

Characteristics and treatment of invasive cervical resorption in vital teeth. A narrative review and a report of two cases

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In brief

Discusses diagnostic methods for invasive cervical resorption (ICR) in vital teeth.

Details treatment approaches to such cases.

Suggests suitable dental materials for the treatment of ICR.

Invasive cervical resorption (ICR) is a significant defect of the root surface where the hard dental tissues are undermined and become translucent due to the resorptive granulomatous tissue. It is mainly detected on radiographs. The radiographic appearance of ICR is asymmetrical radiolucency with irregular margins and an unchanged root canal. The purpose of this study is to present a comprehensive review together with the characteristics and the treatment of ICR through two cases which were followed-up for 36 months. In both cases the teeth were vital and the management consisted of raising a flap, granulomatous tissue removal, thorough debridement, restoration of the resorptive defect by placing a suitable filling material and follow-up examinations. Our results indicate that the specific treatment protocol has very positive outcomes. After 36 months pulpal vitality tests revealed a healthy pulp, and clinical and radiographic examination confirmed a stable periodontal condition.

Introduction

Root resorption is a physiologic or a pathologic process of the loss of hard dental tissues, that is, cementum and dentin, due to odontoclastic (cementoclastic, dentinoclastic and ameloclastic) activity.^{1,2} The physiologic process concerns primary teeth, it is desirable and leads to root absorption and exfoliation of deciduous teeth allowing the eruption of permanent teeth, whereas the pathologic process can affect either the deciduous or permanent teeth. According to the glossary of endodontic terms, root resorption can be classified as internal and external resorption.³ However, a uniform

classification regarding internal and external resorption and their subdivisions does not exist in the literature.

Invasive cervical resorption (ICR), the least understood type of external root resorption, constitutes a distinct and pathological entity and has been described thoroughly by Heithersay.^{4,5} It is defined as 'a localised resorptive process that commences on the surface of the root below the epithelial attachment and the coronal aspect of the supporting alveolar process, namely the zone of the connective tissue attachment.'⁶ Numerous terms have been used in the literature to delineate external cervical resorption, such as: invasive cervical resorption, invasive resorption, cervical external resorption, progressive intradental resorption, extracanal invasive resorption, late cervical resorption and burrowing resorption.⁷ Due to the aggressive character of this lesion it is preferred that the term invasive cervical resorption rather than external cervical resorption will be used for the purposes of this paper.^{4,5}

External root resorption can be subclassified into the following types according to certain authors: external surface resorption,

external inflammatory resorption, external replacement resorption, external cervical resorption and transient apical breakdown.^{1,2} Another classification has been given by Hiremath *et al.*,⁸ who distinguished external surface resorption, external inflammatory root resorption, replacement resorption and ankylosis. Conversely, Andreasen has divided the external root resorption as surface, inflammatory and replacement.⁹

Histologically, the resorption site of ICR is characterised by highly vascular fibrous tissue with multinucleated osteoclastic cells along the dentin surface. A basic question to be answered is whether resorption is an inflammatory process activated by sulcular microorganisms or, alternatively, a type of fibro-vascular or fibro-osseous disorder in which microorganisms have no pathogenic role. Thereafter, the nature of this inflammatory process is not absolutely clear.^{4,10}

The purpose of this study is to present a comprehensive review together with the characteristics and the treatment of ICR through two cases which were followed-up for 36 months.

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Aetiology

The exact aetiology of cervical resorption is still unknown. ICR has been described as an 'aseptic resorptive process, which may, on occasions, become secondarily invaded with microorganisms'.²⁷ On the contrary, it has been suggested that the process sustaining ICR lesions is stimulated by microorganisms either by the gingival sulcus or the pulp space and dentinal tubules, in cases of teeth with necrotic pulp.^{6,10-12} However, according to other scientists the pulp tissue plays no role in the aetiology of ICR.^{4,13,14} The process usually begins from the cement-enamel junction where the epithelial attachment is generally established. Gold and Hasselgren proposed three environmental factors that may contribute to root resorption:¹⁵ absence of protection for the root surface such as in cases where the cement-enamel junction is open and there is exposed dentin, presence of vascular conjunctive tissue and some inflammatory stimulus. Any damage to the cementum below the epithelial attachment exposes the root surface to osteoclasts derived from the periodontal ligament which might lead to dentin resorption.^{12,15-17} Several etiologic factors have been blamed for causing ICR lesion.^{2,18} These include: dental trauma;^{4,7} poor occlusion pressure exerted by supernumerary teeth and/or orthodontic movement;¹⁹⁻²¹ tooth whitening;^{22,23} periodontal disease;⁸ periodontal therapy;^{4,10} and idiopathic aetiology.^{2,20,24} ICR has also been attributed to guided tissue regeneration.²⁵⁻²⁷ Systemic conditions, such as neoplasia,²⁰ normocalcemic hypercalciuria and nephrolithiasis,²⁸ have also been proposed as possible causes. Additionally, hyperoxaluria and oxalosis have been considered as possible causes of external root resorption, due to increased blood concentration of oxalates which results in the precipitation of crystals in hard tissues and the onset of the resorptive process.²⁹ It has also been suggested that there might be genetic predisposition to ICR.³⁰

