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External cervical resorption: A Review

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Abstract:-External cervical resorption (ECR) is a type of external root resorption. ECR is relatively uncommon and the etiology is not very clear. Often misdiagnosed leading to improper management or tooth loss.Clinicallyit's often asymptomatic unless pulpal or periodontal involvement begins. ECR is mostly noted in routine radiograph. Accurate assessment of the true nature and accessibility of ECR is important for its effective management

Key words :- External cervical resorption, root resorption, Etiology, Management

1. INTRODUCTION

Tooth resorption is the loss of hard dental tissue (i.e. cementum and dentin) as a result of odontoclastic action[1]. Based on its location in relation to the root surface root resorption is classified as internal or external resorption. External root resorption is further classified into external inflammatory resorption, surface resorption, transient apical breakdown, external replacement resorption, and external cervical resorption [1]. External cervical resorption (ECR) is the least understood of all other types of external.External resorption has been described by Heithersay, who preferred the term invasive cervical resorption, which describes its invasive and aggressive nature[2]. ECR are also given various terminologies which includes peripheral inflammatory root resorption, extracanal invasive resorption, peripheral cervical resorption, supraosseous extracanal invasive resorption, and subepithelial external

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root resorption. ECR usually occurs at the cervical region of the tooth immediately below the epithelial attachment[1].External cervical resorption (ECR) usually manifests in the cervical aspect of teeth; it develops as a result of damage to, and/or deficiency of the periodontal ligament (PDL) [3]and the subepithelial cementum. External cervical resorption (ECR) is a dynamic process that involves periodontal, dental and in later stages pulpal tissues [4].

ETIOLOGICAL FACTORS

Several etiological factors are suggested which may damage the cervical region of the root surface and thus initiate ECR.Heithersayinvestigated 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 and other factors include periodontal therapy idiopathic etiology and others like: bruxism, intracoronal restorations, developmental defects, systemic diseases[2, 5]. There is a controversy among investigators about nature of the lesion. Some have regarded ECR as a purely inflammatory reaction [6].Describing as an ‘‘aseptic resorptive process, which may on occasions become secondarily invaded with microorganisms’’ [7]. Others have suggested that microorganisms either from the pulp space and dentinal tubules in teeth with necrotic pulps or from the gingival sulcus provide the necessary stimulus to sustain ECR lesions[8].

ORTHODONTIC TREATMENT

Excessive orthodontic forces at the cervical region of the tooth might end in tissue necrosis adjacent to exposed root dentin. This might end in mononuclear precursor cells being stimulated to differentiate into odontoclasts, which are attracted to and resorb the exposed root dentin. Heithersay found that orthodontic treatment alone was a potential predisposing factor for 24.1% of teeth with ECR and found that the most commonly affected teeth were maxillary canines, maxillary incisors, and mandibular molars. Due to the location of orthodontic bands in Mandibular molars which may damage the vulnerable cementoenamel region of the tooth thus they are susceptible to resorption[9].

TRAUMA

Luxation and avulsion injuries are the recognized complication of ECR [9]. Dental trauma was a major potential predisposing factor confirmed by heithersay (15.1% of teeth) which then increased to 25.7% of teeth when other contributory factors (for example, orthodontic treatment, intracoronal bleaching) were included. Maxillary central incisors are the most commonly traumatized that subsequently developed ECR[9]. This is according to their location within the dental arch and associated susceptibility to trauma [10]. The use of splints (especially interdental wiring) might additionally damage the cementoenamel junction and thus predispose to ECR [11]. Andreasen and Andreasen found that orthodontic extrusion seems to provide better marginal bone healing as compared with surgical respositioning[3].

INTRACORONAL BLEACHING

Heithersay reported 3.9% and 13.6% of ECR cases in intracoronal bleaching as a sole and associated predisposing factor respectively [9]. Several suggestions are suggested to eluciate the particular mechanism by which intracoronal bleaching would end in ECR. Rotstein et al demonstrated that during intracoronal bleaching with 30% hydrogen peroxide the presence of cemental defects at the cementoenamel junction result in escape of hydrogen peroxide from the pulp chamber of root-filled teeth to the external tooth surface via dentinal tubule. It has

been suggested that hydrogen peroxide might denature dentin and provoke an immune response [12]. In addition, intracoronal placement of a ‘‘walking bleach’’ paste the pH at the root surface of teeth is reduced to about 6.5 .This slightly acidic environment is known to provoke or enhance the osteoclastic activity, which might result in ECR [13]. Friedman et al found ECR in about 6.9% teeth (n = 58) after bleaching. The authors supported the conclusions of previous reports concluding heat and trauma as the etiologic factors in bleaching-related cases of ECR. They also stated that the type of bleaching was not related to occurrence of resorption [14].

