International Journal of Research in Health and Allied Sciences

Journal home page: www.ijrhas.com

Official Publication of "*Society for Scientific Research and Studies*" [Regd.]

ISSN 2455-7803 Index Copernicus value 2016 = 68.10

CASE REPORT

Surgical Management of rare Heithersay class IV external root resorption of maxillary lateral incisor with Ketac Molar GIC and Resorbable Membrane: A Case Report

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ABSTRACT

Invasive cervical resorption is a relatively uncommon form of external root resorption. There may be no external signs, and the resorptive condition is often detected by routine radiographic examination. Where the lesion is visible, the clinical features vary from a small defect at the gingival margin to a pink coronal discoloration of the tooth crown resulting in ultimate cavitation of the overlying enamel. The condition is usually painless unless pulpal or periodontal infection supervenes. Radiographic features of lesions vary from well delineated to irregularly bordered mottled radiolucencies, and these can be confused with dental caries. A characteristic radiopaque line generally separates the image of the lesion from that of the root canal, because the pulp remains protected by a thin layer of pre-dentin until late in the process. Histo-pathologically, the lesions contain fibro-vascular tissue with resorbing classic cells adjacent to the dentin surface. More advanced lesions display fibro-osseous characteristics with deposition of ectopic bonelike calcifications both within the resorbing tissue and directly on the dentin surface. Secondary invasion of microorganisms into the pulp or periodontal ligament space will elicit a normal inflammatory response.Effective management and appropriate treatment can only be carried out if the true nature and exact location of the ECR lesion are known. This case presents 12 month follow up of Heithersay class 4 external resorption. Even though readiograph suggested presence of radiolucency, patient was clinically asymptomatic and mobility was also reduced considerably.

Keywords: Heithersay treatment, external cervical resorption, ketac molar gic, resorbable membrane.

Received: 14 October 2018 Revised: 27 October 2018 Accepted: 28 October 2018

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This article may be cited as: Parmar PD, Kaur R, Singh N. Surgical Management of rare Heithersay class IV external root resorption of maxillary lateral incisor with Ketac Molar GIC and Resorbable Membrane: A Case Report. J Adv Med Dent Scie Res 2018;6(11):34-37.

INTRODUCTION

Root resorption is the loss of hard dental tissue (ie, cementum and dentin) as a result of odontoclasticaction[1]. Physiologic root resorption associated with primary teeth is desirable because it results in exfoliation of the teeth, thereby allowing eruption of the permanent successors. However, root resorption of permanent dentition is usually unfavorable because it might result in irreversible damage and/or eventual tooth loss. Root resorption might be classified by its location in relation to the root surface, ie,internal or external resorption. External root resorption can be further classified into surface resorption, external inflammatory resorption, external replacement resorption, external cervical resorption, and transient apical breakdown[1]. One of the least understood of the types of external resorption is external cervical resorption (ECR). This form of external resorption has been described at length by Heithersay[2–5], who preferred the term invasive cervical resorption, which describes its invasive and aggressive nature. Other terms used to describe ECR include odontoclastoma[6], peripheral cervical resorption[7],extracanal invasive resorption[8], supraosseous extracanal invasive resorption[9], peripheral inflammatory root resorption[10], and subepithelial external root

resorption[11]. In this article it will be described as ECR, which reflects its starting point on the tooth. ECR usually occurs immediately below the epithelial attachment of the tooth at the cervical region[1,2,12]. ECR defects can be difficult to diagnose and manage.

The exact cause of ECR is poorly understood. Cementum is considered to protect the underlying root dentin from being resorbed. It is broadly accepted that damage to or deficiency of this protective cementum layer below the epithelial attachment exposes the root surface to osteoclasts, which then resorb the dentin[10,13]. The anatomic profile of the cementoenamel junction might also predispose this region to ECR. Microscopicanalysis of the cervical region of teeth has shown that there appears to be frequent gaps of cementum in this area, leaving the underlying mineralized dentin exposed and vulnerable to osteoclastic root resorption[14]. The pulp tissue plays no role in the etiology of ECR[2,9,12]. Several etiologic factors have been suggested that might damage the cervical region of the root surface and therefore initiate ECR. These include dental trauma[2,15], orthodontic treatment[2,16], intracoronal bleaching[2,17], periodontal therapy[2, 11], and idiopathic etiology[18,19]. There appear to be polarized views on the nature of the resorptive process. Some have regarded it as a purely inflammatory reaction[17,20]. ECR has been described as an ‘‘aseptic resorptive process, which may on occasions become secondarily invaded with microorganisms’’[15]. Others have suggested that microorganisms from either the gingival sulcus [11,21,22]or the pulp space and dentinal tubules in teeth with necrotic pulps provide the necessary stimulus to sustain ECR lesions. Heithersay[2]investigated the potential predisposing factors in 257 ECR lesions in 222 patients. He concluded that a history of orthodontic treatment, dental trauma, and bleaching were the most commonly associated predisposing factors for ECR. Heithersay[2]classified ECR according to the extent of the lesion within the tooth: class 1, a small invasive resorptive lesion near the cervical area with shallow penetration into dentin; class 2, a well definedinvasive 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, notonly involving the coronal dentin but also extending at least to thecoronal third of the root.; and class 4, a large invasive resorptiveprocess that has extended beyond the coronal third of the root canal.Endodontic treatment might be necessary with some class 2 and usually class 3 lesions when pulpal involvement has occurred or is very close to occurring. Heithersay has reported a 100% success rate in the treatment of class I and II ECR lesions treated in this way. The success rate in class 3 lesions was 77.8% and only 12.5% of teeth in class 4 cases. Heithersay concluded that classes 1–3 were treatable, but class 4 lesions were not amenable to treatment, and these cases would have benefited from alternative treatment such as extraction and replacement with an implant retained crown restoration.