Clinical features

ICR is a significant defect of the root surface, located at the cervical region of the tooth and, mainly, palatally. Adversely, buccal cases have been very rarely referred.^{31,32} It is usually detected at maxillary central incisors,^{8,33,34} Molar,^{35,36} lateral incisor,³¹ canine^{27,37} and premolar^{36,38} resorptive defects have also been reported. Highly vascularised resorptive

granulomatous tissue is found inside the tooth and hard dental tissues are undermined becoming thinner with irregular margins and translucent.^{1,39} The edges of the cavity are usually sharp and narrow.² This situation usually changes the colour of the region into a pinkish spot, which is the main recognition sign for the patient and/or the dental practitioner.^{1,34,39} However, in some cases, there is no pinkish appearance and the condition develops unnoticed until periodontal and/or pulpal implications appear. ICR is mainly asymptomatic and painless.² Nevertheless, sometimes symptoms including pulp pain and sensitivity could be observed.

A periodontal pocket occurs in the majority of cases. Profuse bleeding of the tissues is often observed during probing of the ICR and/or the associated periodontal pocket due to the high vascularisation of the granulomatous tissue.³⁹ Also, in some cases a localised gingival growth or a mucosal fenestration is noticed over the ICR lesion.^{8,40}

Radiographic features

The primary lesions of ICR are detected by chance on radiographs. ICR has a specific radiographic appearance which is consistent with expanding, asymmetrical coronal and apical radiolucency with irregular margins and an unchanged root canal. As the lesions become more and more advanced, the radiolucency expands in all directions and might involve adjacent alveolus, resembling an intrabony defect.^{2,6,39} Usually, even in advanced cases, there is no pulp involvement because of the protective predentin layer.^{6,41} However, the thin, remaining dentin layer is at risk of perforation.^{13,14}

Radiographic techniques mainly used are: the intraoral periapical parallel technique; the computed tomography; and, more recently, the cone beam computed tomography.^{36,42-44} The exact ICR size and location can be easily diagnosed with computed tomography, which is not necessary in all cases but only in selected ones.⁴²

Diagnosis

ICR can be diagnosed on the basis of radiographic evaluation of the tooth involved, which is mandatory, and through clinical examination. A clinical classification for assessment of ICR categorises the cases into 4 types:^{5,7} Class 1: a small, invasive resorptive lesion characterised

by shallow penetration into the dentine near the cervical area; Class 2: a well-defined, invasive resorptive lesion that has penetrated close into the coronal pulp chamber but has little or no extension into the radicular dentine; Class 3: a deeper invasion of the dentine that involves the coronal dentine and extends into the coronal third of the root; Class 4: a large invasive resorptive process that has been extended beyond the coronal third of the root. Moreover, tooth vitality tests are used and, generally speaking, the pulp of the tooth is vital and reacts physiologically. It should be noted that the pulp is involved only in very advanced ICR lesions.³⁴ ICR is often misdiagnosed. The differential diagnosis should consider ICR and internal root resorption, based on the radiographic appearance of the root canal.³⁴ Furthermore, ICR must be differentiated from root caries. This is achieved by using a periodontal probe: in the case of caries probing gives a soft and sticky sensation, while in the case of ICR one senses something hard with a scraping sound.^{1,23}

Case reports

Two cases with ICR are presented. Initially the patients' medical and dental history, clinical and radiographic examination and photographs were taken. They were treated with the same therapeutic protocol that is followed in the clinic.

