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A Review of External Cervical Resorption

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INTRODUCTION

External cervical resorption (ECR) is a pathological process distinguished by its localization in the cervical root area, its invasive nature and the formation of a mineralized replacement tissue resembling bone or cellular cementum, notably not associated with ankylosis (Fig.1).[1] During initiation of cervical root resorption, the portal of entry is the cementum below the gingival epithelium, and resorption starts with localized destruction and/or removal of PDL. Response to PDL injury includes formation of a blood clot and inflammation, followed by granulation tissue and recruitment of macrophages to the affected area. Impaired vasculature in the area leads to hypoxia, which promotes multinucleated-cell differentiation and activity (Fig.2).
As the osteoclastic/odontoclastic resorptive lesions expand toward the pulp space by destroying cementum, dentin, and enamel, several resorption channels and interconnections with the PDL are created, generating a 3D space (Fig.3).
A basic question to be answered by researchers is whether this resorptive process is purely inflammatory in nature, activated by sulcular microorganisms, or alternatively a type of benign proliferative fibrovascular or fibro-osseous disorder in which microorganisms have no pathogenic role but may become secondary invaders.. (Fig.4).

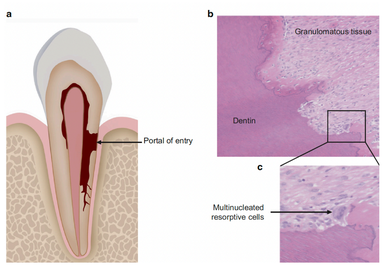

Figure 1. Schematic image and histological sections of ECR. **a** Schematic image representing the histopathological patterns of ECR lesion. **b** Pathological section of ECR lesion showing granulomatous tissue and the impaired dentin. **c** Pathological section of ECR lesion showing multinucleate resorptive cells located in resorption lacunae.
(Chen et al. 2021)

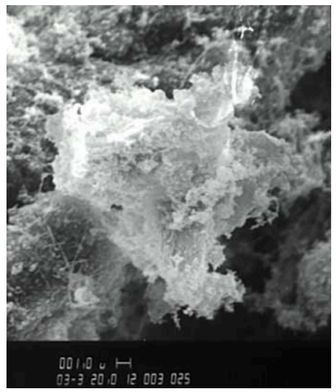

Figure 2. Multinucleated Giant Cell

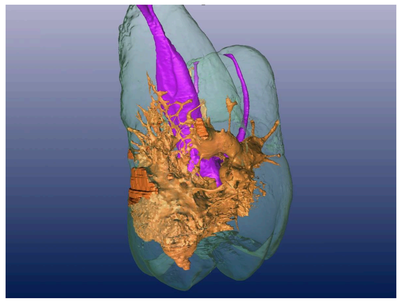

Figure 3. Three- dimensional representation of External Cervical Resorption

Figure 4. Histological image of tooth #30 with ECR sowing a small portal of entry and ingrowth of bone-like tissue. Active blastic cells are found in the reparative bone-like tissue. Histological staining was performed with a Stevenel's blue and Von Gleson's picrofuchsin.
(Patel et al, 2017)

METHODS

Using keywords such as external cervical resorption, pathogenesis, histopathology, osteoclastogenesis in PubMed and Google Scholar search engines, pertinent literature was obtained. Seventeen articles were collected, and the search was run with no language, place or time restrictions. The review aims to summarize the latest findings on pathogenesis of ECR, so as to provide an evidence-based reference for clinicians.