CASE REPORT

A 32 year old male patient came to department of conservative dentistry and endodontics with the history of pain and sinus formation in relation with the maxillary right lateral incisor since last 2 weeks. He had a dental history of trauma 12 years before and undergone root canal treatment for the same. He also underwent surgery for maxillary right central incisor twelve years before. Radiograph suggested Heithersay class 4 extensive invasive cervical resorption of lateral incisor (Fig. 1A). Sinus tracing also revealed periapical pathology related to the same tooth (Fig. 1B). Detail explanation were given to the patient about possible outcome of treatment and survival chances of the tooth, but patient wanted his natural tooth to be saved so surgical intervention was explained and appointment was scheduled for the same after prescribing five days course of augmentin (625 mg) and ibuprofen (400 mg) thrice a day. Patient was also instructed to rinse chlorhexidine mouthwash for 5 days three times a day.

All the surgical procedures were carried out in minor operation theatre of the department. Patient was advised to do 2% chlorhexidinemouthrinse for 1 minute. Local anaesthesia was achieved with lignocaine plus adrenaline (1:80,000). Careful full mucoperiosteal flap was reflected only on palatal side (Fig. 1C). Only crevicular incision was given without vertical incision. After reflecting flap guttapurcha removed from the canal and only 3 mm of guttapurcha left remained inside (Fig. 1D). Thorough curettage was done on resorption site and apically of the tooth. 90% of TCA (trichloroacetic acid) was used for one minute on the resorpted root and around surface. Topical application of trichloroaceticacid results in coagulation necrosis of the ECR resorptive tissue, with no damage to adjacent periodontal tissues [5]. It also infiltrates the small channels and recesses of ECR that would otherwise be unreachable by mechanical instrumentation. After complete debridement MTA base (Fig. 1E) was given in the apical root before building the tooth with Ketac Molar GIC (Fig. 1F & 1G).

After creation of wall around the root with GIC palatal site was covered with Healiguideresorbable membrane (Fig. 1H) and site sutured with 5-0 reverse cutting black silk suture (Fig. 1I). Because of mobility lateral incisor was splinted with composite together with central incisor and canine (Fig. 1J). Sutures were removed after 1 week and patient was asymptomatic at that time. Patient was kept on regular follow up for 1 year (Fig. 1K). Splinting was removed after 9 month only. There was considerable reduction in mobility of tooth and patient was asymptomatic even though x-ray suggested presence of radiolucency around root.

