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Management of Internal Root Resorption: A Review

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Abstract: ***Internal root resorption (IRR) is a particular category of pulp disease considered as the loss of dentine as a result of the action of clastic cells stimulated by pulpal inflammation. This review article explains the etiology, the prevalence of internal root resorption, and, in addition to the clinical data, the contribution of the 3D imaging (cone beam computed tomography) to the diagnosis, the clinical decision, and the therapeutic management of internal root resorption. The authors reviewed the different therapeutic possibilities including the orthograde or retrograde fillings of the root canal resorption areas. Root canal treatment stays the treatment of choice of internal root resorption as it eliminates the granulation tissue and blood supply of the clastic cells. The authors describe with different clinical cases the modern endodontic techniques including optical aids, ultrasonic enhancement of chemical debridement, and the usage of alternative materials like calcium silicate combined with thermoplastic filling (warm gutta-percha). In these conditions, the prognosis of the conservative management of internal resorptions, even if root walls are perforated, is good. Mineral trioxide aggregate is being progressively used as a root canal filling material, preferably in cases of perforation.***

Keywords: Internal root resorption, , internal inflammatory root resorption, perforation, root canal, molecular pathogenesis, tissue resorption, granular tissue, dentinoclasts, thermoplastic filling with gutta-percha, fiber-glass post, glass ionomer cement, mineral trioxide aggregate, implant

1. Introduction

Resorption is a state related with either physiologic or a pathologic process leading to a loss of dentin, cementum, and/or bone. [(1)] The etiology for resorption originates from various injuries to the tooth, including thermal, mechanical, and chemical. [(2)] There are two different kinds of resorption — external and internal. External resorption is resorption initiated in the periodontium and initially affecting the external surfaces of a tooth — can be characterized as surface, inflammatory, or substitution, or by areas as cervical, lateral, or apical; may or may not invade the dental pulp space. Internal resorption is an inflammation process initiated within the pulp space with loss of dentin and possible invasion of cementum. [(1)]

Physiological resorption

Resorption is an essential part of a congregation of physiological and pathological processes in the human body. Resorption can affect hard tissues such as bone and dental hard tissues [(3)], but it may also affect soft tissue and foreign material like necrotic pulp tissue or materials used in pulp capping or root filling extruded through the apical foramen [(4,5,6)]. A good paradigm of physiological hard tissue resorption is resorption of bone by osteoclastic activity, known as bone turnover. Parathyroid hormone (PTH), secreted by the parathyroid glands, amplifies the amount of calcium in the blood by various methods, one of which involves release of calcium from bones [(7)]. Pathological overproduction of PTH, hyperparathyroidism, will result in imbalance in the physiological bone resorption – apposition cycle, and can cause radiolucent hyperparathyroidism lesions in the jaws [(8,9,10)]. PTH and PTH-related protein (PTHrP) stimulate unprompted osteoclast formation and are needed for tooth eruption [(11,12)]. Resorption of primary teeth is another relatively well characterized example of physiological resorption [(13,14)]. Pressure from the permanent teeth is the influential energy for resorption of the roots of primary teeth. A complex network of events on a cellular level including several activating and inhibiting cytokines and other compounds is needed to lead the resorption to the primary tooth and the bone while protecting the permanent developing tooth from resorption. It is essential to record that there is no infectious (microbiological) component in the various types of physiological resorption.

Pathological resorption

The rare incidence of internal root resorption is possibly the major reason for its etiology being poorly understood. It is generally assumed that damage to the organic sheath, predentin and Odontoblast cells covering mineralized dentine inside the root canal must occur to expose the mineralized tissue to pulpal cells with resorbing potential. However, it is not known with certainty what kind of trauma or other event may be required to produce the damage needed to initiate resorption. The predisposing factors to internal root resorption as suggested in the test include trauma, pulpitis, pulpotomy, cracked tooth, tooth transplantation, restorative procedures, invagination, orthodontic treatment and even a Herpes zoster viral infection [(15,16,17,18)]. In an interesting study of the etiology and pathogenesis of internal inflammatory root resorption, Wedenberg & Lindskog [(19)] stimulated development of internal resorption lesions in monkey teeth, which were then extracted after varying observation periods and examined using a variety of microscopic and radiographic methods. The root canals of monkey incisors with vital pulps were accessed and injected with Freund's complete adjuvant (a non-specific stimulator of the immune response) and either sealed aseptically or left open to the oral cavity. The authors reported that colonization of the dentin wall by macrophage-like cells was observed in both experimental groups. Interestingly, the colonization was transient in the sealed teeth with no pulpal infection, whereas the exposed teeth whose pulps turn out to be infected by the oral bacteria exhibited a more extensive and prolonged colonization of the dentin surface by the resorbing cells. Spreading of the macrophages/multinuclear giant cells could be seen only in areas where dentin was denuded. According to the study, this occurred either by mineralization of predentin in the affected location or by degeneration of the odontoblasts and predentin layer. In the sealed teeth without pulpal infection,