First case

A 44-year-old female patient was referred to the Department of Preventive Dentistry, Periodontology and Implant Biology from the Department of Endodontology of Aristotle University of Thessaloniki. The patient had been complaining of bleeding and sensitivity in the region of her right maxillary central incisor. The medical history of the patient was uneventful. There was no history of trauma, orthodontic treatment or bleaching. The patient's oral hygiene was good. The periodontal examination, with PLI 29% and GBI 22%,^{45,46} revealed chronic localised gingivitis. Dental plaque and calculus were obvious mainly in the interdental regions. Clinically, at that time, there was a localised, palatal, erythematous overgrowth of the palatal surface of the tooth with a change in the gingival color. On probing the soft tissue, gingival bleeding was observed. Neither caries nor discoloration was present but only a temporary filling material on its palatal surface. Probing of the defect walls with an explorer produced a sense of hard underlying dentin. Moreover, it revealed a periodontal pocket of 5 mm mid-palatally

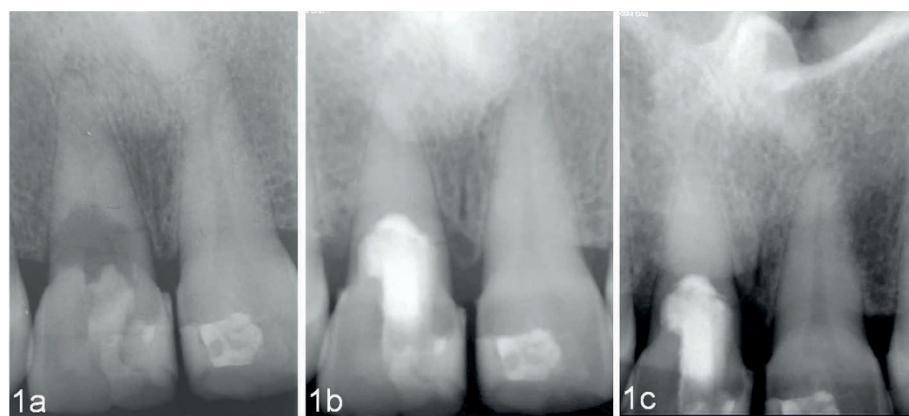


Fig. 1 Periapical intraoral radiographs of the first case: (a) pre-treatment, (b) 6 months after treatment, (c) 36 months after treatment

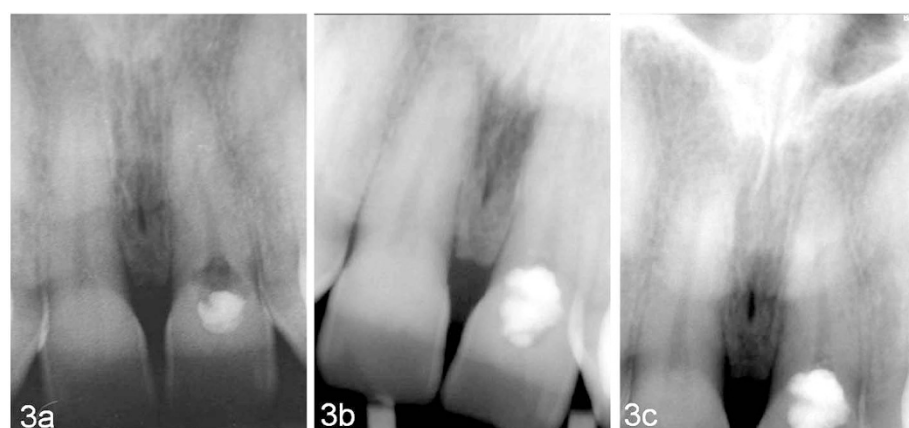


Fig. 3 Periapical intraoral radiographs of the second case: (a) pre-treatment, (b) 6 months after treatment, (c) 36 months after treatment

and a sulcular depth of 3 mm mesio-palatally and disto-palatally. Buccally, the periodontal condition was normal. Furthermore, the tooth responded normally to cold, hot and electric vitality tests. Also, no pain could be evoked on percussion. The periapical radiograph showed a well-defined irregular cervical radiolucent lesion extending over the cervical third of the root and projecting over the root canal outline. There was no evidence of resorption elsewhere on the root. There was no interdental bone loss and the periodontal ligament space and lamina dura were intact. A provisional diagnosis of ICR associated with the right maxillary central incisor was made (Figs 1a and 2a).

