External Cervical Resorption – Review of the Literature

Zewnętrzna resorpcja przyszyjkowa – przegląd piśmiennictwa

1 Chair and Department of Periodontology and Oral Medicine, Dental Institute, Medical Faculty, Jagiellonian University Collegium Medicum, Cracow, Poland
2 Department of Integrated Dentistry, Dental Institute, Medical Faculty, Jagiellonian University Collegium Medicum, Cracow, Poland

A – research concept and design, B – collection and/or assembly of data, C – data analysis and interpretation, D – writing the article, E – critical revision of the article, F – final approval of article

Abstract

Cervical resorption (ECR) is an uncommon form of an external root resorption. This problem mainly affects permanent teeth. Resorption lesions in deciduous teeth are rare. Resorption usually affects single teeth, although there also have been observed cases of multiple teeth resorption. Cervical resorption is difficult both to diagnose and to treat. If untreated, it leads to progressive, irreversible loss of hard dental tissue. The change is usually diagnosed by routine radiological examination. Clinical manifestation of changes often occurs only in advanced stages. Histologically, resorption does not differ from any other inflammatory resorptive lesions. Factors predisposing a patient to external cervical resorption: orthodontic treatment, trauma, internal tooth bleaching, surgical procedures, periodontal treatment and bruxism, delayed eruption, intracoronal restorations, developmental defects, systemic diseases. The purpose of the work is to present the phenomenon of external cervical resorption. Knowledge of clinical and radiological symptoms makes it possible to make a diagnosis and select the best possible treatment. The most important factor, from the point of view of the practitioner, seems to be the etiology of changes and prevention (Dent. Med. Probl. 2014, 51, 1, 101–107).

Key words: external cervical resorption, odontoclasts, tooth bleaching.

Streszczenie


Słowa kluczowe: zewnętrzna resorpcja przyszyjkowa, odontoklasty, wybielanie zęba.
Root resorption is a defect that affects hard dental tissue (i.e. cementum and dentin) as a result of odontoclastic processes. It occurs physiologically in deciduous teeth, allowing them to be replaced by permanent teeth. However, the appearance of resorption in permanent teeth is always undesired and can lead to irreversible damage and/or tooth loss [1].

Depending on the surface of the root on which it appears, resorption can be divided into external and internal resorption, and can take several different forms:
1. Surface resorption,
2. Replacement resorption associated with ankylosis,
3. Inflammatory resorption, which itself can be divided into 2 subgroups:
   - external inflammatory root resorption (EIRR),
   - peripheral inflammatory root resorption (PIRR) [2].

The least known and described resorption type is peripheral inflammatory root resorption. The more common name used in the literature is external cervical resorption (ECR). English-language studies also make use of other definitions: invasive cervical resorption, odontoclastoma, peripheral cervical resorption, extra-canal invasive resorption, supraosseous extra-canal invasive resorption, peripheral inflammatory root resorption, subepithelial external root resorption [1]. This is an agressive form of external root resorption that occurs relatively rarely in permanent teeth and leads to progressive loss of hard tissue. It begins on the surface of the root below the epithelial attachment in the connective tissue attachment zone [1, 3, 4]. A change in the position of the epithelial attachment in different clinical situations (the condition of the marginal periodontium, the depth of the periodontal pocket) results in lesions in different locations other than a cervical position [5, 6]. In the majority of cases, the bone in this region remains unchanged unless the problem occurs in conjunction with periodontitis [3, 6]. Resorption usually affects single teeth, although there are also cases of multiple teeth resorption. Cervical resorption is difficult both to diagnose and to treat. If untreated, it leads to progressive, irreversible loss of hard dental tissue [1, 3, 5].

Little is known of the causes of resorption and the mechanism of root hard tissue resistance. One theory ascribes a protective role to root cement and predentin surrounding the dentin. It has been observed that clastic cells do not achieve any adhesion to the cementoid and predentin, which manifest poor mineralisation and the absence of specific extracellular binding proteins (RGD proteins). One also suspects in them the presence of additional factors inhibiting resorption. As long as the most external layer of the cementum is covered with a layer of cementoblasts above the non-mineralised cementoid zone, there is no surface that can ensure sufficient conditions for clastic cell activity [7–10].