the number of macrophage-like cells declined 6–10 weeks after the initiation of the experiment and an increase in the number of fibroblast-like cells as well as hard tissue barrier formation were observed. However, in the open teeth with infected coronal pulps, no reduction in the number of cells with resorbing potential could be detected. Brown and Brenn staining of the histological sections showed that most of the dentinal tubules in the area were invaded by bacteria of different morphotypes [19]. The authors concluded that for internal root resorption to occur, mineralized dentin must be exposed. Moreover, they suggested that internal root resorption can be divided into two different types: transient resorption and progressive internal root resorption. The first one would thus be analogous with transient external resorption of the root and is self-limiting while the latter is induced by the presence of bacteria and will continue to expand. Sahara et al. [20] studied the resorption by odontoclasts of a superficial non-mineralized layer of predentin prior to the shedding of human deciduous teeth by light and electron microscopy. They found multinucleated cells on the predentin surface of the coronal dentine between the degenerated odontoblasts and resorption lacunae in the non-mineralized predentin. In some other areas in the same study, the multinuclear cells 'simultaneously resorbed both non-mineralized and calcospherite-mineralized matrix in the predentin.' The authors suggested that multinucleate odontoclasts can resorb the non-mineralized predentin matrix in vivo, probably in the same way that they resorb the demineralized organic matrix in the resorption zone underlying their ruffled border [20]. In another study of shedding primary teeth, it was found that the pulpal tissue did not undergo any major structural changes, as long as the roots were actively resorbed by odontoclasts,. When root resorption was nearly finished, increased numbers of inflammatory cells were detected in the pulp [21]. At this stage, odontoblasts began to degenerate, multinucleate odontoclasts appeared on the dentin surface, and resorption proceeded from the predentin to the dentin. The odontoclastic activity was first demonstrated at the cervical area of the crown pulp, but eventually the resorption of dentin spread coronally toward the pulp horns. The uncommon occurrence of dentin resorption can be explained by the dominance of osteoclast/odontoclast inhibitory substances such as OPG over activators such as RANKL. However, it has also been suggested that dentin contains a non-collagenous compound/ component which may function as a resorption inhibitor in dentin. Wedenberg & Lindskog [22] studied the ability of induced and non-induced peritoneal macrophages to spread in vitro on different inorganic and organic components of dental tissues in order to establish morphologic evidence of a presence of a resorption inhibitor in dentin. Macrophages attached and spread on enamel, dentin, and collagen coated coverslips; however, the same cells attached but did not spread when incubated on predentin or demineralized dentin. The authors concluded that the resistance to resorption of predentin and dentin may be caused by an organic, non-collagenous component of the tissue. Subsequent experiments indicated that when demineralized dentin and predentin were treated with guanidium hydrochloride, macrophage spreading could readily be detected [23]. In another series of experiments, osteoclasts were segregated from neonatal rats and seeded onto pieces of completely mineralized dentin, demineralized dentin, and predentin with or without prior extraction with guanidium hydrochloride. Osteoclasts (odontoclasts) colonized and resorbed fully mineralized dentin, whereas clastic cells were not observed on unextracted demineralized dentin and predentin. However, after guanidium hydrochloride extraction, osteoclasts also attached and spread on demineralized dentin and predentin [24]. Damage to the cells of the odontoblastic layer may occur not just as a consequence of trauma but also because of inflammation as a reaction of the pulp connective tissue to infection approaching either through dentin (caries) or from the more coronal pulp. Odontoblast cell death is frequently seen in areas of micro abscesses in the peripheral pulp tissue in teeth with a deep caries lesion. The fact that internal inflammatory root resorptions are also found in the middle and apical parts of the roots of mandibular premolars and molars, which are well protected against trauma, may be regarded as an indication that pulpal inflammation can initiate internal inflammatory. Internal inflammatory root resorption with an asymmetric shape in the lower second mandibular premolar [25]. This could occur if Odontoblast are killed at the advancing front of inflammation but the pulp still maintains vitality in the location. Cells with resorbing capability may become activated and gain contact with exposed predentin/dentin. As mentioned earlier in this article, several bacterial species have been shown to increase RANKL expression and osteoclast activation [26,27,28]. It may be of interest that internal inflammatory root resorption in its most conventional form spreads proportionally in all directions into the dentin surrounding the pulp. The initiation of internal root resorption throughout the full circle of dentin at certain depths in the coronal-apical direction can perhaps be explained either by a slowly advancing pulpitis (inflammation) or by mechanical trauma. However, the fact remains that internal inflammatory root resorption is uncommon and the amount of facts on it is inadequate. Thus, the key events in the initiation of internal inflammatory root resorption remain largely unknown. Irrespective of the possible initiating factor (trauma, inflammation or some other reason), there is a general agreement that the progress of internal root resorption is dependent on two things: the pulp tissue at the resorption area must be vital, and the pulp coronal to the resorption must be partially or completely necrotic, allowing bacterial infection and microbial antigens to enter the root canal. Microbial stimulus is necessary for the continuation of internal inflammatory root resorption. Otherwise, limited internal surface resorption might be the only consequence of the initial phase. The microbiology of internal inflammatory root resorption has not been studied. However, as several recent studies have shown that both Gram-negative and Gram-positive bacteria as well as spirochetes have the ability to induce RANKL expression and osteoclast activation, it can be speculated that there may not be specific species which are more likely than others to be involved in the initiation and pathogenesis of internal inflammatory root resorption.