Second case

A 58-year-old female patient was referred to the Department of Preventive Dentistry, Periodontology and Implant Biology of the Aristotle University of Thessaloniki after her dentist had discovered an unusual lesion at the palatal cervical region of the left maxillary

central incisor. The patient complained of food impaction and her general dental practitioner had filled the tooth with temporary material. The medical history of the patient was unremarkable. There was no history of trauma, orthodontic treatment or bleaching, her oral hygiene was good and there were clinically healthy gingivae in all areas of her mouth. However, localised, marginally palatal gingivae were slightly erythematous. On probing the soft tissue, bleeding was observed. Probing of the defect walls with an explorer produced a sensation of hard underlying dentin. The tooth responded normally to thermal and electric vitality tests and was not tender to percussion. Periodontal probing revealed sulcular depth of 3 mm buccally and pocket depth of 4 mm palatally. A periapical radiograph revealed a well-demarcated area of radiolucency at the cemento-enamel junction extending coronally. The resorptive radiolucency laid over but did not appear to alter the pulpal outline. The pulp canal and chamber spaces were regular, well defined,

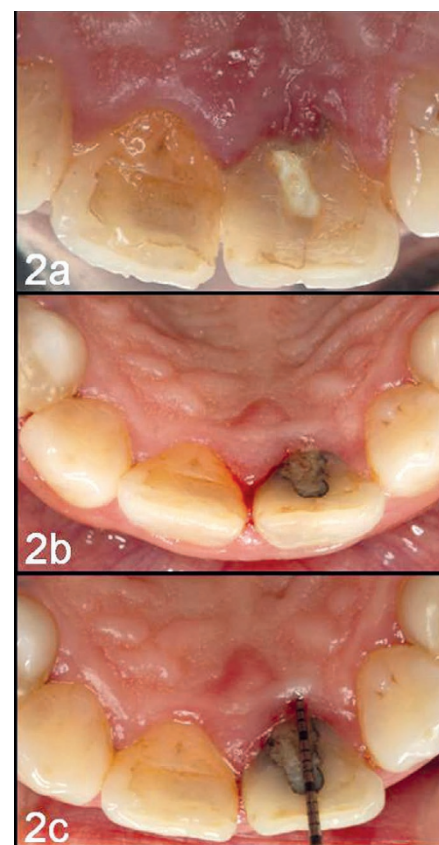


Fig. 2 Clinical pictures of the first case: (a) initial, (b) after 6 months, (c) after 36 months

and symmetric to the adjacent central incisor. There was no interdental bone loss, the lamina dura was intact, and the periodontal ligament space was normal. A provisional diagnosis of ICR associated with the left maxillary central incisor was made (Figs 3a and 4a).

The treatment protocol which followed for the management of both ICR cases in the clinic included surgical intervention under local anesthesia by raising a full-thickness mucoperiosteal flap expanding to the adjacent teeth with intra-sulcular incision, followed by excision of the overgrown fibrous and granulomatous tissue from the resorptive defect. The tissue excised was submitted to histopathologic examination. The excised tissue had been housed within the palatal defect, extending apically into the cervical region of the root. The rest of the root appeared intact. The defect had not caused any changes in the adjacent alveolar bone. The resorptive cavity was thoroughly debrided – after the temporary filling material in the first case was removed – and no soft dentin on the floor of the defect was found. After cleaning the defect carefully with 4R/4L Gracey surgical curette (Hu-Friedy, Chicago, IL), no perforation into the pulp was found, but the pink pulpal hue

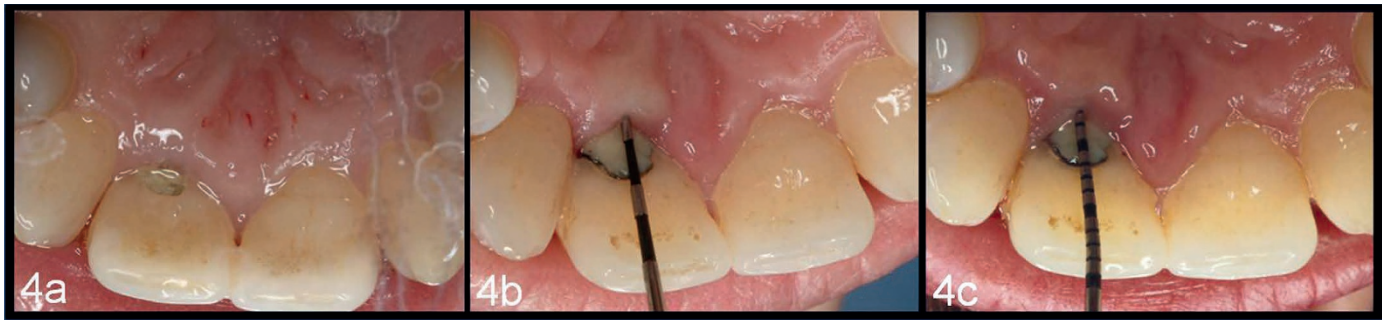


Fig. 4 Clinical pictures of the second case: (a) initial, (b) after 6 months, (c) after 36 months

