a cementumlike substance were noticed on enamel surfaces, indicating that resorptive process was coupled with repair. Complete destruction of dentin was associated with no response by the tooth to cold stimulus, supporting the hydrodynamic theory of dentinal sensitivity. The results of this study indicate that in addition to predentin and cementum, the outer surface enamel may be resistant to the resorptive process as well. **(Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103:e49-e54)**

Invasive cervical resorption (ICR) is characterized by the invasion of the cervical region of the root by fibrovascular tissue that progressively resorbs dentine, enamel, and cementum.[1] Most cases of ICR are reported in anterior teeth,[2-5] probably due to ease of detection. Only a limited number of articles dealing with ICR in posterior teeth have been published in the dental literature over the past 10 years.[6,7]

There seems to be no consensus on the nomenclature used for this resorptive defect. It has been variously termed in literature as progressive intradental resorption,[8] invasive resorption,[9] ICR,[10] external cervical resorption,[4] periodontal infection root resorption,[11] and external invasive resorption.[7]

With a majority of cases, the etiology of ICR has been designated as idiopathic, but damage to or presence of natural cementum defects in the normally protective cementum layer appears to be prerequisite for initiation of the process.[12] The common contributing factors include trauma, chronic periodontal inflammation, internal bleaching, and pressure from orthodontic tooth movement.[10,13] Generally, ICR is not believed to be related to pulpal pathology but appears to be the result of injury to the cervical attachment apparatus.[2,14]

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Scanning electron microscopic (SEM) examination has been used to describe the topographical features of dental hard tissue resorption in deciduous teeth[15,16] and internal resorption defects.[17,18] To our knowledge, SEM features of active ICR have not been reported in the literature. Furthermore, ultrastructural details of enamel resorption have been observed rarely in association with idiopathic human permanent tooth resorption.

The purpose of this case report is to present the clinical and radiographic features of ICR in a maxillary permanent molar. In addition, this case also reports more information about the topography of the enamel and pulpal tissue involved in the resorptive process as visualized by the SEM.

CASE REPORT

A 40-year-old Middle Eastern male was referred for evaluation after his dentist had incidentally diagnosed, from a routine periapical radiograph, a large radiolucent lesion in the crown of the maxillary right first molar. The medical history was unremarkable, and the patient denied any history of trauma to the involved area. The patient did not have any subjective complaints related to the involved tooth. Oral hygiene was poor and probing depths of 6 mm were noted in the distal interproximal area of the involved tooth. The tooth did not respond to repeated cold testing with CO_2 on all accessible surfaces, whereas adjacent and contralateral teeth gave positive responses to cold testing. A periapical radiograph revealed an irregular and multiloculated radiolucent area involving the crown of the tooth (Fig. 1). The tooth showed regions of rosy pink coloration in the crown. There was bone loss in the interdental crestal regions between the maxillary right first and second molars (Fig. 1).