Etiology

Etiology of internal root resorption is quite uncertain. Numerous etiologic factors have been suggested for the loss of predentin, and trauma seems to be the most advocated. Persistent infection of the pulp by bacteria causes the colonization of the walls of the pulp chamber by

macrophage-like cells. The attachment and proliferation of such cells is the primary prerequisite for initiation of root resorption [29]. It can be concluded that trauma and pulpal inflammation or infection are the main causative factors in the induction of internal resorption, although the complete etiologic factors as well as the pathogenesis have not yet been completely elucidated [30]. Resorption processes can develop by shifting of pH-value to acid for example in irreversible pulpitis, so that the dentin and enamel substances are dissolved by chelation [31]. The untreated internal resorption can progress into external or vice versa which causes a fracture of the tooth [32,33]. In the cases of tooth trauma, the interpulpal hemorrhage can develop. Developed blood clots are then organised and substituted by granular tissue which compresses dentin wall of the pulpal chamber or root canal [34]. They differentiate into dentinoclasts which is the cells responsible for resorption of the hard tooth structure, with activation of non-differentiated mesenchymal cells of the pulpal tissue [35,36]. The chronic trauma or inflammation can be the reason of transformation of non-differentiated cells of connective pulpal tissue into giant multinuclear cells which are responsible for the resorption process [31]. These cells bond with diluted dentin ingredients and develop into single visible defects like Howsip's lacunae which include groups of dentinoclasts. With differentiation of dentinoblasts, the biochemical processes are concomitantly triggered when the hard angle structures are starting to dissolve due to shifting of pH-value to acid. Together with the formation of lacunae, the granular tissue which is beginning to swell can with its pressure on dentin substance also provoke and sustain the resorption process. Granulation tissue is a very good vascular tissue containing leukocytes, macrophages, monocytes, lymphocytes, and the fact which prompts a possible consideration that in the pulpal tissue the specific immunologic reaction can be included.

Symptomatology

Internal resorption is the progression in the pulpal area which can be present with no clinical symptoms, or symptoms that can be similar to those of asymptomatic chronic pulpitis with occasional acute exacerbations. The clinical picture is recognized by the pink color of enamel due to irradiation of the granular tissue through the remaining still thin wall of the enamel [37,38].

