REVIEW PAPER

EXTERNAL CERVICAL RESORPTION – CURRENT APPROACH TO ETIOLOGY, DIAGNOSTICS, AND CLASSIFICATION

Wojciech Eliasz[1], Maria Gawriołek[2], Kinga Kubiak[3], Piotr Pukacki[3], Robert Juszkat[3], Anna Surdacka[1]

[1]Department of Conservative Dentistry and Endodontics, Poznan University of Medical Sciences, Poznan, Poland
[2]Department of Integrated Dentistry, Poznan University of Medical Sciences, Poznan, Poland
[3]Department of General and Interventional Radiology, Poznan University of Medical Sciences, Poznan, Poland

ABSTRACT

External cervical resorption lesions (ECR) are becoming more and more frequently reported in dental literature, which is related to the fact that patients retain their natural teeth for longer, and also to an increase in the popularity of three-dimensional diagnostic imaging modalities. ECR constitutes an entity whose treatment and diagnostics remain difficult, and the prognosis for teeth affected by the disease is still relatively poor. Even though research studies have been conducted in various research centers worldwide, and even though the histopathological pattern and molecular events that accompany the development of ECR have been studied to a significant extent, the clinical factors that contribute to initiation and progression of ECR lesions have not been determined yet. The aim of the paper is to present the current state of knowledge concerning ECR etiology and diagnostics, and to present the current 3D classification system of ECR lesions that is based on cone beam computed tomography findings, as well as the clinical classification involving the accessibility of the lesion. The data may facilitate the choice of the most appropriate treatment method in patients who present with all stages of ECR, from the early ones, in which conservative treatment has a relatively good prognosis, to the most advanced ones, in which extraction is the treatment modality of choice.

Key words: tooth resorption, cone beam computed tomography, invasive cervical resorption.

J Stoma 2019; 72, 1: 29–33
DOI: https://doi.org/10.5114/jos.2019.86493

INTRODUCTION

Tooth resorption processes are characterized by irreversible loss of enamel, dentine, and cement; they develop due to a variety of factors that promote dental hard tissue loss [26]. Basically, such lesions can be classified into physiological and pathological resorption. Resorptive processes starting from the apex of a deciduous tooth constitute a physiological phenomenon in exfoliation, and are crucial for maintaining appropriate function within the stomatognathic system. In permanent teeth, the occurrence of resorption constitutes a symptom of various pathological processes developing within the tooth, both odontogenic and of other etiologies [4]. Such processes may further be classified into internal and external resorption. In the former type, the odontoclastic process takes place within the inside pulp chamber and the root canal. Such disorders may, eventually, lead to perforation and communication between the pulp and periodontium [25]. External resorp-

JOURNAL OF STOMATOLOGY
CZASOPISMO STOMATOLOGICZNE

Address for correspondence: Wojciech Eliasz, Department of Conservative Dentistry and Endodontics, Poznan University of Medical Sciences, 70 Bukowska St., 60-567 Poznań, Poland, e-mail: wojciecheliasz@gmail.com

Received: 30.01.2019 • Accepted: 29.05.2019 • Published: 28.06.2019

tion is another type of lesion that develops primarily on the tooth surface, until it reaches the pulp chamber or root canal lumen.

External resorption lesions may be further classified into several types, according to the location of the primary focus and the features of the process [22]. The following types of external resorption have been identified in the endodontic literature: surface resorption, external inflammatory resorption, external cervical resorption, external replacement resorption, and transient apical resorption.

Within the last two decades, the process of external cervical resorption (ECR) has become a popular topic for laboratory and clinical studies, which can be attributed to both the fact that patients retain their own teeth for a longer period of time, and to an increase in the efficacy of diagnostic imaging, particularly thanks to the introduction of 3D imaging techniques – cone beam computed tomography (CBCT) in particular [3]. Therefore, the aim of the paper is to communicate the most contemporary views on ECR etiology, diagnostics, and classification involving management options. A literature search was performed using the PubMed database (https://www.ncbi.nlm.nih.gov/pubmed/) with the keywords *external cervical resorption, invasive cervical resorption, tooth resorption, root resorption*, and the most comprehensive papers published until November, 2018, were selected.