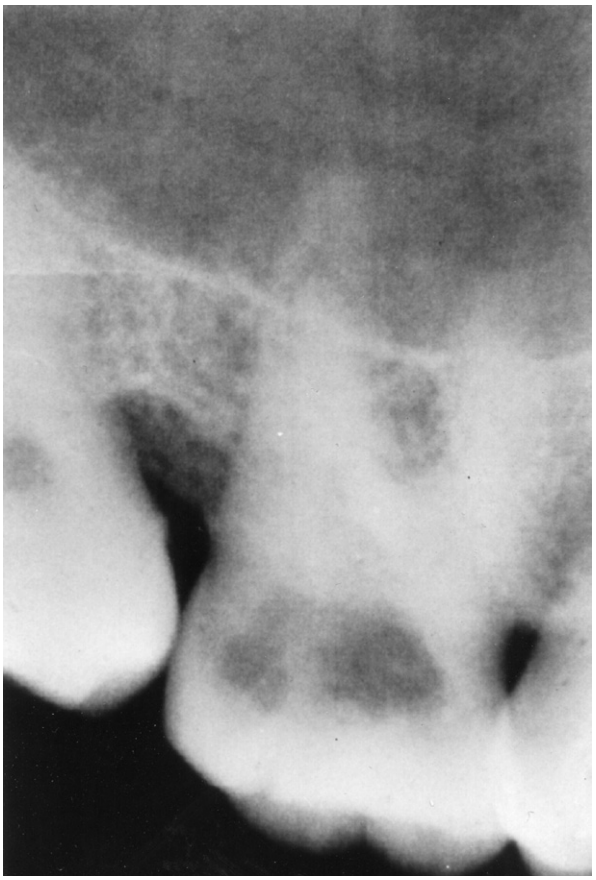

Fig. 1. Periapical radiograph of tooth 3 at initial presentation. A multilocular radiolucency in the crown and a loss of radiographic density in the distal interdental crestal region can be seen.

A diagnosis of ICR was made, and endodontic treatment was initiated after local anesthesia. A thin fissure bur in a high-speed handpiece was used to make cut lines on the occlusal surface corresponding to the quadrilateral shape of an access cavity. Care was taken to avoid damage to the inner pulpal tissue, just cutting through the enamel until the pulp tissue was identified by bleeding. Upon completing the cut lines, the inner table could then be gently fractured along the access outline with a thin-bladed excavator. In this manner, a 3 × 4–mm section of the roof of the pulp chamber was obtained for biopsy and immediately fixed in 10% solution of formalin. Upon completing access, a profuse hemorrhage was encountered, indicating the continued vitality of the pulp (Fig. 2). The crown of the tooth was found undermined by the resorptive defect, and the prognosis of the tooth was guarded. Root canal treatment on the tooth was initiated, but the patient failed to return for completion of root canal treatment.

The occlusal sample was dehydrated in a graded series of alcohol and CO_2 critical-point dried, mounted on an aluminum stub, and gold sputtered. The pulpal surface of the sample was examined using a JEOL JSM-T330A (Jeol Ltd, Tokyo, Japan) SEM.

Fig. 2. Profuse bleeding during access cavity preparation, indicating continued vitality of the pulp.

DISCUSSION

This case report describes an unusual presentation of ICR that extended to the outer enamel surface of the tooth. This offered an excellent opportunity to study the clinical and topographical features of ICR and the cells associated with the resorptive process.

In the present case, the causative factor was not proven, but it seems plausible that the inflammatory changes in the periodontium induced by the distal interproximal periodontal defect may have played a role in the initiation of the resorption. One may hypothesize that the resorption began on the distal cervical margin of the tooth (close to the cementoenamel junction) and progressed to involve the entire coronal dentine. This assumption is also supported by the close proximity of the periodontal defect to the distal terminus of the resorptive defect (Fig. 1). A number of reports have implicated periodontal disease and its treatment as one of the causative factors for ICR.[6,9,19] The possibility of a coincidental association between ICR and the presence of a periodontal defect in the neighboring gingival tissue cannot be entirely ruled out.

The treatment recommended for ICR usually consists of gaining surgical access to the lesion, removal of the fibrovascular tissue, and repair of the defect with a restorative material. Endodontic intervention is initiated when the lesion invades the pulpal space. A nonaggressive treatment consisting of periodontal debridement, oral hygiene instruction, and systemic metronidazole was found effective in treatment of multiple lesions of ICR.[6] The defects were repaired with woven bone, and new attachment was established after the woven bone was coated with a layer of acellular intrinsic fiber cementum. Teeth healing with woven bone formation would still exhibit radiographic features sim-

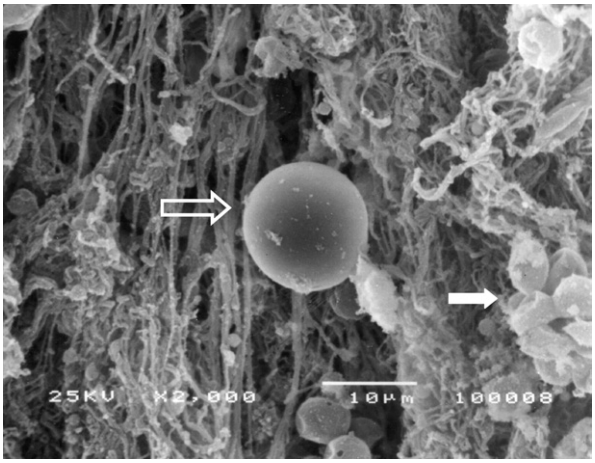

Fig. 3. A large, smooth-surfaced cell, probably belonging to the monocyte-phagocyte system can be seen in the center (*open arrow*). Background shows fibrovascular tissue, with abundant collagen fibrils. Discoid-shaped red blood cells indicate presence of hemorrhagic tissue (*filled arrows*).