was evident through the overlying dentin. The defect margin was smoothed using a high-speed bur under water spray. Then, the defect was lined with mineral trioxide aggregate (MTA) (Dentsply International Inc) and restored with glass ionomer cement (Fuji IX GC America Inc). The flap was repositioned and sutured (Fig. 5). The definite diagnosis as ICR lesions of these cases was made following surgical intervention and the histological evaluation which revealed fibrous connective tissue, multinucleated giant cells, and chronic inflammatory cells.

One-month recall showed satisfactory gingival healing of both cases. A good level of plaque control (<15%) was maintained. Six-month follow-up of the cases did not reveal any recurrence of the resorption and gingival healing appeared complete (Figs 1b, 2b, 3b and 4b). Pulp vitality tests revealed a healthy pulp. Probing depths on the palatal surface were around 3 mm. At the 36-month follow-up, clinical and radiographic examination confirmed a stable periodontal state and no deterioration of the cervical defect (Figs 1c, 2c, 3c and 4c).

Discussion

The exact causative factor of the present cases is unknown similar to other cases.^{33,35} Our results indicate that ICR management with the specific treatment protocol has very positive outcomes. At the 36-month follow-up the two teeth remained vital which is in agreement with the results presented in other papers.^{8,21,27,28,33,36,37,47,48} It is important that most of the external cervical resorptive lesions should not be treated as endodontic problems, because, in many cases, this resorptive condition may be treated surgically, without sacrificing pulpal vitality. However, there are ICR reports where endodontic treatment was performed additionally to surgical intervention.^{36,49} There have also been reports of cases with pulp necrosis.^{27,38}

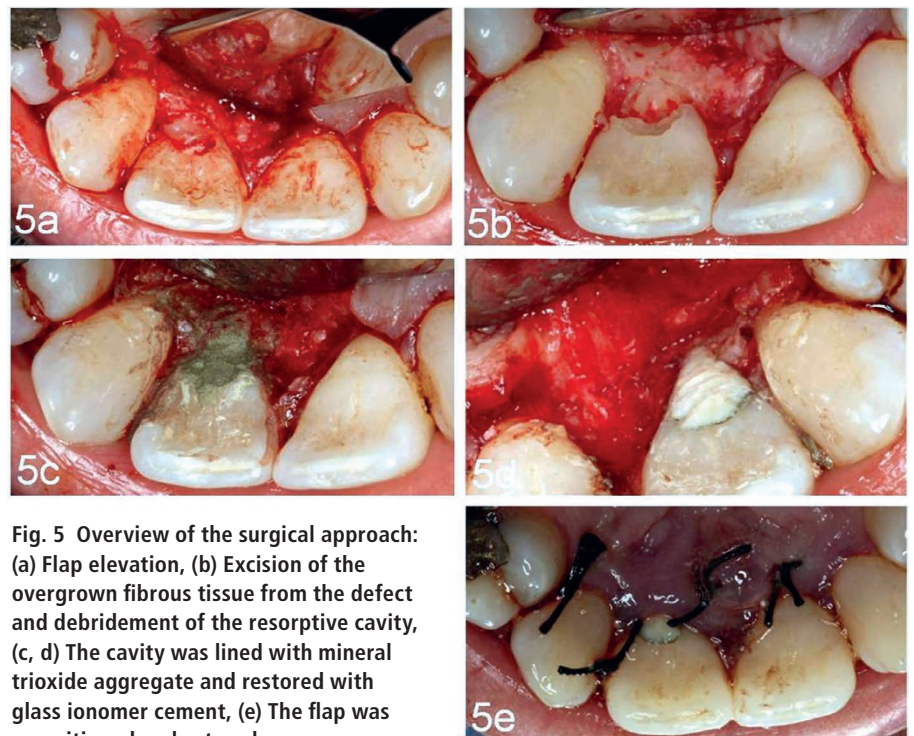


Fig. 5 Overview of the surgical approach: (a) Flap elevation, (b) Excision of the overgrown fibrous tissue from the defect and debridement of the resorptive cavity, (c, d) The cavity was lined with mineral trioxide aggregate and restored with glass ionomer cement, (e) The flap was repositioned and sutured