EXTERNAL CERVICAL RESORPTION ETIOLOGY

ECR develops primarily within the cervical portion of the affected tooth and is characterized by dynamic destruction of marginal periodontium tissues and, eventually, dental pulp. Destruction of the periodontal ligament (PDL) or disorders affecting the functioning of its cells constitute the primary reason for the development of such lesions [7]. However, it has not been established until today what factors are responsible exactly for this phenomenon. Due to its rather rare incidence, the majority of studies that have been published are case reports/series, and histopathological or molecular reports constitute a small part of the literature. Therefore, it is difficult to implement statistical methods for determining what factors have a statistically significant influence on the development of ECR.

Such lesions may develop both in vital and non-vital teeth, even after root canal therapy [16, 18]. The largest number of cases was analyzed and published in the articles by Heithersay and Mavridou [10, 17]. Both studies aimed to determine the factors that may predispose the patient to ECR development. After all datasets were analyzed, the authors determined several predisposing factors, including orthodontic treatment, dental trauma, parafunctions, bad oral hygiene, malocclusion, extraction of a neighboring tooth, viral infection (e.g. herpes zoster or feline herpes virus), orthognathic surgery procedures, playing wind instruments, non-vital tooth bleaching, disorders affecting tooth eruption, systemic disorders, systemic drug therapy (e.g. bis-phosphonate use), cracks, periodontal surgery, conservative and endodontic treatment, pressure related to the type of frenum, and interproximal stripping [24]. Moreover, in many cases, the development of ECR was not associated with any of the aforementioned factors and could not be explained. It should be underlined that ECR developed in more than 50% of cases after several factors were present in the same patient, which seems to indicate that the etiology of the disease may be multifactorial [27]. Due to the fact that both studies were published 18 years apart, Patel *et al.* tried to compare the incidence of all factors and observed a decline in the incidence of ECR after non-vital tooth bleaching, which may result from the fact that more aggressive procedures are used less frequently nowadays [24]. A growing tendency was observed for the relationship between ECR incidence and orthodontic treatment. It may be explained by the rise in popularity of orthodontic treatment, as well as by an increasing awareness of the need of comprehensive treatment of oral disorders. Viral infection is an interesting example of an etiological factor. It has been suggested in the literature that herpes infection, and also feline herpes, predispose the patient to ECR development [1, 13]. The incidence of such infection in cats is estimated at 14.3-85% and results in development of feline odontoclastic resorptive lesions (FORL), also known as tooth resorption [8, 15]. Von Arx *et al.* [1] were the first to suggest the relationship between the occurrence of multiple ECR foci after virus transmission from the cat to the human. Nevertheless, no further studies could clarify this disorder.

HISTOPATHOLOGY OF EXTERNAL CERVICAL RESORPTION

The histopathological image of ECR consists of several characteristic zones, out of which the *portal of entry* is the first site at which the lesion initiates [9]. ECR may develop only after a local destruction of PDL has occurred, including its rupture. Then, two phenomena occur – firstly, the development of granulation tissue covered by a layer of connective tissue with areas of dense lymphoplasmacytic infiltration and hyperplasia of epithelial tissue. [19]. Secondly, reparative tissue, which resembles bone, grows into the affected area. This may result in fusion of osseous tissue with resorbed enamel and dentine. Also, local cement destruction is observed at this stage, and, simultaneously, a blood clot forms. It impairs the physiological blood supply and promotes inflammation that affects dentine [24]. Macrophages then migrate to the affected area. Apart from "cleaning" the ECR site, they stimulate the development of granulation tissue. Due to dentine exposure, granulation tissue

touches it directly, and the cells that migrate there may stimulate various processes. If osteoblasts prevail, ankylosis is most likely to occur. ECR develops if the clastic cell count is higher. They are activated mainly due to binding of RANKL protein (Receptor activator of nuclear factor kappa-B ligand) that stimulates osteoclast maturation and activation [29].

