REVIEW ARTICLE

A Review of External Cervical Resorption

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ABSTRACT

External cervical resorption (ECR) is a relatively uncommon yet aggressive form of dental hard tissue destruction. It is initiated at the cervical aspect of the root surface and extends apicocoronally and circumferentially inside the dentin. Despite the large number of case reports and clinical studies that have investigated ECR, its etiology remains unclear. Recent advancements in clinical assessment measures, such as the use of cone-beam computed tomographic imaging, have provided additional insights into the nature of this lesion. This has facilitated the continued development and improvement of treatment methods for this condition. In this article, we provide an overview of the latest research pertaining to the etiology, histopathology, predisposing factors, diagnosis, classification, and treatment of ECR. Furthermore, we provide a summary of the different classification schemes for ECR and highlight the relevant therapeutic principles. *(J Endod 2021;47:883–894.)*

KEY WORDS

Cone-beam computed tomography; diagnosis; etiology; external cervical resorption; treatment

SIGNIFICANCE

Understanding the etiology and histopathology of external cervical resorption may facilitate its prevention and early diagnosis. We provide an overview of relevant therapeutic principles and treatment options that may assist clinicians in the management of external cervical resorption.

Root resorption refers to the loss of dental hard tissue as a result of odontoclastic action[1]. Physiological root resorption occurs during the exfoliation of deciduous teeth and is believed to facilitate the eruption of the permanent successors. In contrast, pathologic resorption involves the permanent dentition and leads to undesirable outcomes[2]. Pathologic root resorption is classified into external and internal resorption based on the site of the lesion. External resorption can be further categorized as surface resorption, inflammatory resorption, replacement resorption, and cervical resorption[2]. External cervical resorption (ECR), also known as invasive cervical resorption, is a specific class of external resorption that initiates at the cervical aspect of the tooth[3]. Clinical and experimental findings suggest that the resorption process is caused by the overproliferation of periodontal ligament (PDL) tissues[4]. Epidemiologic studies in different regions of the world have reported that the prevalence of ECR ranges from 0.02%–0.08%[5,6]. Although the majority of case reports indicate that ECR usually involves a single tooth[5,7], a small proportion of cases involve more than 3 teeth; the latter condition has been termed multiple cervical resorption[8–12].

An increasing number of case studies have reported on the diagnosis and management of ECR in recent years. This has led to significant progress in the understanding of its etiology, histopathology, and predisposing factors. We reviewed the latest research on ECR, with the aim of providing a reference guide for clinical practice.

ETIOLOGY

Studies on the etiology of ECR have mainly focused on 3 aspects, namely the pathogenesis of root resorption, histopathologic analysis of the resorption lesion, and potential predisposing factors[3,13,14]. In recent years, several case reports and retrospective studies have revealed many risk factors for ECR[5,7,15]. 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[7,16].

Pathogenesis

The exact cause and pathogenesis of ECR remain poorly understood[3,16]. This uncertainty has given rise to different perspectives on the nature of ECR. For example, the theory of inflammatory response holds that ECR is initiated solely by an inflammatory response[17–19]; it is assumed that external stimulation (eg, internal bleaching and abnormal external pressure) to the PDL induces an immune response, which in turn activates osteoclastogenesis[20]. Therefore, some researchers have claimed that ECR is an aseptic process[16]. On the other hand, the theory of infection advocates that microorganisms originating from the

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gingival sulcus or pulp are the driving force of ECR[21,22]. This theory posits that resorption can be initiated by either endotoxins or an inflammatory response to microbial infection. Indeed, previous studies have reported an interval between microbial stimulation and ECR that may vary from months to years, thus indicating a chronic pathogenic process. Therefore, ECR induced by microbial stimulation is also referred to as late external resorption[23].

Despite the diverse perspectives on the nature of ECR, the generally held view is that inflammation is a prerequisite for the initiation of resorption[24]. Histopathologic analysis of resorption lesions also suggests that the degradation of dental hard tissues occurs subsequent to an inflammatory response[14]. Nevertheless, the exact correlation between inflammation and osteoclastogenesis in ECR remains unclear[15]. Studies have shown that proinflammatory mediators (eg, interleukin 6 and interleukin 1 beta), which have a primary role in external resorption, are also up-regulated in periodontitis[25–27]. Therefore, osteoclastogenesis in ECR and periodontitis may possibly be similar in mechanism. Whole exome sequencing in patients with ECR has revealed a mutation in the interferon regulatory factor 8 gene (IRF8G388S), which is believed to facilitate osteoclastogenesis[28]. Another study on the differential expression of microRNAs has shed light on the role of epigenetic regulation in initiating root resorption[29]; to be specific, signaling pathways involved in the proinflammatory response, as well as osteoclastogenesis, were found to be activated in tissues undergoing resorption.

Resorptive cells in ECR are morphologically similar to osteoclasts and are capable of degrading dental hard tissues[3,14,30]. Upon the initiation of osteoclastogenesis, a stimulating factor is required to sustain root resorption. The majority of ECR cases have exhibited signs of mechanical stress or inflammation, both of which can cause severe impairment to the periodontal vasculature and result in microcirculation dysfunction[31]. Therefore, ischemia and hypoxia may alter the homeostasis of bone metabolism by enhancing osteoclastogenesis[14,32]. Furthermore, hypoxia can also hinder the recovery of periodontium while concurrently promoting the proliferation of fibrovascular tissue[33].