Dental pulp, which usually remains unchanged, is excluded from the aetiology of ECR. Two theories have been proposed. The first assumes the occurrence of an inflammatory reaction as a result of the activity of an infectious agent. The second identifies the resorption process with mild fibrovascular or fibro-osseous proliferation, in which microorganisms do not play a pathogenic role. Immunohistological tests appear to confirm the infectious character of the lesions [3, 8, 11, 12].

The anatomic diversity of the cemento-enamel junction predisposes the cervical region to the onset of resorption [1]. The resorption process itself proceeds in parallel with the activation of clastic cells, known as odontoclasts, which morphologically do not differ from, or are completely identical with osteoclasts [3]. The presence of dentin not covered with a layer of enamel or cementum, trauma causing damage to the surface of the cementum and cementoblasts as well as the exposure of specific dentin proteins identified as foreign antigens increase susceptibility to the action or activation of clastic cells [2, 6, 13]. Two factors appear to play a role in the onset of resorption lesions. One triggering factor is the exposure of the root surface through its separation from the protective layer of blast cells. A second common factor supporting and stimulating resorption is infection or mechanical trauma [2]. The state of the pulp has no effect on the aetiology of resorption and does not exhibit the characteristics of pathology [1]. The source of the infection is not the pulp. Hence, it is believed that it is bacteria from the gingival sulcus that stimulate and support any inflammatory response [7]. It is not fully known why resorption begins years after the action of the triggering factor. One suggestion is that a change in the composition of the cementum makes it less resistant to resorption triggered by a minor trauma [14].

Even a minor trauma can lead to the damage of the non-mineralised tissue covering the external root surface. The area affected by such damage may initially be small, but can quickly be covered with resorption cells. This is a passing process and without further stimulation comes to an end by itself after 2–3 weeks through its repair with cement-like tissue. In the case of extensive damage, osseous cells adhere to the surface before cementum-forming cells, which results in ankylosis. Any possible continuation of resorption depends.
on stimulation through pressure or infection. One possible infectious agent that supports resorptive activity are bacteria, which penetrate through the dentinal tubules in the cervical region from the gingival sulcus and from the surface of the tooth, and more rarely from the root canal [12, 15].

**Factors Predisposing a Patient to External Cervical Resorption**

**Orthodontic Treatment**

Numerous theories have emerged to explain the possible influence of orthodontic treatment at the onset of resorption. One of these identifies a relationship between the original damage to the cementum and the removal of necrotic periodontal tissue (the "hyaline layer") at pressure points. According to another theory, the cementum layer is directly damaged by excessive orthodontic forces [16]. This theory focuses on the role played by local hypoxia at pressure points, which increases clastic activity [8]. The stimulated polynucleated cells resorb the exposed dentin. Often resorption lesions occur only several years after the conclusion of the treatment. They are most commonly found in the upper incisors and canines as well as in the lower molars. Heithersay presents results in which orthodontic treatment emerges as the most common and likely cause of the onset of ECR. However, this does not indicate any correlation between the occurrence of resorption lesions and the type of treatment techniques used. Some authors report a higher risk of general resorption when using rectangular wire in the aligning phase, during extractions, in cases of intrusion, flaring, torquing, round-trip movements, and when using intermaxillary elastics [16].

**Trauma**

ECR can be a complication connected with tooth luxation or avulsion. It may also occur after trauma in the cervical region caused by a luxated or intruded deciduous tooth. In such cases, it is important to pay particular attention to this region when repositioning teeth. According to Heithersay’s study, trauma is the second most common factor triggering cervical resorption [1, 3].

**Internal Tooth Bleaching**

The proresorptive action of 30% hydrogen peroxide is explained by its penetration through dentinal tubules down to the root surface. Hydrogen peroxide can also denature dentin and, as a consequence, activate an immune response. The bleaching agent reduces pH to 6.5, which increases osteoclast activity. Other authors suggest that a bleaching agent has indirect proresorptive properties thanks to toxic by-products remaining in the dentin [1, 3, 13]. The majority of studies concern intracoronal bleaching of endodontically treated teeth. Certain individual cases of cervical resorption are a consequence of bleaching vital teeth with carbamide peroxide [17].

**Surgical Procedures**

These are relatively rare causes of ECR. Procedures which may lead to external cervical resorption (usually through damage to the cemento-enamel region) include the following: extraction of a neighbouring impacted third molar or supernumerary tooth and the trauma sustained as a result, the transplantation or surgical exposure of a canine, as well as a radectomy [1, 3].