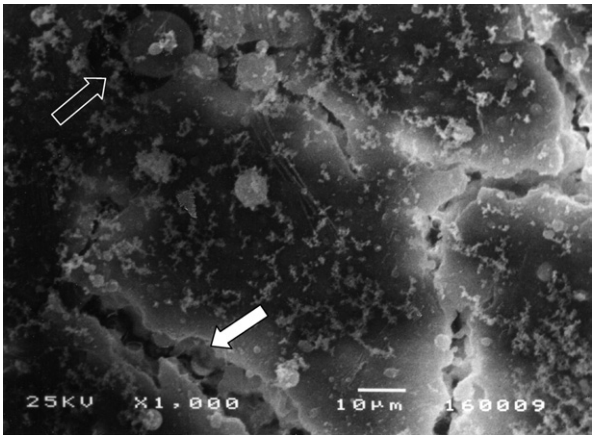

Fig. 4. Enamel surface affected by invasive cervical resorption featuring a flat surface associated with cemental deposition. Beneath the artifactual cracking cobbled appearance of enamel can be seen (*white arrow*). An odontoclastlike cell is seen within a typical resorption lacuna (*black arrow*).

ilar to progressive ICR. Therefore, teeth exhibiting signs of ICR may be in the healing stages and probably do not require any elective intervention.

The multilocular appearance of the resorptive defect in the present case is most likely due to a circumferential spread of the lesion around the pulp chamber, owing to the inhibitory effect of the unmineralized predentin.[20] Dentine is protected from resorption, initiated in the connective tissues, internally by the predentin and externally by the cementum. The root resorption is thought to occur whenever the protective coverings are damaged.[21,22] The resorption characteristically stops short of the pulp, leaving a narrow strip of dentine and predentin, which gives the lesion its typical multilocular radiographic appearance.[23]

An interesting observation in this case was the nonresponsiveness of the tooth to repeated cold tests with carbon dioxide. Recently, a case report on ICR described similar results with carbon dioxide cold testing and positive response to electric pulp test but did not provide any explanations.[7] Unfortunately, in the present case report electric pulp testing was not performed.

Many agree that the hydrodynamic theory of dentine sensitivity best explains the stimulus-response relations with most painful stimuli.[24-27] The theory of dentin sensitivity holds that pain is evoked by stimuli producing minute shifts in tubule fluid in intact dentine.[28] The complete resorption of dentin, as with this case, would lead to lack of response to cold testing. Therefore, the nonresponsiveness of the tooth to cold provides support for the hydrodynamic theory of dentin sensitivity, or at least lends credence to the notion that the presence of intact dentine is necessary for evoking pain upon application of cold stimulus.

In a case report describing ICR in anterior teeth, Coyle et al.[3] noted a variety of histological patterns. There were areas of fibroblastic proliferation lacking inflammation, some of which were cellular and others more fibrous in appearance. The SEM pictures of the resorptive tissue in this study also showed similar results (Fig. 3). The use of scanning electron microscopy has certain limitations because the characteristic morphological features in the nucleus and cytoplasm cannot be observed. Therefore, the individual cell size and surface morphological features were relied upon to distinguish between cells.

A striking feature observed in this study was the presence of a number of ≥15-µm-large, spherical, and smooth-surfaced cells (Fig. 3). Monocytes are precursors of osteoclasts and can vary from 16 to 20 µm in diameter.[29] It can be speculated that these cells were odontoclastic in nature, because similar cells were also found nested in resorption lacuna (Fig. 4). However, these cells were devoid of fimbriae or ruffled borders, which are indicative of osteoclastic cells. Odontoclasts with a round form supported by connective tissue have been reported in deciduous teeth undergoing resorption.[30] It seems more likely that the loss of fimbriae was an artifact, because cells such as lymphocytes present as mostly smooth-surfaced cells as a result of air-drying preparative procedure.[31] Nevertheless, the present study did not use the histochemical tartrate-resistant