Histopathologic Structures

ECR usually initiates on the cervical aspect of the root, inferior to the epithelial attachment[3]. A possible explanation for this distinctive feature is that a morphologic defect at the cementoenamel junction (CEJ) increases susceptibility to root resorption[34]. This is supported by the presence of a gap (detected with scanning electron microscopy) between the enamel and cementum at the CEJ in a certain proportion of teeth[35]. In addition, extensive orthodontic treatment or dental trauma may also result in a deficiency of cementum at the cervical region[36]. Compared with cementum and enamel, dentin is more susceptible to resorption because of its higher composition of noncollagenous matrix protein[37,38]. Furthermore, the exposure of arginine-glycine-aspartic acid (RGD) peptides facilitates the recruitment and activation of resorptive cells[39]. Intracanal bacteria and endotoxins may also reach the PDL through dentin tubules, thereby amplifying the inflammatory response[40].

In most cases, the resorption tissue does not penetrate into the pulp space despite the extensive destruction of the tooth structure[3,14]. Therefore, the vitality of the pulp tissue is maintained, which distinguishes ECR from internal resorption. The protective layer, referred to as the pericanalar resorption-resistant sheet (PRRS), is regarded as a prominent structure in the histopathology of ECR[14]. X-ray microfocus computed tomographic scanning has shown that the PRRS has an average thickness of 210 μm and consists primarily of predentin[41,42]. Notably, the predentin layer contains several specific resorption inhibitors as well as antiresorption factors[42]. Thus, rather than extending further toward the pulp space, resorptive tissues proceed circumferentially and apicocoronally once they reach the PRRS[41]. In the terminal stages of ECR, a local disruption or penetration of the PRRS layer may occur; this is followed by the gradual formation of small interconnections between the resorption cavity and the pulp as the resorption extends further toward the pulp space[14].

Pathologic Stages of ECR

ECR is a dynamic and progressive condition that can result in severe impairment of the tooth structure. However, the nature of ECR is considered not only to be destructive but also reparative because of a certain degree of regeneration in its advanced stages[14,19]. Based on histomorphologic observations and clinical examinations, ECR is divided into 3 pathologic stages: the initiation stage, progressive stage, and reparative stage[14].

Initiation Stage

ECR is activated by the disruption of the homeostasis of the periodontal microenvironment, which subsequently triggers localized inflammation. The recruitment of inflammatory cells then facilitates the ingrowth of granulation tissues[31]. Dentinal exposure at the cervical region provides a portal of entry for the granulation tissue to penetrate further into the dentin[35]. Although resorptive tissues are believed to originate from the PDL, they differ from healthy periodontal tissues in both structural and biological behavior[4,43]. The presence of bacteria in the outer layer of the resorption tissues has been confirmed by the assessment of histopathologic sections[14]. In addition to bacterial infection, continuous mechanical forces can also induce and sustain the resorption process[31].

Progressive Stage

The progressive stage is characterized by the penetration of the resorption tissues through the portal of entry and their further advancement toward the pulp cavity[14]. Multinucleated resorption cells, which morphologically resemble osteoclasts, form within the resorption lacunae. Consequently, resorption channels extend circumferentially and/or apicocoronally inside the dentin[14]. In most cases, the pulp cavity is intact due to the protective role of the PRRS[41]; however, disruption of the PRRS and calcification of the extracellular matrix inside the pulp tissue may occur if the resorption tissue comes into close proximity with the pulp cavity[14].

Reparative Stage

A distinctive feature of ECR is lesion repair, which occurs in parallel with the progression stage at different sites on the tooth[14,17]. After the formation of resorption cavities, osseous tissues originating in the adjacent alveolar bone extend into the lesion from the portal of entry[14]. The granulation tissue is replaced by bonelike tissue, and active remodeling is initiated[3]. Depending on the type of tissue adjacent to the portal of entry, lesion progression may result in different outcomes (Table 1).

Potential Predisposing Factors and Distribution Patterns

In view of its low morbidity, ECR was previously considered as an uncommon disease[3,17]. However, the number of case reports and epidemiologic studies on ECR has increased rapidly in recent years, providing a more in-depth understanding of its potential predisposing factors[5,7,15].

TABLE 1 - The Outcome of the Lesion in Accordance with the Tissue Adjacent to the Portal of Entry[14]

Tissue adjacent to portal of entry	Outcome of the resorption defect
Periodontal ligament	Regeneration of periodontium and cementum
Gingival connective tissue	No repairment
Alveolar bone	Ankylosis

Three cross-sectional studies on the distribution and potential predisposing factors of ECR have been performed in Australia, Europe, and Asia, respectively[5,7,15]. Orthodontic treatment, trauma, and restorative procedures were the most commonly cited risk factors. However, the proportion of cases involving each of these factors differed because of regional disparities.

In 1999, the first cross-sectional study on the potential predisposing factors for ECR was performed in Australia (Fig. 1)[5]. A total of 11 risk factors were reported, among which orthodontic treatment (24.1%), trauma (15.1%), intracoronal bleaching (13.6%), and restorative treatment (14.4%) were the most prevalent. Surgery (5.8%), bruxism (2.4%), and periodontal treatment (2.4%) were also cited as risk factors in a relatively large proportion of cases. Under one sixth (14.9%) of the cases were classified as idiopathic ECR because no identifiable risk factors were found[5].