**Periodontal Treatment**

Heithersay’s study presents periodontal treatment as a rare cause of cervical resorption. The author focuses on 2 healing mechanisms, in which clastic cells have limited access to the root surface. The first is connected with the migration of epithelial cells and the formation of the long junctional epithelium. The second is the migration of progenitor cells from the periodontal membrane. Only a very few cases of cervical resorption caused by periodontal surgery (GBR, coronal flap repositioning) are described in the literature [1, 3, 18–22].

**Others**

Bruxism, delayed eruption, intracoronal restorations, developmental defects, systemic diseases [1].

These include cases identified as such after all other causes have been excluded. According to Heithersay, this group may include undetected defects, such as hypoplasia or hypomineralisation of the cementum [3]. According to a number of studies, patients with hyperoxaluria, hypercalcuria, nephrolithiasis and Rend Osler Weber syndrome are more predisposed to resorption and sometimes also have a genetic predisposition [1, 3, 23]. Cases of idiopathic resorption with an undetermined cause can affect several teeth. Such lesions can affect both dental arches and usually with similar degrees of severity [5].
Clinical Classification

Heithersay [3] prepared a clinical classification of external cervical resorption, which serves not only academic purposes, but also helps systematise the therapeutic procedure (Fig. 1).

Class 1 – minimally invasive resorption lesion in the cervical region with shallow penetration into the dentin.

Class 2 – well-circumscribed, invasive resorption lesion reaching into the pulp chamber area, with no, or only slight penetration of the root dentin.

Class 3 – deeper lesions in dentin affecting not only the coronal dentin, but also the coronal third of the root.

Class 4 – advanced resorption spreading beyond the coronal third of the root.

Diagnosing External Cervical Resorption

Histologically, resorption does not differ from any other inflammatory resorptive lesions. Initially, a small resorption lacuna is observed in the cervical region. Hard tissue defects in the root contain a large amount of granulation. Besides resorption cells, the presence of lymphocytes, plasma cells, histiocytes, macrophages and fibroblasts is also confirmed. In defects increasing in size, calcification loci may appear inside the granulation. Calcified, poorly organised bone-like tissue is indicative of slow progress in lesion formation, during which time repair processes also take place. Even in the case of very extensive lesions layers of predentin and odontoblasts remain undamaged [3, 6, 7, 12].

In clinical terms, cervical resorption is usually asymptomatic and is detected during a routine radiograph. This problem mainly affects permanent teeth. Resorption lesions in deciduous teeth are rare [24]. They usually affect single teeth. Multiple resorption most commonly affects orthodontically treated patients. In every case where resorption lesions are detected, the patient’s dental status should be examined so as to exclude the possible presence of multiple lesions [3].

Resorption in a cervical location makes diagnosis and classification significantly more difficult. Areas that can be examined with a dental probe are characterised by sharp boundaries between the area affected by resorption and the surrounding hard tissue. Simultaneous damage of the mineralised part and of the organic matrix of the dentin leaves the hard floor of the defect which causes a scraping sound when using a dental probe, in contrast to the soft structure of caries [2, 9, 25]. Long-term lesions can cause granulation to be translucent through the enamel, thereby giving the teeth a pink-like colour. The “pink spot” is traditionally regarded as pathognomonic for internal resorption. Hence, cervical resorption is often erroneously diagnosed and treated as internal resorption [7].

In the case of mesially or distally located lesions, moderate translucence is often observed radiologically on the root surface, progressing in an apical and coronal direction inside the dentin, reaching but not perforating the tooth canal. Buccally/palatally located lesions become visible on radiograms, depending on the degree of severity. They can appear in the form of translucence in the region of the periodontal attachment or spread out significantly in a coronal or apical direction. Owing to the absence of pathological changes in the pulp, it is difficult to distinguish radiologically the shape of the chamber from a resorption defect [7]. The clinical and radiological symptoms of external cervical resorption are presented in Table 1. The radiological characteristics are presented in Table 2.

Diagnosis of External Cervical Resorption

A clinical division into 4 classes allows us to make a comparative assessment of the surgical and non-surgical treatment for specific types of resorption. The basic principles of treatment:
– correctly diagnosing resorption process
– reconstructing a damaged root surface
– preventing resorption from progressing
– improving dental aesthetics \[1\].