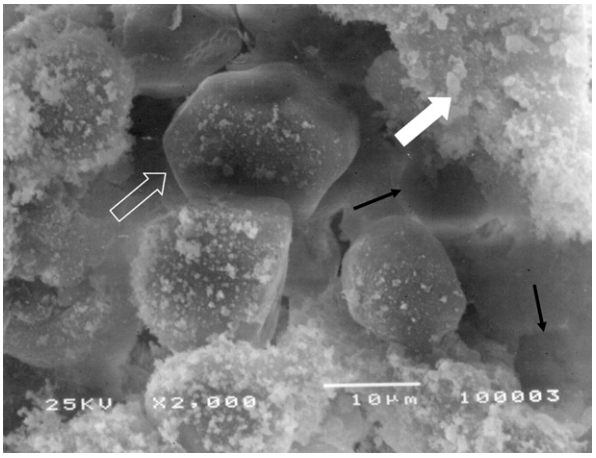

Fig. 5. Resorption front on enamel showing exposed enamel prisms some of which displayed perfect hexagonal geometry (*open arrow*). Invaginations matching the tapered ends of enamel rods can be observed on rodless surface enamel (*black arrows*). A large odontoclastic cell can be seen in the upper right corner of the picture (*white arrow*).

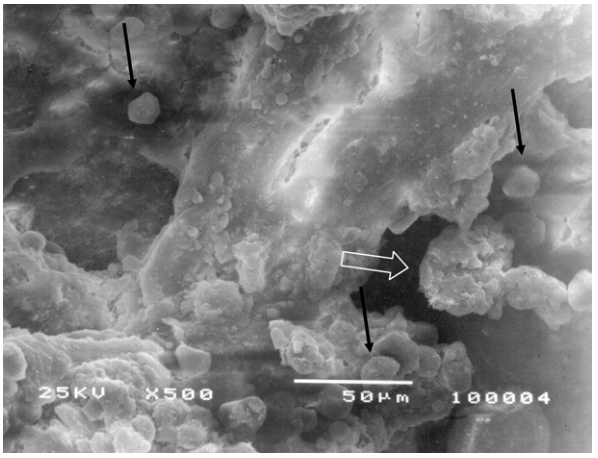

Fig. 6. Image of demineralized outer enamel exposed by resorptive process, revealing a complete destructuring of the enamel. Remnants of a few hexagonal enamel rods are seen (*black arrows*). Repetitive resorption phases have led to caving in of the enamel. Large chunks of mineralized globular materials can also be seen (*open arrow*).

acid phosphatase activity test, which is considered specific to odontoclasts; therefore, the exact nature of these cells remains unclear.

A number of round cells corresponding to the size of white blood cells were also encountered in the studied section. Scanning electron microscopy cannot differentiate-types of white cells by their surface structure. These cellular accumulations might represent foci of inflammation that have been infrequently encountered in histological examination of ICR.[3]

The fundamental organizational unit of enamel is the rods and interrod enamel. The enamel rod was first described as hexagonal in cross section, but recently this has changed because rods do not exhibit a consistent shape. The majority of rods observed in this study did not exhibit a regular geometry; however, a few displayed typical hexagonal or keyhole cross-sectional outlines (Fig. 5). A number of factors are known to affect the shape and size of enamel rods. Changes in the sectioning angle of enamel are usually accompanied by changes in prism size and spacing.[32] Although real prisms may be hexagonally spaced, enamel may produce prisms that are inherently compressed or extended. Recently, it has been shown that single enamel rods did not maintain the same outline throughout their path; arcade outlines predominated close to the dentinoenamel juction, with keyhole outlines at the enamel surface.[33] The sample used in this study exhibited many enamel rods with hexagonal cross-sectional outlines, indicating that the resorptive process had extended into the outer rodless enamel surface (Figs. 5 and 6).

The enamel layer is composed of a rod containing a (prismatic) layer sandwiched between thin rodless initial and final layers. The initial and final enamel layers are aprismatic, that is, they do not contain any rods.[34] In Fig. 5, invaginations matching the tapered ends of enamel rods can be observed next to the enamel prisms. This, as well as the vertical orientation of enamel rods, further proves that the resorptive process was in close proximity to the final rodless layer of enamel.