As the process approaches the root canal lumen, internal channels and interconnections are formed between several resorptive foci within one lesion. The presence of multi-nuclear resorptive cells, also known as clastic cells, is characteristic for this process. They are approximately 20-30 μm in diameter and they resemble osteoclasts [2]. The resorptive process develops in all directions, and its progress depends on inflammatory mediators and endocrine factors, such as parathyroid hormone or calcitonin. The pericanalar resorption-resistant sheet (PRRS) constitutes the deepest part of the lesion [19]. It consists of dentine and, in some cases, tissue that resembles bone, and it is 70 to 490 μm thick. It constitutes a kind of a barrier that protects the pulp from resorption. However, in severe cases, it may be broken and different lesions within the dental pulp may develop. Such lesions include calcification foci (both pulp stones and diffuse calcification), root canal hyalinization, and increased deposition of predentin. It is not understood in what way this layer may protect the pulp from clastic cells, and only two hypotheses have been proposed. According to some authors, PRRS contains an antiresorption factor, while according to others its resistance is related to the lack of proteins containing the RGD peptide sequence (Arg-Gly-Asp) [12, 30]. The sequence is recognized by transmembrane integrins – αvβ3 dimers – whose expression is high in osteoclasts and which are responsible for their adhesion to other cells [11] .

At the same time, reparative processes take place. They are characterized by the ingrowth of bone into the cavity and remodeling of osseous tissue. The processes are fast and dynamic within this layer, which makes the clinical and radiographic picture of lesions that developed for a longer period of time, non-homogeneous foci [20].

EXTERNAL CERVICAL RESORPTION DIAGNOSTICS

Clinical diagnostics of ECR is neither easy, nor straightforward. In the majority of cases, its course is asymptomatic, particularly at earlier stages [5]. They may be diagnosed accidentally in radiography, and computed tomography provides a more precise image of its range and extent. In clinical examination, initial lesions may be mistaken for class V caries (according to Black's classification). Lesion consistency is an important feature for differentiation – caries cavity is softer and sticky; ECR lesions, on the other hand, are hard, and their surface is rough. In more advanced stages, when granulation tissue shows through the enamel, a pink spot or band may be visible in the cervical region [28]. This symptom is easier to see in front teeth, and spotting it in posterior teeth may be difficult for the patient and the clinician [23].

CONTEMPORARY EXTERNAL CERVICAL RESORPTION CLASSIFICATION SYSTEMS

The literature provides several systems for classifying such lesions. The majority of them, however, are based on the clinical picture and periapical radiographs. Due to the fact that these systems are of limited usefulness in providing an accurate image of the lesions, a new ECR classification was prepared on the basis of 3D evaluation of the lesion extent. CBCT scans are required in order to use it. Data from research studies indicate that inter-rater agreement in ECR diagnosis using CBCT is high [14]. Nowadays, two classification systems may be used – one involving its picture in radiographs according to Patel *et al.*, and the second based on the possibility of accessing the resorptive lesion, according to Espona *et al.* [6, 22].

The classification system proposed by Patel includes three indices: resorption height, its spread within the circumference of the tooth, and pulpal involvement [24]. Resorption height is defined as its extent in the vertical dimension. The value 1 describes lesions that develop supracrestally up until the level to the cemento-enamel junction (CEJ); 2 – subcrestally, within the coronal 1/3 of the root canal, 3 – lesions that extend to the middle 1/3 of the root canal, 4 – lesions that involve the apical 1/3 of the root canal. The second category, i.e. the extent of the lesion within the perimeter of the tooth, consists of 4 classes: A-D. Category A includes lesions that affect less than A ≤ 90° of the tooth circumference, B – 90°-180°, C – 180°-270°, D > 270°. The last criterion, i.e. pulpal involvement, includes two categories: d – if the lesion is confined to dentine, p – if there is (even probable) pulpal involvement A short summary of the classification is presented in Table 1.