Prevalence

Internal root resorption is believed uncommon, but the occurrence of internal resorption is not well known. Depending on the accuracy of the means evaluating the pathology, results may strongly differ. Histological studies showed a higher rate of occurrence of internal root resorption than by a simple examination of the X-rays [2]. It is also unknown whether there is any geographical, age- or sex-related differences in the occurrence of internal root resorption. Typically, only one tooth per patient is affected by internal root resorption. However, occasionally two adjacent teeth have internal root resorption, with trauma being the likely initiating factor in such cases [39].

2. Clinical Radiographic Diagnosis

The clinical characteristics of internal root resorption are dependent on the development and location of the resorption. Most teeth with internal root resorption are asymptomatic. However, when the resorption is actively progressing, the tooth is at least partially vital and may present symptoms typical of pulpitis. If the resorption occurs in or near the crown, it may in severe cases appear as a pinkish or reddish color through the crown if only a thin layer of enamel is left to cover the resorption. The red color is caused by the highly vascularized connective tissue adjacent to the resorbing cells. In internal resorption, the color is typically located centrally, whereas in cervical resorption the color ('pink spot') may also be mesially or distally located. Teeth with untreated internal resorptions in the coronal area often turn gray/dark gray if the pulp becomes necrotic. Internal inflammatory root resorptions continue to expand until either endodontic treatment is started or the pulp becomes necrotic. Eventually the resorption will perforate the root unless it is stopped by one of the above-mentioned events. Perforation of the root is usually followed by the development of a sinus tract, which confirms the presence of an infection in the root canal. After the perforation, the continuation of the resorption may no longer be dependent on the presence of vital pulp tissue because the resorbing cells may now obtain nutrients from tissues surrounding the tooth. After perforation, the control of infection is more difficult than in an unperforated root canal. The tooth structure is also weaker in teeth with perforation as a consequence of loss of hard tissue. In addition to the sinus tract, swelling might present, while most patients complain of only mild or no pain. With advancing infection, the entire pulp becomes necrotic and internal root resorption ceases because the resorbing cells are cut off from the circulation and nutrients, unless root perforation has occurred before the development of total necrosis. Pulpal necrosis can therefore be regarded as an effective protection against spreading of the resorption. The consequence of pulp necrosis is, as usual, apical periodontitis. There is no data indicating that the (previous) resorption has any effect on the pathogenesis or symptoms of apical periodontitis. In its most classical appearance, internal inflammatory root resorption is comparatively easy to recognize radiographically and the right diagnosis can be made. The resorption is seen as a radiolucent, round and symmetrical widening of the root canal space. At the area of the resorption, the original canal shape can no longer be observed. However, not all internal root resorptions show similar progression, and oval as well as asymmetrically shaped internal root resorptions can be found. In the coronal pulp/ crown area, internal resorption can be symmetrical in teeth with one root canal and a narrow pulp chamber where pulp horns are situated close to each other. However, in molar teeth with several roots and a wide pulp chamber, internal resorption may begin at one part of the chamber and spread locally into the surrounding dentin. In such cases, it may be difficult to make the diagnosis between internal and external cervical resorption until the resorptive area is accessed directly, cleaned and carefully studied under a surgical microscope during endodontic treatment. However, cervical resorptions in the crown area often have a more irregular outline and contain randomly shaped thin opaque

lines which are not seen in lesions of internal resorption. However the early diagnosis of the internal root resorption is difficult by examination of a conventional X-ray. If IRR is inferred, numerous shots under different angles of incidence are recommended. But an accurate diagnosis is essential for an appropriate treatment plan to be devised. CBCT has been effectively used to assess the true nature and severity of resorption lesions in isolated case reports indicating that the clinician could more confidently diagnose and treat the defect [40]. The use of CBCT provides a 3-dimensional appreciation of the resorption lesion with axial, coronal, parasagittal views of the anatomy. In the serial of cross-sectional views, the size and the areas of the resorption are evidently determined with high sensitivity and an excellent specificity. CBCT has a high accuracy in detecting root lesions at the earliest stages [41]. Sometimes, the resorption area is filled with a deposition of metaplastic hard tissue that looks like bone or cementum. This replacement resorption material has an aspect of enlargement of the pulp chamber with a fuzzy emergence of the canal space. CBCT gives evidence about the following: (i) location, size, and shape of the lesion, (ii) root wall thickness, (iii) presence of root perforations, (iv) presence of an apical bone lesion, (v) localization of anatomical structures: maxillary sinus, mental foramen, and inferior alveolar nerve. All these criteria verify the differential diagnosis with external root resorption and let the prognosis assessment of the tooth, if the lesion is amendable to treatment.