One interesting finding of this paper is the inability of the resorptive process to infiltrate into outer rodless surface enamel. Therefore, it seems that in addition to cementum and predentin the outer surface enamel is also resistant to odontoclastic resorption. This is supported by the fact that in all the reported cases the invasive cervical resorption defect is covered with a thin layer of intact enamel.[2-8] Enamel that participates in the formation of cemento-enamel junction primarily consists of outer surface rodless enamel. Therefore, it plays an equally important role, along with cementum, in protecting the dentine from resorptive processes. In essence, the antiresorptive effect of predentine and the outer surface enamel give the lesion its characteristic appearance. The resorption characteristically stops short of the pulp, leaving a narrow strip of dentine and predentin, giving the lesion its irregular radiographic appearance. On the enamel side, the resorption stops short of the outer surface enamel leaving a thin layer of outer rodless enamel which transmits the pinkish coloration of the resorptive tissue.

The basic organizational pattern of mammalian enamel is described as cylindrical rods embedded in the interrod enamel.[34] The prisms in the resorbed enamel surface were brought into prominence according to unequal rates of removal of the differently inclined and packed enamel crystals within them. It is interesting to note that the dissolution of interrod enamel was preceded by the dissolution of rod enamel despite the fact that rod and interrod enamel are of identical composition and differ only in the orientation of their crystallites. This observation confirms previous reports, indicating that removal of enamel by odontoclasts depends upon the orientation of enamel structures rather than the different degrees of mineralization.[35]

The dissolution of enamel noted in this study corresponds with the type II acid etch pattern (cobblestone) reported by Galil and Wright.[36] The pattern observed in this study contrasts with that of carious lesions, in which preferential dissolution of prisms with hollowing out of prism cores has been reported.[37] However, it is realized that enamel crystals would behave differently according to whether the acid secreted from odontoclasts penetrated in a direction parallel or perpendicular to the prism lines.[38] This pattern of dissolution could also be due to a higher solubility of the intraprismatic mineral in the outer or surface enamel.[39]

This case highlights a fundamental difference between carious and resorptive processes. It has been reported that if the supportive layer of dentine is destroyed by caries or improper cavity preparation, the unsupported enamel fractures easily.[34] However, in this study—despite advanced destruction of dentine and enamel—the crown of the tooth neither crumbled nor showed any perforations. These differences may be due to deposition of a cementumlike tissue during resorptive process, which increases in width and appears to undergo mineralization as time progresses.[40]

The SEM pictures of section obtained in this study revealed a random pattern of resorptive defects. The patterns ranged from several irregular surfaces to smooth surfaces that appeared flat (Fig. 4). The presence of flat surfaces in a resorptive defect is probably due to deposition of cementum on the caved surfaces, which made them relatively flat. According to Sahara et al.,[41] regardless of the type of matrix of dental hard tissues, tooth resorption may be coupled with tooth repair. It has been suggested that mononuclear cells and an organic thin layer found on the previously resorbed enamel surface play an important role in the repair process initiated after resorption of the enamel in primary teeth.[41]

The results of this study indicate that topographical features of ICR display a complex array of patterns. Unlike carious lesions, advanced stages of ICR may not cause collapse of enamel infrastructure because of continued reinforcement by deposition of a cementumlike substance. In the small area of outer enamel studied in this case report, the dissolution of enamel followed a pattern quite opposite to one reported for acid etching and carious dissolution of enamel. The case report suggests periodontal disease as a causative factor of ICR. With ICR, the odontoclasts first remove the coronal dentin and then resorb large areas of enamel. In advanced stages of ICR, despite the presence of vital pulp, teeth do not respond to cold testing due to complete destruction of the dentinal complex. The occlusal biopsy method described in this case report can be effectively used for studying other pulpal pathologies. One of the interesting results revealed by this study indicates that in addition to predentin and cementum, the outer surface enamel may be resistant to the resorptive process as well. Although not a conclusion of this study, further work needs to be done in differentiating between progressive and healing ICR lesions.

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