In 2017, another retrospective study conducted in Europe reported that orthodontic treatment (45.7%) and trauma (28.5%) were the predominant risk factors (Fig. 2)[7]. Yet, several novel risk factors were also identified, including parafunctional habits (23.2%), poor oral health (22.9%), malocclusion (17.5%), and the extraction of neighboring teeth (14%). Notably, intracoronal bleaching (2.7%) was not identified as a major risk factor; this was attributed to recent modifications in bleaching agents that have reduced the risk of ECR. The proportion of multifactorial ECR cases was also shown to have increased from 28.9% in 1999 to 59% in 2017. In addition, synergisms were reported for specific predisposing factors, such as orthodontic treatment and occlusal overloading[7].

The latest cross-sectional study was performed in Asia in 2020 (Fig. 3)[15]. A notable difference compared with prior studies was that the proportion of cases associated with orthodontic treatment (15.87%) was much lower compared with orofacial trauma (33.33%) and periodontal treatment (26.98%)[15]. This was attributed to variations in craniofacial patterns as well as perceptions of esthetics among patients from different regions, which may have in turn affected the modality of orthodontic treatment[44,45]. Restorative treatment (17.46%) and tooth extraction (7.94%) were also identified as major predisposing factors. Notably, none of the cases were classified as idiopathic ECR in this study[15].

The majority of ECR cases have been observed in the maxillary arch, with anterior teeth being more commonly affected than premolars and molars[5,7,15]. Indeed, a previous study reported that maxillary central incisors accounted for one fourth of ECR-affected teeth[5]. The higher incidence of ECR in anterior teeth was attributed to the fact that they are subjected to greater tooth movement during orthodontic treatment and are more susceptible to trauma[46]. Two recent retrospective analyses indicated that anterior teeth accounted for half of all ECR cases[47,48]. In terms of posterior teeth, mandibular molars are considered to be the most susceptible to ECR[7].

The age predilection of ECR is strongly linked to specific risk factors. For instance, patients with parafunctional habits are mostly middle-aged, whereas posttrauma and postorthodontic ECRs are primarily observed in younger patients. Nevertheless, the majority of patients evaluated in prior studies have been in the 35- to 60-year-old age group[5,7,15]. Current evidence suggests that ECR does not exhibit a predilection for either sex[5,7,15].

ASSESSMENT

The assessment of ECR has proved to be challenging,[3,13,17] particularly because of its quiescent onset. Although ECR may be incidentally detected during radiographic examination, the majority of cases are not diagnosed until symptoms of pulpitis or apical periodontitis are evident.[13]

Clinical Assessment

The clinical presentation of ECR is determined by the location and extent of the lesion. As the primary step of the treatment procedure, basic endodontic examinations assist clinicians in obtaining a general view of the lesion[13,49].

Inspection

In cases in which the portal of entry is located in the labial/buccal or lingual/palatal aspect of the tooth, a pink discoloration may be visible on the crown. As a pathognomonic feature of ECR, this pink spot is believed to be a result of the granulation tissue becoming visible through the impaired dental hard tissue overlying the resorption cavity[10,12]; however, it may not be apparent if the lesion is located on the interproximal or palatal aspect of the tooth. This pink spot may also not be readily detected if the resorption cavity extends apically rather than coronally on the root[3,17].

Probing

The texture of the resorption cavity is an important feature for the differential diagnosis of ECR and subgingival caries. Caries generally feel soft and sticky on probing, while the texture of ECR lesions are similar to that of dentin[11]. When probing the base of the resorption cavity, a scraping sound may be heard, and the resorption tissue may bleed profusely[3,13].

Tooth Mobility

The following grades of tooth mobility may be assigned based on its severity: grade I mobility (labial-palatal direction) or grade II mobility (labial-palatal direction and mesial-distal direction)[12]. Tooth mobility in cases of ECR is due to the degradation of dental tissue at the cervical section. Nevertheless, it should be noted that tooth mobility is seldomly reported because it only occurs in the advanced stages of the lesion.

Gingival State

The gingival contour appears slightly irregular in the early stages of ECR[17]. Swelling and hyperemia of the gingiva have also been reported[9,12]. Although these features may facilitate an early diagnosis of ECR, they are not distinctive features of this condition.

Pulp Cavity Condition

Because the pulp space is spared by the resorptive cells in the majority of cases, the affected tooth shows a positive response to sensibility tests[49]. However, in the advanced stage of the lesion (during which the resorption tissue penetrates through the PRRS), patients may report symptoms of pulpitis or apical periodontitis and respond negatively to the sensibility test[13,50].