Such classification makes it possible to evaluate the lesion comprehensively and to plan treatment in a more predictable manner. It should be underlined that such lesions are often progressive, which, eventually, leads to tooth loss. Appropriate assessment of the extent of ECR for the purpose of implementing relevant therapy may prevent the destruction of crestal alveolar bone. Such a deleterious effect may make prosthodontic rehabilitation impossible in future. The team by Patel *et al.* provided a systematic approach to management strategies that facilitate implementation of appropriate therapy in patients with ECR [21].

According to the system proposed by Espona *et al.*, ECR is classified into three classes, depending on the possibility of accessing the lesion [6]. The three classes are E (external access), I (internal access), and N (no access).

TABLE 1. External cervical resorption classification criteria according to Patel *et al.* (2018)

Resorption height	Circumferential spread	Involvement of the pulp
1 – supracrestal lesion (coronal to bone crest level or at cemento-enamel junction)	A: ≤ 90°	d – the lesion affects only dentin
2 – subcrestal, affecting the coronal 1/3 of the root	B: > 90°; ≤ 180°	p – pulpal involvement may be probable
3 – lesion that involves the middle 1/3 of the root	C: > 180°; ≤ 270°	
4 – lesion extending into the apical 1/3 of the root	D: > 270°	

Class E lesions include resorption foci that can be approached after flap elevation. After the surgical procedure is performed, the tissue is removed from the focus, and the tooth may be restored with conventional restorative materials, e.g. composite resin. For class I lesions, the focus of resorption can be accessed from the inside of the tooth during conventional endodontic treatment – external access would cause unnecessary removal of hard tissue. After the tissue is removed, the cavity is restored using bioceramic cement and another restorative material to cover it. Class N lesions are considered non-restorable, as no access is feasible in such cases. The only treatment option involves intentional replantation after removal of granulation tissue.

EXTERNAL CERVICAL RESORPTION TREATMENT

If the extent of the lesion is not large, e.g. 1Ad, 2Ad, 2Bd according to Patel *et al.*, and the lesion can be accessed from the outside (E, according to Espona *et al.*) [6, 22], a surgical procedure involving flap elevation, granulation tissue removal (mechanical, using burs and ultrasonic devices, and chemical, using trichloroacetic acid or sodium hypochlorite), and restoration of the defect with a bioceramic, conventional glass-ionomer, or resin-modified glass-ionomer material. Composite material may also be used if appropriate moisture control can be achieved. If the extent according to CBCT is larger and it may be accessed from the inside of the tooth (class I according to Espona *et al.*) – particularly for 2Cp, 2Dp, 3Cp and 3Dp, lesions – endodontic therapy may be the best option, followed by appropriate restoration placement. The most radical procedure, reserved for large lesions that cannot be accessed, involves tooth extraction, cleaning of the affected site outside of the oral cavity, and restoration with appropriate material (bioceramic cements are indicated in this case), followed by tooth replantation. Extraoral time must be limited to 15 minutes, and splinting should be performed according to the generally accepted guidelines for replantation procedures, which is usually approximately 2 weeks. If the lesion is so large that it involved the majority of dental tissue, and if the patient does not express consent to extraction, a "wait and watch" approach may be used. The patient should, however, be instructed that the prognosis is bad, and that follow-up appointments are mandatory. The patient should also be told that a prolonged observation period may result in pain and destruction of hard and soft tissue to an extent that would make implant placement and prosthodontic rehabilitation impossible.

CONCLUSIONS

ECR constitutes an important issue in specialist endodontic practice. Thanks to advances in diagnostic and therapeutic methods, such lesions may be classified appropriately, as 3D imaging techniques provide an adequate picture of the extent and severity of the lesion. Along with improvements in biomaterials science, the most predictable treatment option may be chosen, which ensures the patients' safety. However, the etiology of the disease remains a mystery, as the clinical factors that contribute to its development are poorly understood, even though the molecular mechanisms of ECR are believed to be well proven in laboratory tests.

CONFLICT OF INTEREST

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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