As a preventative step before on an internal bleaching treatment, radiographic and clinical examination must be done to make sure that there is no cervical defect that would allow excessive penetration of hydrogen peroxide. to reduce the likelihood of periodontal and cervical leakage of the bleaching agent ,Coronal sealing of the root canal with a protective material (for example, placement of glass ionomer cement or intermediate restorative material at the cervical level of the root canal) is necessary [15]. A safer alternative to hydrogen peroxide is the Sodium perborate mixed with water as an intracoronal bleaching agent. It has also been suggested that 35% carbamide peroxide (urea peroxide) appears to combine the efficacy of 35% hydrogen peroxide together with the safety of sodium perborate [16].

SURGICAL PROCEDURE

Surgical procedures which result in damage to the cementoenamel junction were found to be a significant predisposing factor in the study by Heithersay [9]. This category represents a comparatively low incidence, considering the frequency of such procedures. These include orthognathic surgeries, surgical exposure of teeth for orthodontic purposes, teeth adjacent to disimpaction transplantation [7]. Impairment of blood supply, heat damage to bone is important factors associated to root resorption though there exact association is uncertain. Apart from these, individual’s age,sex, nutrition, systemic factors and genetic propensity can be the associated factors.

PERIODONTAL THERAPY

According to Heitherssay,periodontal surgeries which may end in damage to cementum can result in resorption in 1.6% of cases. Usually, the resorption is prevented after periodontal debridement because the contact of connective tissue cells with the root surface is prevented which in turn prevents inflammatory process[17]. But the cases of resorption are reported after regenerative periodontal procedures and after tetracycline root conditioning. Reason might be due to the damage to the root surfaces not protected by junctional epithelium are repopulated by connective tissue cells. The increased migration of connective tissue fibroblasts can enhance the danger of resorption [18].

SYSTEMIC FACTORS

Various systemic factors have also been reported to be associated with ECR though the association is still not confirmed. Moskow et al reported a case of ECR in patient with hyperoxaluria and oxalosis. Liang et al reported five patients of ECR that were associated with hormonal abnormalities; although, it is still unclear whether these abnormalities were

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initiating or contributing factors. The periodontal tissues are sensitive to hormonal fluctuations during puberty and pregnancy but still there is no clear evidence of role of hormonal changes in pathogenesis of ECR [5]. Viral etiology has to be still investigated;Thomes et al studied the association between multiple ECR and neutralization tests for feline Herpes Virus Type I[19].

OTHER FACTORS

These include intracoronal restorations and bruxism [9]. Developmental defects like hypoplasia or hypomineralization of cementum have also been suggested as predisposing factors. A majority of the 14.9% of patients who had no other obvious potential predisposing factor associated with their ECR lesions and 15.3% of patients were categorized as having intracoronal restorations as their predisposing factors[9]. However, it appears that when there have been no other potential predisposing factors identifiable and a coronal restoration was present, it had been by default classified because the potential predisposing factor for ECR. This won't be a true reflection of the actual etiology.

DIAGNOSIS

A pink spot in the cervical region of the tooth is usually the clinical sign noticed by the patient and/or dentist this discoloration is a result of the highly vascular granulation (resorptive) tissue within the tooth becoming visible through the thinned out (resorbed) dentin and translucent overlying enamel [1].It must be stressed that pink spots are relatively rare. Teeth with ECR may also have grey discoloration due to pulp necrosis

It is important to differentiate ECR from subgingival caries when pink spot is absent and it feels sticky on probing [1]. The base of an ECR defect will feel hard with a scraping sound when probed [9]. Profuse bleeding is seen while probing ECR and/or associated with periodontal pocket due to underlying highly vascular resorptive tissue. Once the granulation tissue is removed the cavity walls will feel hard and mineralized on probing. The edges of the cavity usually appear narrow and sharp. Teeth with ECR respond positive to sensibility testing until it advances to root canal perforation and pulpal necrosis [20].