3. Therapeutic Decision

The decision-making must take in to concern several criteria: (i) patient's age, (ii) tooth location, (iii) shape of the clinical crown, (iv) occlusion, (v) resorption location, (vi) presence or not of root perforations and their wideness, (vii) resorption wideness, (viii) resistance/weakness of the remaining root hard tissue, (ix) periodontal status, (x) ability to realize a restorative treatment on the concerned tooth [2]. There are three basic diagnostic methods of internal resorption: (i) visual examination based on the changed color in tooth crown, (ii) Rtg diagnosis, (iii) light microscopy, (iii) electron microscopy. Light microscopy shows different levels of inflammation of the pulpal tissue with infiltration of predominant lymphocytes, macrophages and some leukocytes, dilated blood vessels and multinuclear dentinoclasts in resorptive lacunae on the pulpal-dentin surface. Electron microscopy shows the pulpal-dentin wall without odontoblasts. Dentinoclasts, large in number, have size of 50µm and with numerous phyllopod are turned toward dentin surface and attached to it. Resorption process of the endodontic space can be divided in the internal resorption, external resorption and periapical resorption. The internal resorption is furthermore divided into intracoronal and intracanal, and the intracanal resorption can be found in the coronal, middle and apical third of the root canal [42]. Internal inflammatory root resorption is usually first detected radiographically. Many lesions are found accidentally during routine check-up radiographs, as teeth with internal resorption are typically asymptomatic. As indicated earlier, diagnosis of symmetrical, round or oval lesions in the root canal can easily be done. For more irregularly shaped resorptions, the key diagnostic feature is the disappearance of the original canal shape in the area of the resorption. The diagnostic challenge with internal root resorption is external cervical resorption when it projects over the root canal on a radiograph. Cervical root resorption is known for its inability to penetrate into the root canal with vital pulp tissue. Micro computed tomography (CT) scans of teeth with cervical resorption show a zone of 0.1–0.3mm dentin which separates the (external) cervical resorption from the pulp; this zone being ca. 10–20 times wider than the thickness of the predentin layer [43,44,45]. The thin layer of dentin thus includes enough mineralized tissue to make it visible on the radiographs as an opaque line next to the root canal. This opaque line is a reliable differential diagnostic sign of cervical root resorption. If the cervical resorption projects on top of the root canal, the original shape of the canal can in many cases be seen through the resorption. Internal and cervical resorption can also both occur in the crown area. Although the point of invasion on the root surface of cervical resorption can sometimes be difficult or impossible to detect on the radiograph, the presence or absence of the opaque lines surrounding the pulp is still a useful indicator to differentiate between the two types of resorption. Recent evolution of radiographic techniques has begun to have an impact on the diagnosis of tooth resorptions, including internal root resorption. Assessment of the resorptive lesions by three-dimensional imaging using various modifications of the CT techniques will greatly facilitate differential diagnosis and help to determine the location, dimensions, spreading, and possible site(s) of perforation in much greater detail than has been previously possible [46,47,48,49,50]. Reduced radiation dosage, improved resolution, and better tolerance of disturbances caused by metal structures such as posts and metal filling materials already help to make better recommendations and decisions of optimal treatment choices and to minimize the loss of dental hard tissue. Cervical or root surface caries seldom create a diagnostic problem even in cases where the radiolucent carious lesion projects on top of the root canal. Color changes that are clinically noticeable exists only in a minority of cases of internal and cervical resorptions. The color change related to internal resorption can be pink, red, dark red, gray or even dark gray depending on the size of the resorption and the vitality status of the pulp. As soon as the coronal pulp becomes necrotic, it is likely that the initial pink or reddish color will steadily change to a dark red or dark gray. Cervical resorption is independent of the pulp. Therefore, it is less likely that a pink spot which is detected in the crown area in some cases of cervical resorption will turn dark as in internal resorption. Another characteristic that may be an indication of the origin of the resorption is the location of the spot: a color change from internal inflammatory resorption is typically seen in the middle of the tooth in the mesio-distal direction (except in multi-rooted teeth), whereas a color change from the cervical resorption can be located mesially, centrally, or distally. Clinical examination by probing may be useful in the differential diagnosis between root surface caries and cervical resorption: the former is practically always accessible by probing while cervical resorption is usually not because it starts apical to the junctional epithelium. However, in cases of wide-spreading cervical resorption (types 3 and 4), one can sometimes probe into the resorptive lesion when the patient is anesthetized. Even in these situations, the caries lesion usually feels softer (sticky) while the resorption feels