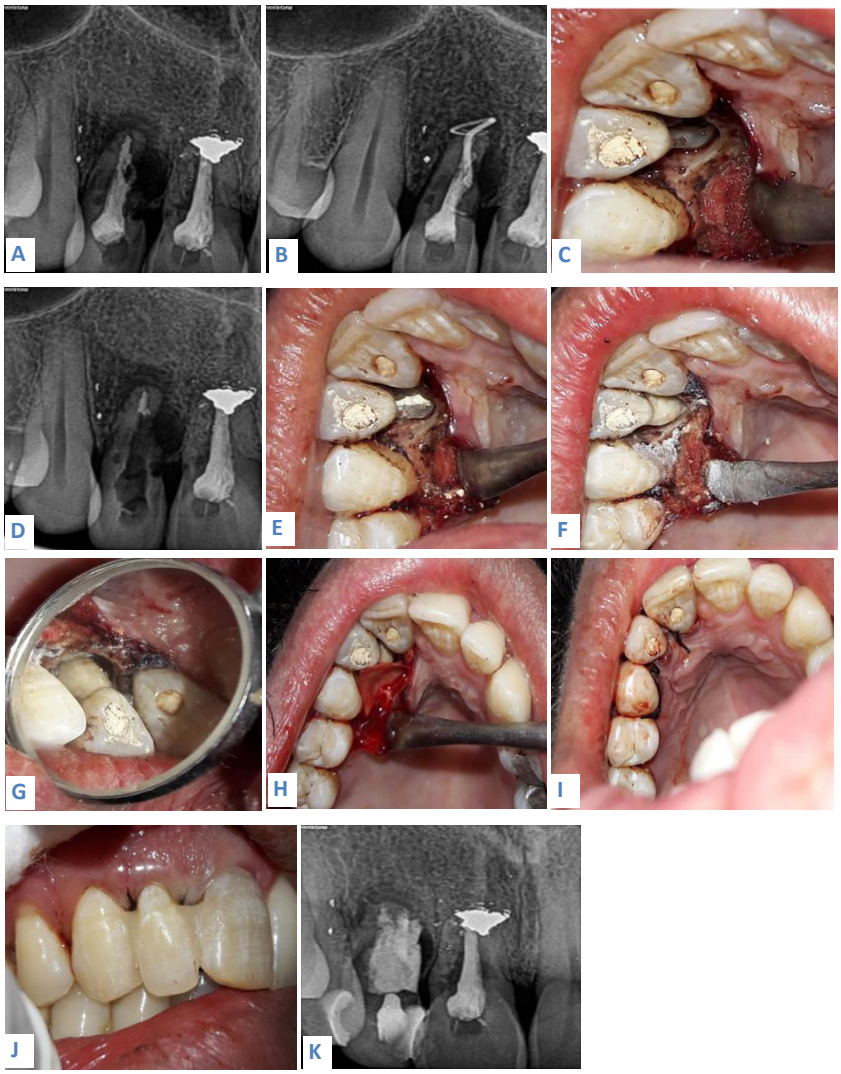

FIGURE 1: **A**-pre-operative radiograph, **B**-sinus tracing, **C**-flap elevation, **D**-x-ray after removal of guttapurcha and disinfection, **E**-MTA application, **F**&**G**-Ketac molar GIC build-up of root, **H**-resorbable membrane application, **I**- suturing of flap, **J**-composite splinting, **K**-one year follow up.

DISCUSSION

ICR is an external resorption that is characterized by invasion of the cervical region of the tooth by fibrovascular tissue[2]. The invading tissue arises from the periodontal ligament but differs from periodontal tissues in both structure and behaviour. The precursor cells of the periodontal ligament have the potential to differentiate into cells capable of laying down fibrous or calcified tissue. For invasion to occur, a defect in the cementum/cementoid layer is a likely prerequisite[8]. This may be of developmental origin in a small zone near the cervical area or the result of physical or chemical truma. ICR is primarily caused by dental trauma or injury of the cervical periodontal attachment. In a study, a group of 222 patients with a total of 257 teeth displaying varying degrees of ICR had been assessed. Of the potential predisposing factors had been identified, orthodontics and trauma was the most common two factors in that study[2].

In a study, one of the few research article in the literature, was revealed that prevalence of ICR was less than 0.1%[3]. This study showed that prevalence of ICR was almost 0.08% and consistent with the literature. Although relatively small number of patients, the data provided comparison to other studies. Frequently ICR is detected incidentally in a routine intraoral or panoramic radiograph, because the lesion is usually painless and do not elicit any clinical signs[1,2,5,12,15]. They present an irregular radiolucency with indistinctexamination. ICR is often misdiagnosed as internal resorption. While a pink discoloration of the crown may indicate ICR; it may as well result from an internal resorption[1,2,22]. Radiographs taken using the parallax technique may also be used to differentiate ICR from internal resorptive lesions. If the

lesion is ICR, the radiographic position of the lesion alters when the angle of the X-ray beam is changed, two radiographic features are considered as signs of ICR:A communication of the resorptive defect with the periodontal space and an intact dentinal wall against the pulp cavity.

This type of resorption difficult to diagnose and it is even more challenging to identify the extent and nature ofthe process, especially in cases where the resorptive defect is buccal or palatine location. With conventional radiographic images, there are limitations that not only prevent the proper identification of the resorptive process, but also hinter the planning and the evaluation of the prognosis with a treatment[1,13-17]. CBCT is a relatively new three dimensional imaging technique requiring a significantly lower radiation dose than conventional computed tomography. The use of CBCT is very helpful in diagnosing the exact size and location of the resorption.

CONCLUSION

Early detection is essential for successful management andoutcome of ECR. Patients with an ECR lesion with no apparent identifiable etiologic factor should have their entire dentition assessed to ensure that no other teeth are affected by ECR. Patients with a history of 1 or more predisposing factors should be monitored closely for initial signs of ECR. The very low risk of developing ECR does not justify taking additional radiographs. However, every radiographic investigation carried out for general examination or diagnostic purposes should be routinely checked for ECR lesions if the teeth in question have been exposed to 1 or more of the predisposing factors.This article presents one year survival of Heithersay class 4 external root resorption. More longer follow up and CBCT as a diagnostic tool would be more appropriate to include in treatment plan for such lesions.

ACKNOWLEDGEMENTS

The authors reported no conflicts of interest related to this study.

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