Radiology

Because of a lack of pathognomonic clinical features, the definitive diagnosis of ECR is highly dependent on imageological examination[51,52]. Although ECR does not have diagnostic radiographic features, its radiographic presentation is generally

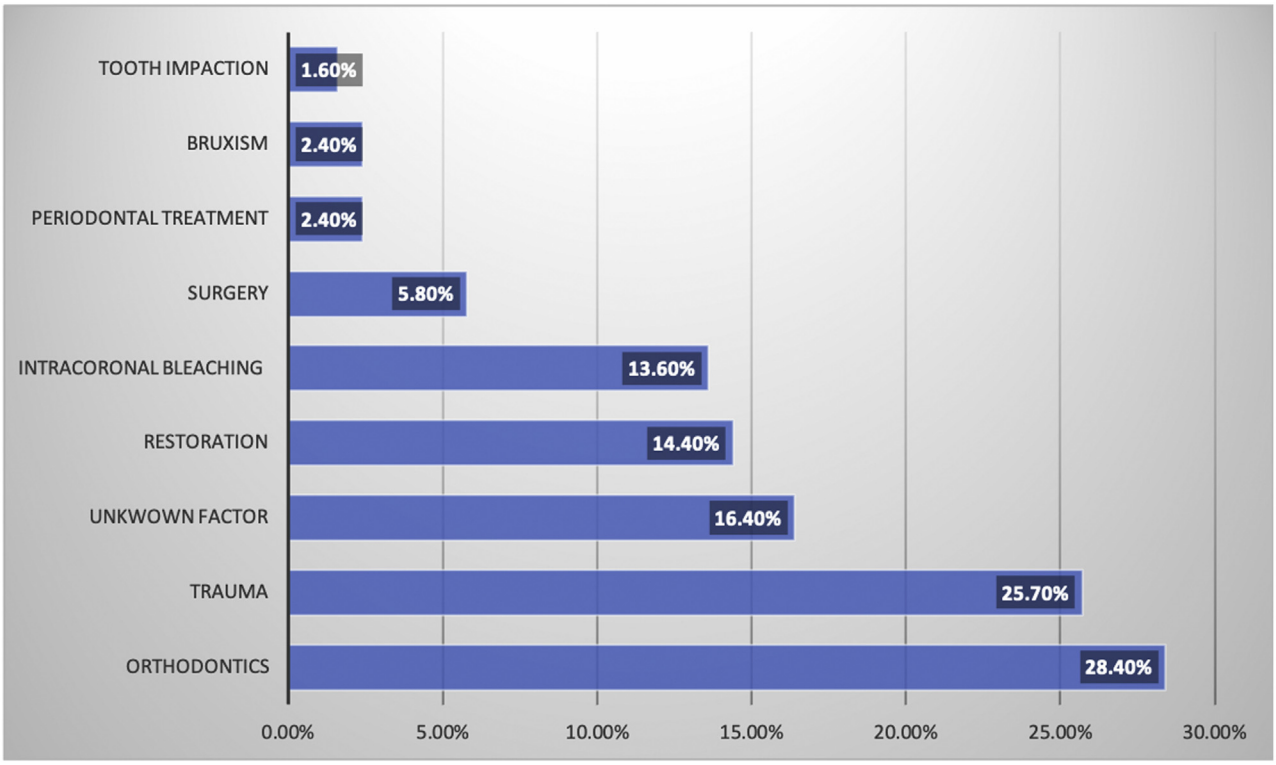

FIGURE 1 – The distribution of major potential predisposing factors identified in a study in Australia[5].

consistent with the extent of the lesion[13,17]. Thus, current classifications of ECR are mainly based on imageological manifestations[20,53].

The periapical radiograph (PR) has been traditionally considered to be the imaging modality of choice for the evaluation of ECR[17]. However, its disadvantage is the inability to reveal the 3-dimensional structure of the lesion[16]. Clinicians often encounter difficulties in detecting early lesions, which lead to an underestimation of the ECR incidence rate[54,55]. However, once a lesion is detected, there are further difficulties in assessing its extent with a PR because of the complex anatomy of the lesion as well as the proximity