more like normal dentin. Only in extremely rare cases can internal inflammatory root resorption be clinically probed. A prerequisite for this is that the resorption has perforated the root or the crown at the level of or coronally to the marginal bone. In such situations, the probe will easily penetrate deep into the resorption because of the typical shape and type of spreading of internal resorptions, whereas in cervical resorption the depth of probe penetration is quite limited[39]. From the data collected by clinical examination and CBCT, numerous alternatives may be considered: (1) therapeutic abstention and monitoring, in absence of infectious signs and symptoms, (2) orthograde root canal treatment, with three choices based on the absence or presence of perforation of the radicular wall: complete root canal filling with gutta percha on non-perforated lesions; combined gutta percha in the root canal and MTA fillings for the perforated location; complete filling with a bioactive material (MTA or Biodentine) on apical perforated lesions located in a short root length, (3) retrograde apical treatment, (4) extraction and replacement by implants: the non-conservative treatment is indicated if the tooth is too weakened to be treated or restored[2].

4. Management of Internal Root Resorption

Instrumentation of teeth with internal resorption

Instrumentation and cleaning of the root canal space of teeth with internal resorption faces a few challenges different from those of normal endodontic treatment. In cases where the resorption is active, there is typically brisk bleeding from the pulp tissue, which may make it difficult to locate the root canal openings. However, as soon as the apical pulp tissue has been cut off and removed using large enough instruments in the apical canal, the bleeding stops or is greatly reduced, allowing better visibility into the work area. Irrigation by concentrated sodium hypochlorite will in most cases help to reduce the bleeding. Sometimes it is preferable to pack calcium hydroxide into the pulp chamber and the canal(s) and seal the tooth with a temporary filling. A few days later, bleeding of the soft tissue is no longer a problem because calcium hydroxide effectively necrotizes the granulation tissue. For teeth where the resorption has perforated the root, placement of calcium hydroxide is recommended to necrotize the resorptive tissue and to stop the bleeding. Although the majority of the literature on internal inflammatory root resorption is case reports, there is no generally accepted protocol for the chemo-mechanical instrumentation of the root canal system in these cases. However, it is obvious that a great importance should be placed on the chemical dissolution of the vital and necrotic pulp tissue. Therefore, irrigation with sodium hypochlorite is an important part of the treatment of teeth with internal resorption. Small perforations do not seem to require abandonment of the use of hypochlorite; on the contrary, hypochlorite will help to control bleeding from the perforation and disinfect and clean the area as experienced with accidental perforation complications. However, with large perforations, low-concentration hypochlorite solutions should be used and other irrigants such as chlorhexidine should be considered. The shape of a resorbed root canal prevents instrument access to all areas of the canal. Creating a straight line access to the resorption cannot be done in many cases because it would weaken the tooth structure too much. This is one reason why the use of ultrasound has been advocated for the treatment of internal resorptions [51]. A great emphasis must be placed on the chemical dissolution of the vital and necrotic pulp tissue with sodium hypochlorite. The use of ultrasonic devices activates and aids the diffusion of the irrigation solution of hypochlorite to all the areas of the root canal system [39]. The non-traumatic plastic tips of Endo Activator are particularly indicated to achieve a complete chemo-mechanical debridement of the root canal. In order to better reach the most distant areas of the resorption, hand instruments are often bent at 1–4mm from the tip to help to gain contact with the walls of the resorption cavity and help to remove all soft tissue. Although use of hypochlorite and ultrasound are mainly responsible for cleaning of the most challenging areas, the importance of careful mechanical cleaning should not be underestimated [39].