There is no 'classic' radiographic appearance of ECR. Lesions may be asymmetrical or symmetrical; margin being defined and smooth to poor definition or ragged or even with no clear delineation between ECR and healthy root structure. ECR lesions will be radiolucent in 'resorptive' phase and more radiopaque in 'reparative' phase that is a mottled or cloudy appearance due to ossification of the granulomatous resorptive tissue. Distinct radiopaque striations of hard tissue are seen in some cases [21].

The parallax technique helps to distinguish between external and internal resorption. With ECR the defect will either move in same (palatal / lingual) or in the opposite (labial) direction of the x-ray tube whereas with internal resorption the defect remains centered on the root canal system no matter the angle of the radiograph exposure[1].

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-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

It has been shown that conventional radiographic techniques reveal limited information on the true extent and nature of the resorptive lesion compared to cone beam computed tomography (CBCT)

Patel et al. (2018) suggested a 3-dimensional classification for ECR based on the radiographic findings of periapicalradiograph and CBCT. This classification considers the height of a lesion, its circumferential spread and proximity to the root canal[22].

The new classification takes into account the ECR lesion height (1: at CEJ level or coronal to the bone crest (supracrestal), 2: extends into the coronal third of the root and apical to the bone crest (subcrestal), 3: extends into the mid-third of the root, 4: extends into the apical third of the root), circumferential spread (A: ≤90° B: ≤180° C: ≤270° D: >270°) and proximity to the root canal (d: lesion confined to dentine, p: probable pulpal involvement), thus classifying ECR in three dimensions.

HISTOPATHOLOGY

The histologic appearance of the ECR is similar to external inflammatory resorption; the resorptive cavity consists of granulomatous tissue. Osteoclasts in resorbing front within the lacunae. In early stage the pulp remains healthy (uninflamed) as it is protected by predentin and innermost layer of dentin [2]. As the lesion advances, bone-like material (replacement resorption) might deposit within the lesion and also in direct contact with the adjacent dentin; this indicates that the lesion is not destructive but attempting to repair itself [1, 7, 9]. The predentin has been shown to contain resorption inhibitor and an anti-invasion factor that prevents ECR affecting the root canal until it gets advanced. Preferential odontoclastic dissolution of interprismatic enamel makes the outer surface of enamel to be resistant to resorption [21]. Early ECR defects mostly won't contain acute inflammatory cells, implying a nonbacterial etiology. In later stage, a secondary bacterial colonization of dentinal tubules might induce an inflammatory response in the associated pulpal or periodontal tissue [7]. Fibroosseous tissue found to deposit within the root canal system when ECR involves pulp [23].

TREATMENT

Treatment objective includes; to Arrest resorptive process, Prevent further resorption, Restore damaged root surface, Improve esthetics of tooth (in cases with pink spot). Management depends on the location, severity, whether the defect has perforated the root canal and the restorability of the tooth. Several treatment regimens have been suggested in the literature based on the severity these include intentional replantation [24], guided tissue regeneration [25], treating the ECR lesion by an internal approach only [9], and forced orthodontic eruption [26].

Essentially, treatment involves complete removal of the resorptive tissue and restoring the defect with a restoration.

To treat the ECR lesions confidently, it is important to reflect a full-thickness periosteal flap for complete access and removal of the ECR lesion from the root, to decrease recurrence, curetting away the granulomatous tissue from the adjacent periodontium to sever the blood

supply had to be done. Endodontic treatment is necessary with lesions where defect has perforated the root canal or very close to pulp and remaining thin dentin layer is at risk of perforation during removal of granulation tissue [27].

The defect might be restored by appropriate restorative material like glass-inomer cement,composite resin, amalgam or MTA etc. for radicular defects MTA is generally suggested because it is most biologically acceptable material [28].

If the defect in cervical third of tooth is inaccessible in oral cavity then apically positioned flap or orthodontic extrusion of tooth can be used but this might give esthetically compromised results[7]. An alternative treatment option is intentional replantation of tooth which is attempted only when no other treatment modality is feasible. When resorptive portal of entry is within the bone and inaccessible, then the affected tooth is endodontically treated, extracted, the defect is debrided, restored and then replanted into its socket [24].

2. CONCLUSION

ECR process in three stages they are initiation, progression/resorption and reparative phase. Resorption and repair/remodeling can progress simultaneously in parallel at different areas of the affected tooth. Early detection is important for successful management and outcome of ECR. CBCT appears to be a promising diagnostic tool for confirming the presence, identifying the true nature and managing ECR. Thus Proper diagnosis, case selection, and its implementation can lead to the successful outcome and long-term retention of the toot

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