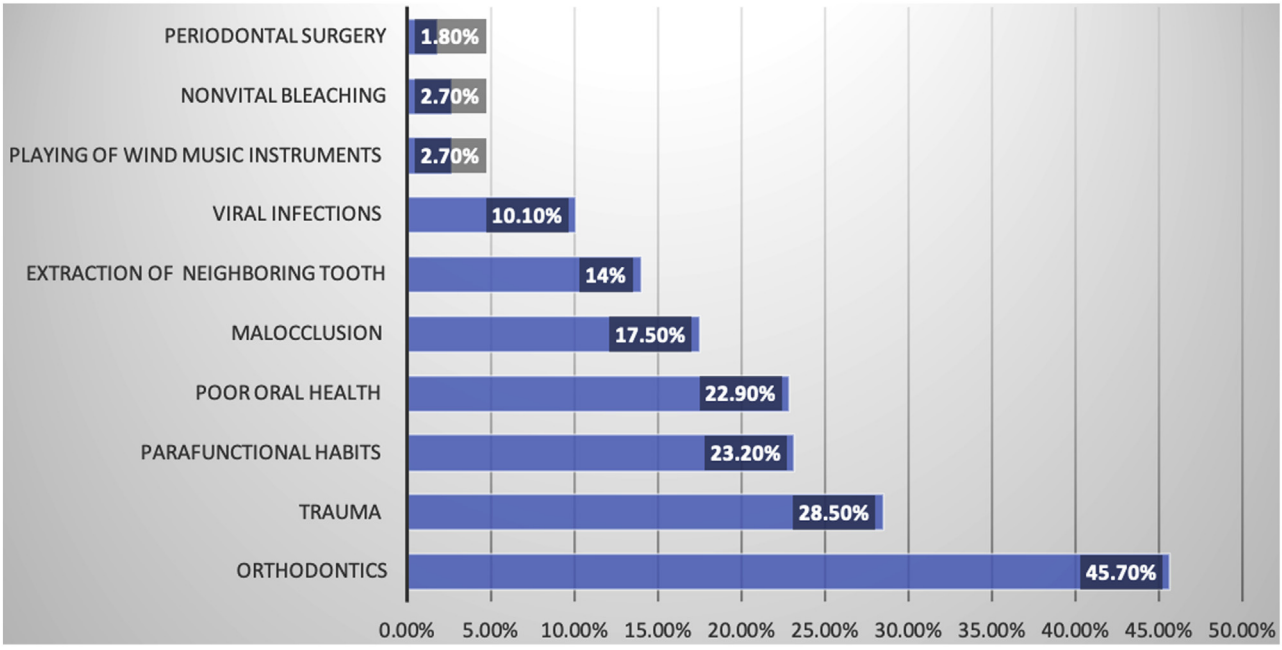

FIGURE 2 – The distribution of major potential predisposing factors identified in a study in Europe.[7]

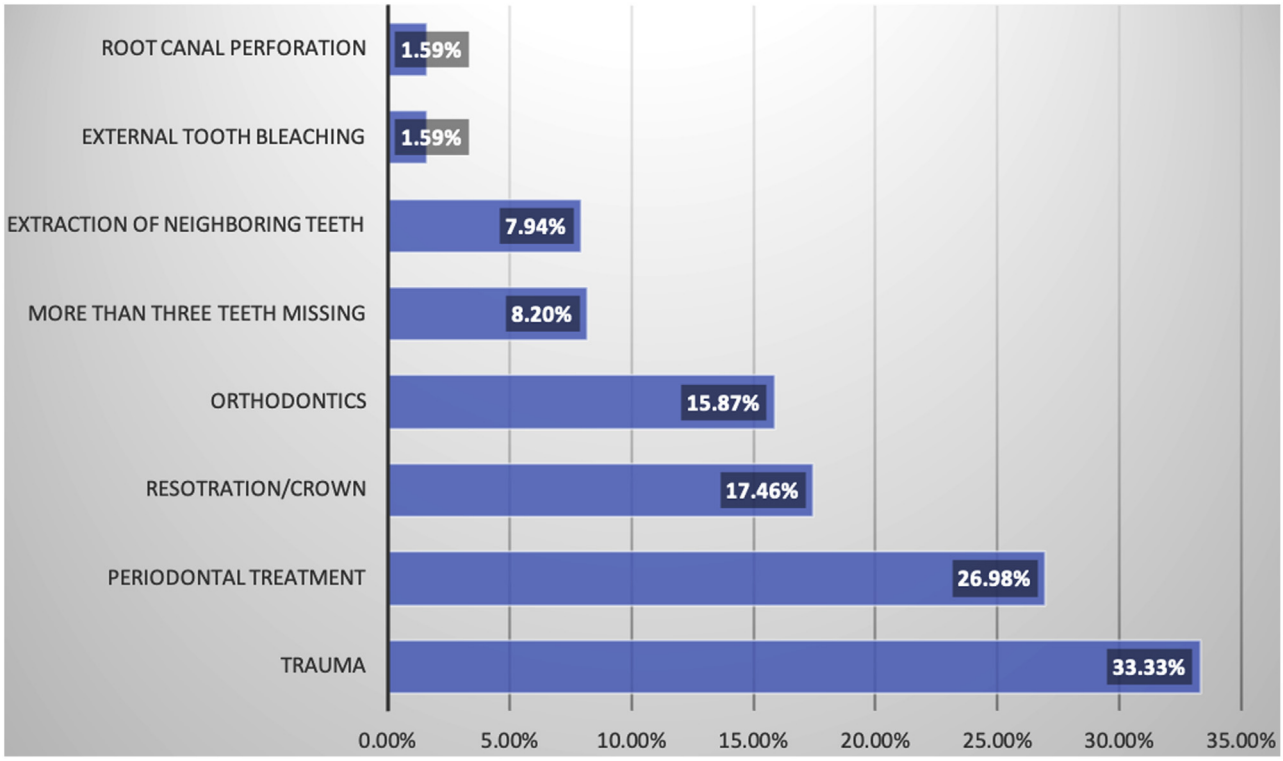

FIGURE 3 – The distribution of potential predisposing factors identified in a study in Asia.[15]

of the adjacent structures[56]. This is especially the case when the portal of entry is located on the buccal, lingual, or palatal aspect of the root; these locations are involved in 72% of ECR cases. Another disadvantage of PRs is that they cannot provide information on potential pulp involvement[48].