The treatment of a minor defect (Class 1 or 2) involves flap surgery during which a curettage of the granulation tissue is performed and the root defect is restored using restorative material \[1\].

Treatment of a Class 3 defect should be preceded by a thorough assessment of the prognosis and should be properly planned. Extraction followed by subsequent implant-prosthetic or prosthetic treatment should be considered. The proposed treatment methods are as follows: surgical treatment using barrier membranes, orthodontic extrusion, and extraction with replantation \[3, 7\].

The prognosis for Class 4 resorption defects is unfavourable. The recommended approach is extraction, orthodontic extrusion with extraction and the prosthetic restoration of missing teeth. Leaving this type of resorption untreated results in bone loss, which in turn will affect future implant and prosthetic treatment \[3, 26\].

Some authors recommend the additional use of a 90% trichloroacetic acid solution in water during procedures \[1\]. A small amount of this solution applied to granulation (for 1–2 min) causes coagulation necrosis of resorption tissue \[3\].

The choice of restorative material depends on the conditions of each specific case. Up until now, a composite resin, glass ionomer cement or an amalgam was used to restore a proresorptive defect. The choice of material for supragingival defects is not a difficult one to make, as it must meet, above all else, the criteria of durability, aesthetics and functionality. Restoration in the subgingival region is only possible when using glass ionomer cement or MTA, or MTA alone with the deepest lesions. MTA is the recommended material of choice in cases where the resorption process has not extended beyond the junctional epithelium. The rough surface of the material may, however, promote the formation of a biofilm. When deciding to restore a defect with classic restorative materials it may be necessary to perform an additional ostectomy so as to preserve the biological width \[9, 20, 26–28\].

The prognosis for treatment depends mainly on the scale of the lesion. Classes 1–3 give a positive result, whereas extraction is recommended for Class 4. Heithersay assessed the effects of treatment in 94 patients with 101 teeth affected by ECR-type lesions. Follow-up examinations conducted 3–12 years after the completion of the treatment showed a completely positive treatment outcome in the case of teeth with Classes 1 and 2 lesions. The success rate for Class 3 lesions was just 77.8% and for Class 4 lesions it was 12.5% \[3, 26\].

Properly diagnosing and classifying a lesion is extremely important in the treatment process. The more important characteristics distinguish-
ing cervical resorption from internal resorption are as follows: communication between the resorption cavity and the periodontium, a dentin layer separating the lesion from the pulp chamber. In a conventional radiograph, these details may be difficult to distinguish. Hence, it is advisable to visualise lesions using cone beam computed tomography (CBCT) [8, 28, 29].

**Summary**

External cervical resorption, just like other resorption lesions, is difficult both to diagnose and to treat. During clinical procedures that may cause ECR it is important to observe specific rules:

1. When bleaching endodontically treated teeth:
   - open the root canal maximally to the bone level and before beginning the bleaching process protect the root canal orifice with cement (e.g. GJC),
   - avoid thermocatayllic methods, (avoid etching the dentin,
   - avoid using acrid preparations (such as, e.g. a 30% hydrogen peroxide solution)
   - observe operation procedures, check the expiry date of the agents used.
   - make sure bleaching trays are prepared precisely, ensure that bleaching agent does not penetrate into the gingival sulcus;

2. During orthodontic treatment:
   - design the orthodontic appliance in such a way that only slight forces are used so as not to damage the periodontal membrane;

3. During surgical procedures:
   - avoid procedures that damage the cervical area when bringing impacted teeth into the dental arch (e.g. when using a loop for impacted canines);

4. During periodontal procedures:
   - avoid procedures which deprive the root surface of ligaments and epithelium [7, 17].

**References**


Address for correspondence:
Jolanta Pytko-Połończyk
Department of Integrated Dentistry
Jagiellonian University, Collegium Medicum
Montelupich 4/058
31-155 Cracow
Poland
E-mail: jolanta.pytko-polonczyk@uj.edu.pl

Received: 3.01.2014
Revised: 4.02.2014
Accepted: 5.02.2014

Praca wpłynęła do Redakcji: 3.01.2014 r.
Po recenzji: 4.02.2014 r.
Zaakceptowano do druku: 5.02.2014 r.