RESULTS

The etiology and pathogenesis of ECR are largely obscure. A hypothesis regarding its development claims that a local discontinuity of the protecting layer of radicular cementum renders the subjacent dentin accessible for osteoclasts or dentoclasts.[2] In fact, voids in cementum and exposed dentin frequently occur in the cervical root areas close to the CEJ.[3,4] These voids can arise as a primary malformation of enamel-cementum closure or secondarily result from a physical or chemical trauma. In agreement with this assumption, antecedent orthodontic treatment (24.1%), trauma (15.1%), or intracoronal bleaching (3.9%) have been found to be the most prevalent sole etiologic factors associated with ECR and the suceptibilty of exposed dentin to injury due to its higher protein content. However, 16.4% of the lesions occurred without any apparent reason, i.e. were idiopathic.[5]
The presence of inflammatory cells is not necessarily indicative of a microbiological etiology and there are cases in the literature, which show no inflammation.[1] This indicates that invasive cervical resorption is an aseptic resorptive process, which may on occasions become secondarily invaded with microorganisms.
ECR is mainly characterized by 3 stages:
-resorption initiation (first stage),
-resorption progression (second stage),
- repair (third stage).
In the first stage: a local destruction of the normal PDL structure and homeostasis takes place.[6,7]
Injury of the PDL will lead to the formation of a blood clot and a subsequent localized inflammation[8].
This healing process is evident by the formation of granulation tissue from the adjacent alveolar bone often leading to vulnerability in the dentin matrix. [9]
Resorption Progression: resorption invades the tooth structure by destroying cementum, dentin, and enamel. It was observed that clastic cells are involved in this process.[10]
Reparative Stage: Finally, repair takes place by an ingrowth of bonelike tissue into the resorption cavity. In particular, the bonelike tissue grows into the cavity through the portal of entry and substitutes the resorbed tooth tissues.
Based on a study by Heithersay, in which 257 ECR teeth were analyzed, orthodontic treatment (28.4%) was the most common factor and previous traumatic injury was the second. Additional factors such as internal bleaching, surgery and restorative treatment were identified as predisposing factors. According to Mavridou et al, who assessed 337 ECR teeth, several additional predisposing factors were reported.[11] These included: malocclusion, parafunctional habits, poor oral hygiene, extraction of an adjacent tooth, impacted teeth, eruption disorders (pressure generated by canine eruption on lateral incisors), viral etiology, individual genetic propensity and playing wind instruments . Orthodontic treatment as a predisposing factor increased from 28.4 to 45.7% . [12] (Fig.5)

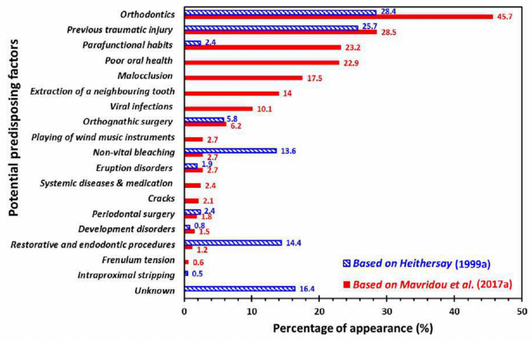

*Figure 5.*Distribution of potential predisposing factors of ECR. Comparison of percentages between Heithersay 1999 and *Mavridou et al, 2017*
(Patel et al, 2019)

CONCLUSIONS

In recent years, case reports and retrospective studies have revealed many risk factors for ECR. Nevertheless, the exact correlation between these factors and the initiation of ECR has not yet been fully elucidated because of our insufficient understanding of ECR pathogenesis due to a lack of focus on its pathogenesis. Our understanding of the etiology of ECR is deficient, which may explain why the majority of patients were diagnosed in advanced stages. In view of the difficulty in early diagnosis along with the undesirable outcome of ECR, a comprehensive understanding of its potential predisposing factors as well as its pathogenesis is essential. Further research on etiology, especially the underlying mechanism of various predisposing factors and the vulnerability of exposed dentin in injury, may provide pragmatic guidance for clinical practice. Lastly, periodical radiography has significant limitations in the detection, assessment and treatment planing of ECR. The increased accuracy of CBCT results in not only more accurate detection and assessment of ECR but also selection of the most appropriate treatment plan. (Fig.6,7)

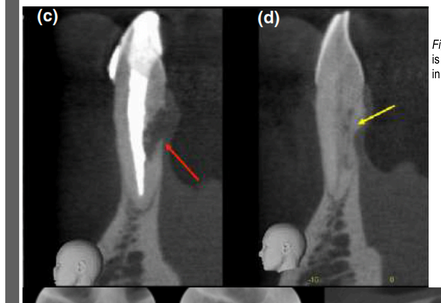

Figure 6. CBCT, sagittal image of teeth #24,25. ECR is in reparative stage in tooth #24 (yellow arrow) and in resorptive stage in tooth #25 (red arrow)

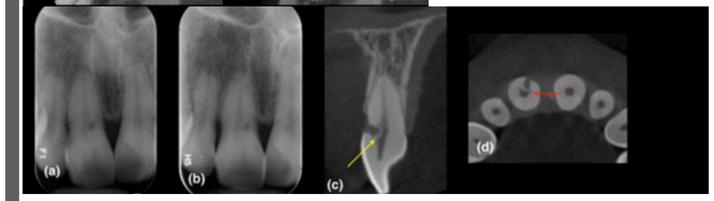

Figure 7. (a, b) PA radiograph of the maxillary left central incisor reveal a radiolucency in the coronal third of the root canal ; (c) saggital and (d) axial CBCT slices confirming the true presence of ECR lesion.
(Foschi et al, 2019)

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