Sometimes, if the lesion is very close to the pulp canal, and during removal of the inflamed tissue from the resorption cavity, pulpal exposure is revealed or has already penetrated the pulp canal or the tooth is devital.^{27,29} endodontic treatment might be necessary before or during repairing of the ICR, additionally to surgical intervention.^{2,10,31,36,49} When the pulp is exposed, options for treatment are direct pulp-capping or root-canal treatment.⁵⁰ Therefore, sometimes root-canal treatment is deemed necessary during surgery so that permanent restoration engages the resorption cavity and the root canal for sufficient retention. This also avoids the risk of accidental displacement of the restorative materials during a second visit for root-canal obturation. However, it is advisable not to perform the root-canal treatment during surgery in a bleeding compromised region, but preferably complete it in a second stage under

more favorable conditions. For this reason, it is highlighted that a very good diagnosis based on a thorough radiographic evaluation, and if necessary CBCT, should precede the surgical treatment. The combined endodontic and surgical approach is also helpful in preventing the complication of persistent bleeding into the root-canal system during treatment.

The first case of this article is classified as a class 3 ICR lesion. It is stated that the treatment of this class is dubious and with a reported success of 77.8%.^{7,18} The second case is classified as a class 2 lesion with a complete success rate.^{7,18} Heithersay⁷ suggested that classes 1–3 can be managed successfully. The treatment of a class 4 case will be difficult to accomplish and these cases are more likely to fail. Therefore, it might be better to leave these teeth without treatment provided they are asymptomatic.^{5,7}

Different approaches have been proposed for the treatment of ICR, depending on its severity and location, on whether the defect has perforated the root canal system and on the restorability of the tooth. The treatment protocol generally applied includes surgical access, total removal of the resorptive (granulomatous) tissue, topical application of a 90% aqueous solution of trichloroacetic, thorough debridement, root canal treatment if the lesion has perforated the root canal, restoration of the resorptive defect by placement of a suitable filling material and follow-up examinations. Flap raising, when necessary, permits complete access and removal of the lesion from the root and proper filling of the defect, thereby decreasing the chances of recurrence.^{33,37,38} The application of the trichloroacetic acid results in coagulation necrosis with no damage to adjacent periodontal tissues.⁵¹ Glass-ionomer cements^{8,33,37,40} and light-cured resin composite materials have been recommended for the restoration of the resorptive cavities. Glass ionomer cement is capable of bonding to the tooth structure and it is well tolerated subgingivally, probably because of the antimicrobial effect caused by its slow release of fluoride.^{52–55} However, a more up-to-date filling material proposed is MTA due to its good sealing capacity in the presence of moisture, a high degree of biocompatibility use as repair material for perforations.^{38,40,43,52,56–58} In agreement with other authors, MTA and glass-ionomer cement were used to restore the resorptive cavities of the present cases.^{40,48,52} Complementary treatment modalities for the exposure and restoration of the resorption defect using orthodontic extrusion, crown lengthening, guided tissue regeneration, and even either intentional replantation or a topical application of bisphosphonate/anticalcic agents have been proposed.^{7,34,56,59,60} In some cases, crown lengthening procedures should be implemented simultaneously with the periodontal surgery and the exposure of the lesion. This depends mainly on the morphology and the location of the lesion as well as the quantity of the remaining tooth structure.

Periodontal debridement that might inadvertently result in cementum removal has not been identified as a major predisposing factor.⁵ ICR might be prevented after periodontal debridement of the root surface by rapid epithelial downgrowth obviating any contact of connective tissue cells with that surface, thus hindering the inflammatory process.⁶

Periodontal reattachment after surgical intervention cannot be expected using

amalgam or composite resin and it is unlikely with glass ionomer cement, but there is experimental evidence to suggest that such reattachment might be possible if MTA is used.⁷ However, in the areas that will be in constant contact with the oral flora, the MTA will be continuously contaminated and subgingival plaque development might be promoted as a result of the rough MTA surface.

Conclusions

ICR is accurately diagnosed on the basis of clinical and radiographic examination findings. It can be treated successfully with an approach including periodontist, so as to sustain the tooth and achieve good aesthetics; a supportive periodontal therapy should be followed. The 36-month follow-up revealed a healthy pulp when applying pulp vitality tests, while the clinical and radiographic examination confirmed the stable periodontal condition and showed no deterioration of the cervical defect.

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