In terms of diagnostic accuracy, cone-beam computed tomographic (CBCT) imaging has been shown to be superior to PRs in several *ex vivo* studies[57–59]. CBCT imaging is also better able to detect small resorption cavities; this suggests its potential ability to identify incipient lesions[60]. The reconstruction of spatial structures enables clinicians to assess a lesion on any plane, thereby avoiding the interference of overlying tissues[52]. Indeed, CBCT imaging is capable of accurately locating the portal of entry, the extent of resorption, and the proximity of the resorption tissue to the pulp space; all of these parameters are crucial for treatment success[61]. A previous study comparing the ability of PRs and CBCT imaging to detect ECR lesions reported a sensitivity and specificity of 78.18% and 59.52%, respectively, for PRs; the corresponding values for CBCT imaging were 97.27% and 97.62%, respectively[62]. A similar study that carried out a receiver operating characteristic analysis reported a significantly higher value for CBCT imaging than PRs[63]. Furthermore, CBCT imaging has been shown to have a better capability in assessing whether a lesion is in the stage of arrest, repair, or progression in the follow-up period after treatment[64].

Position statements made by the American Association of Endodontists as well as the European Society of Endodontology have both highlighted the importance of CBCT imaging in the assessment of ECR[56,65]. The American Association of Endodontists statement also recommended applying a limited field of view CBCT scan to reduce the radiation dose and obtain a higher spatial resolution. However, in cases of multiple cervical resorption, large field of view CBCT imaging may be the preferred choice[56]. It is worth emphasizing that CBCT imaging should not be applied for screening purposes. Instead, inquiry and clinical inspection should be performed before radiologic examination[56].

In addition to improving diagnostic accuracy, new classifications based on CBCT findings provide a more comprehensive understanding of ECR. A significant improvement is the establishment of a 3-dimensional description of the resorption cavity, which facilitates clinicians in assessing the circumferential extension of a lesion[53]. Moreover, by virtue of the higher resolution in CBCT images, new classifications have been able to account for the proximity of the lesion to the pulp space[66]. However, it is notable that current classifications do not consider the reparative nature of ECR; thus, further modifications are required[67,68].

The extent of the radiolucent area on radiographs corresponds with the pathologic stage of ECR[20]. The early phases of resorption can be observed as a diminutive spot at the CEJ. As resorption proceeds, the radiolucent area extends toward the pulp space or the coronal region[3,17,49]. In the early stages, it may be difficult to differentiate ECR from cervical burnout on PRs; the latter is characterized by a radiolucent band or triangular radiolucent area on the mesial, distal, or both aspects of the root[3].

In the advanced stages of the lesion, resorption tissues progress toward the pulp space and extend coronally and apically; there is a corresponding enlargement of the boundary of the radiolucent area[13,17,49]. The profile of the lesion may appear symmetrical or asymmetrical, with varying dimensions[69]. The PRRS can be identified as an intact radiopaque line, which distinctly separates the root canal from the adjacent resorption tissue[13]. Another noticeable feature is that the

TABLE 2 - The Heithersay Classification 5[5]

Heithersay classification	
Class 1	Denotes a small invasive resorptive lesion near the cervical area with shallow penetration into dentin
Class 2	Denotes a well-defined invasive resorptive lesion that has penetrated close to the coronal pulp chamber but shows little or no extension into the radicular dentin
Class 3	Denotes 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	Denotes a large invasive resorptive process that has extended beyond the coronal third of the root canal

adjacent alveolar bone remains uninvaded unless there is a superimposed PDL infection[21].

The reparative stage of ECR is characterized by a mottled or cloudy region, which represents the fibro-osseous tissues. Compared with the progression stage, resorption cavities appear more radiopaque because of the ossification of the granulation tissue[13,20]. Disruption of the PRRS may result in local calcification of the pulp tissue, which can be observed as radiopaque spots in the pulp space[17].

CLASSIFICATIONS

Heithersay Classification

The Heithersay classification is based on the assumption that lesions extend coronally in the early stages and apically in the advanced stages[5]. It uses a 4-point classification system, and the features of the different stages correspond with the extent of resorption identified on the PR (Table 2 and Fig. 4). The Heithersay classification was the first system used to classify ECR lesions and was designed to guide treatment and facilitate the determination of patient prognosis[3]. Despite the establishment of several new classification systems, the majority of guidelines on clinical therapy are still based on the Heithersay classification because of its broad scope of application[3,70].

Patel Classification

The Patel classification (Fig. 3) is a 3-dimensional classification and covers 3 main aspects of the lesion: coronal-apical extension, circumferential extension, and proximity to the root canal (Table 3)[53]. As the first classification system to accurately delineate the spatial structure of ECR, it significantly improves diagnostic accuracy and assists clinicians in assessing the outcome of treatment[71]. The Patel classification provides a more accurate depiction of the resorption defect compared with PRs, which are prone to underestimate the scope of the lesion[48]. Therefore, the Patel classification can play a significant role in shaping future research of ECR[71].

Rohde Classification

The Rohde classification system is a 3-point classification system and is based on CBCT findings[72]. Two important aspects of ECR are taken into account: the amount of dentin loss in the cervical area and the amount of dentin loss on the external surface of the tooth. Based on these 2 features, ECR lesions are divided into 3 classes:

(1) significant dentin resorption in less than one third of the tooth circumference;
(2) significant dentin resorption in less than one third of the tooth circumference, with a presumptive perforation defect (2.5 mm or larger) in any dimension; and
(3) significant dentin resorption involving greater than one third of the tooth circumference. The term *significant dentin resorption* in the Rohde classification system refers to a degradation of more than one half of the original axial wall[72].

TREATMENT

It is generally held that the effective management of ECR is highly dependent on the accurate evaluation of lesion severity[70]. The percentage of mineralized dental tissue affected by resorption is an important factor when selecting the optimal treatment option. The amount of remaining intact tooth structure also has a significant influence on the prognosis[48].

Objectives and Principles

On account of the destructiveness caused by resorption tissues, the primary objective of treatment focuses on inactivating the resorption process[3,70]. Subsequent restoration of the affected dental tissues facilitates the recovery of function and esthetics. The prevention of recurrence is also necessary in order to improve the patient's long-term prognosis[48].

Based on the objectives and treatment options previously mentioned in the literature, we put forward 4 basic therapeutic principles in the order of their significance: thorough removal of resorption tissue, complete sealing of the resorption cavity, minimally invasive operating methods, and retention of the affected teeth.

Therapeutic Options

The extent and location of the lesion are the 2 primary prognostic factors for ECR[71]. The complexity of treatment can be reflected by 2 indicators: curability and accessibility[70,73]. The former represents the extent of the lesion, whereas the latter is primarily determined by the location of the resorption cavity. Basic treatment options fall into 2 broad categories: surgical approaches and nonsurgical approaches. Flap surgery and root canal treatment (RCT) are the 2 most commonly applied methods, whereas intentional replantation (IR), periodic review, and extraction have also been put forward in case reports[49,70]. Nevertheless, no standard protocol for the treatment of ECR has been proposed to date[73]. Lesions are divided into 4 classes based on their curability and accessibility, and treatment options are in line with lesion classification (Table 4).

External Repair

External repair applies to Heithersay class 1 and 2 lesions, which are confined to the cervical and coronal regions. Gingival flap surgery is required to provide visualization and access to the resorption cavity[74]. Complete elimination of the resorption tissue separates the lesion from the adjacent periodontium, which is essential for preventing the recurrence of resorption[3,71]. Because there is difficulty in detecting and accessing diminutive resorption channels with dental instruments, this procedure requires a combination of mechanical curettage and chemical debridement. Topical application of 90% aqueous trichloroacetic acid (TCA) is believed to facilitate coagulation necrosis of residual resorption tissue while leaving the adjacent periodontium intact[69,75]. However, it is important to note that TCA is an aggressive agent and can cause severe chemical burns to the oral mucosa if used inappropriately[76]. Therefore, TCA should be applied with caution. The resorption cavity should then be restored with composite resin or glass ionomer cement[77]. The portal of entry, which is exposed to the oral environment, should be

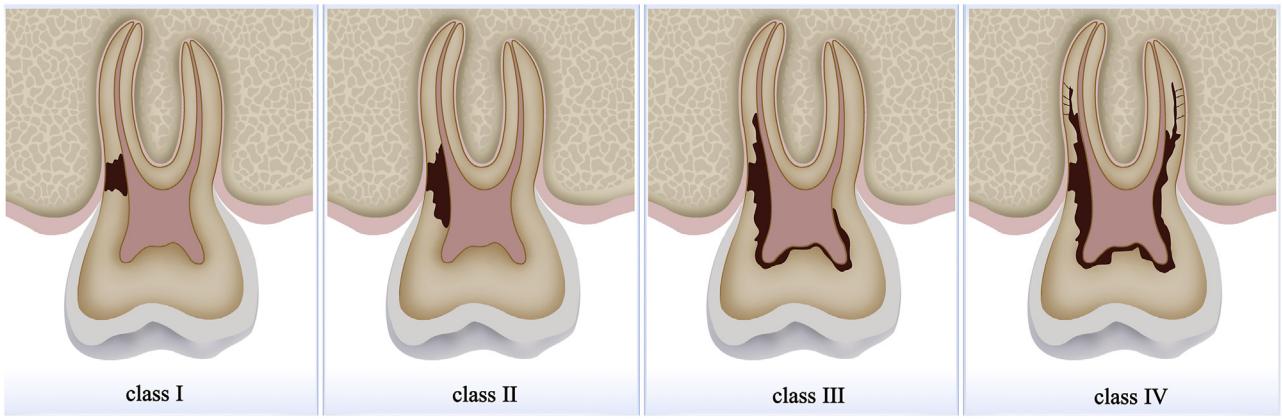

FIGURE 4 – A schematic diagram of Heithersay classification[5].

sealed tightly to prevent the ingrowth of newly formed granulomatous tissue[3,70].

Pulp capping is the preferred treatment option when resorptive tissues are in close proximity to the pulp space and the PRRS is disrupted[77,78]. Mineral trioxide aggregate (MTA) has gained wide acceptance as a pulp-capping agent because of its favorable properties, including excellent sealing capacity, biocompatibility, bactericidal effect, radiopacity, and applicability in the presence of blood[79,80]. Lesions treated with MTA may eventually form a noninflammatory layer, thereby hindering the ingrowth of resorption tissues[81] Furthermore, studies have suggested the potential of MTA to promote the formation of reparative dentin and cementum, which facilitates the forming pf periodontal reattachment as well as functional reconstruction[17].

Complete curettage of the granulomatous tissue may be difficult for advanced resorption defects with extensive coronal-apical and circumferential extension (Heithersay class 3 or 4). This may be especially obvious when CBCT examinations reveal a localized and narrow portal of entry at the root surface. In these cases, flap surgeries may require the removal of a significant amount of bone and dentin to expose the lesion; nevertheless, complete removal of the granulomatous tissue may not be achieved, and the procedure may result in a poorer periodontal prognosis[81]. Lesions with palatal or lingual portals of entry are also considered to be inaccessible via external treatment[48].

Internal Repair

Internal repair refers to nonsurgical approaches through which resorptive tissues are removed mechanically or chemically by RCT[70]. It is used in cases in which external approaches are not practical because of the inaccessibility of the lesion. This inaccessibility may be the result of resorption tissues perforating the PRRS or the portal of entry being located apical to the alveolar crest[70]. Under these circumstances, flap surgeries may require the removal of a significant amount of bone and dentin to access the lesion, which could result in excessive damage to intact dental tissues[49]. In advanced-stage lesions, an interconnection between the resorption cavity and the root canal is likely to be detected[14]; in these cases, internal repair may provide a better access to the lesion. Both the resorption cavity and the access cavity should be restored after endodontic treatment. The prognosis of intractable cases has also been shown to improve with internal repair[17,70].

Palliative Treatment

In a minority of cases, lesions appear curable but remain inaccessible with flap surgery or RCT. In such situations, palliative treatment (eg, IR and periodic review) may be provided as a last resort to preserve the tooth[70].

IR refers to the reinsertion of an endodontically treated tooth that was previously extracted[82]. The interval between extraction and replantation should be limited to 15 minutes, during which the resorption cavity undergoes debridement and restoration. Strict limits on operating time contribute to the healing of periradicular tissues[83,84]. With appropriate treatment, the functional state of the replanted tooth may be retained. Although improvements in bioactive materials and modifications in treatment protocols have significantly improved the prognosis of replanted teeth, clinicians should be cautious in applying IR[83,85]. Contraindications to IR should be thoroughly reviewed before treatment[82], and long-term follow-up is necessary to monitor possible complications[86].

Periodic review is regarded as a compromise solution between extraction and IR. The progression of the resorption is carefully monitored at regular intervals[70]. The affected teeth should be extracted if the patient reports symptoms associated with pulpitis or apical periodontitis.

Extraction

Extraction is the final choice for untreatable cases[3]. Extensive degradation of the cervical dentin may result in a poor prognosis after treatment[48]. Atraumatic extraction techniques are preferred to minimize bone destruction and trauma[87].

TABLE 3 - The Patel Classification[53]

Height	Circumferential Spread	Proximity to the root canal
1: Supracrestal/at the cementoenamel junction level	A: ≤90°	d: Lesion confined to the dentin
2: Subcrestal and extends into the coronal one third of the root	B: >90° to ≤180°	p: Probable pulpal involvement
3: Extends into the mid one third of the root	C: >180° to ≤270°	
4: Extends into the apical one third of the root	D: >270°	

TABLE 4 - The Classification of Lesions and Treatment Options

Category	Curability	Accessibility (external)	Accessibility (internal)	Option
Class 1	+	+	+	External repair (flap surgery)
Class 2	+	−	+	Internal repair (root canal treatment)
Class 3	+	−	−	Palliative treatment
Class 4	−	−	−	Extraction

Prognosis

A retrospective observational study on the treatment outcomes of patients who underwent flap surgery, direct curettage, and restoration reported a survival rate and treatment success rate of 100% and 79%, respectively, at a 20-month follow-up[71]; treatment success was defined as endodontic success, comprehensive restorative integrity, and arrest of the resorptive process. Unsuccessful treatment was mainly due to the continued progression of the resorption, as well as a suboptimal restoration integrity[71]. Although the ability to arrest the progression of resorption is a crucial measure of treatment success, no current methods have been reported to reliably achieve this. Bisphosphonate medication has been considered as an option to prevent the progression of the resorption[88]; however, it is not widely accepted because of an increased incidence of complications (eg, osteonecrosis), especially for patients who undergo implant surgeries[89]. Calcitonin may be a potential alternative because it has been shown to inhibit odontoclast activation and suppress the inflammatory response[90].

It is noteworthy that certain complications such as tooth loss are inevitable, especially for patients with advanced ECR. Therefore, it is necessary for patients to be informed in advance. Moreover, clinicians ought to weigh the pros and cons of each therapeutic option before treatment. This is especially important when treating young patients because their craniofacial growth has not yet been completed[91,92].

CONCLUSION

ECR is a progressive disease that can cause severe damage to dental hard tissues. However, because early lesions often appear to be latent, ECR is likely to remain unnoticed until it is in its advanced stages. Thus, clinicians should be vigilant of subtle changes that suggest the occurrence of ECR. Patients with known risk factors require special attention and should be advised to undergo careful examination to exclude the possibility of ECR. To date, the etiology of ECR remains unclear, and the elucidation of potential predisposing factors requires further research.

Recent advancements in clinical assessment measures have greatly improved diagnostic accuracy. The most prominent example is the use of CBCT imaging to detect incipient ECR, which facilitates timely intervention and leads to better treatment outcomes. Modifications to existing treatment protocols have also been shown to improve the prognosis of advanced-stage lesions. Nevertheless, therapeutic guidelines based on newly proposed classifications have not yet been established. In view of the widespread popularity of CBCT imaging, it is necessary to put forward new guidelines for ECR treatment.

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