Conservative Dental Treatments of Resorbed Teeth

About the root canal filling, the material must to be flowable to seal the resorptive defect. Thermoplastic gutta percha techniques seem to give the best results when the canal walls are respected. When the root wall has been perforated, MTA is the best material to seal the perforation as it is biocompatible, bioactive, and well tolerated by periradicular tissues [52]. The working time can be modified by the adjunction of water if the material begins to harden during its use.

Complete Root Canal Filling with Warm Gutta Percha

This choice is for internal root resorption without perforation of the canal walls which is the most favorable situation in long-term prognosis. The treatment is performed in two sessions. First session is as following: (i) anesthesia, rubber dam, and access cavity, (ii) determination of the root canal length with manual instruments, (iii) shaping of the canal, (iv) disinfection of the canal and resorption lacuna with sodium hypochlorite, (v) activation of the solution with ultrasonic tips, (vi) drying of the canal with sterile paper tips, (vii) filling the canal and lacuna with calcium hydroxide as an interappointment dressing for thorough disinfection of the canal space, (viii) temporary sealing of the access cavity with glass ionomer cement (GIC). The following session is as following: (i) anesthesia, rubber dam, and reopening of the access cavity, (ii) removal of the canal calcium hydroxide by a large irrigation of ClONa activated with sonic tips, (iii) assessment of the root length—fitting of the gutta percha master cone, (iv) radiographic control to assess the good fit of the master gutta percha cone, (v) final irrigation, (vi) drying of the root canal with sterile paper tips, (vii) obturation of the apical third of the root with warm gutta percha, (viii) gutta percha thermo-compaction in the resorption lacunae to completely fill the wide canal space [53,54], (ix) radiographic control, (x) waterproof closing of the access cavity with a GIC.

Sealing of Internal Root Resorption with Bioactive Cements as MTA

This choice is indicated in presence of a perforation of the canal walls providing a communication between the root canal system and the periapical tissue. In this clinical situation, the smaller the perforation size, the more anticipatable the prognosis of the tooth. The treatment is

performed in two sessions. First session is as following: (i) anesthesia, rubber dam placement, and access cavity, (ii) vigorous bleeding which confirms the activity of the resorptive lesion, (iii) intracanal dressing with calcium hydroxide in order to dissolve necrotic soft tissue and to control the bleeding, (iv) sealing of the access cavity with a glass ionomer cement (GIC). Second session is as following: (i) anesthesia, rubber dam placement, and access cavity, (ii) chemical debridement with sodium hypochlorite solution in the canal and the resorption lacuna, (iii) activation of the irrigant with ultrasonic tips, (iv) evaluation of the canal length evaluated from CBCT slides and radiographic control with a gutta percha master cone, (v) canal drying with upside down sterile paper tips, (vi) obturation of the open apex and resorption lacuna with MTA under visual check with an operative microscope, (vii) radiographic control of the obturation, (viii) placement of a water-moistened cotton pellet directly over the material, (ix) provisional sealing of the access cavity with a glass ionomer cement. Considering the area of the resorption and the short length of the root, the canal can be fully filled with MTA. In other case, the healthy part of the canal will be filled with gutta percha [(2)]. A number of recent studies, including two meta-analyses, have shown that MTA is superior to formocresol in pulpotomies of primary molars [(55,56,57)]. A notable difference between the two materials was the absence of resorption complications following treatment in the MTA groups. The excellent performance of MTA as a retrograde filling material is well recognized and filling the complete root canal of immature permanent incisors with MTA has been reported [(58)]. Recently, filling of the internal resorption cavity with MTA in a primary molar was reported [(59)]. Although not supported yet by long-term results from clinical studies, it is possible that the treatment of perforated internal resorptions in the future will consist of a thorough chemo-mechanical cleaning and disinfection of the root canal and resorption area including the perforation site, followed by a short-term calcium hydroxide management. At the second appointment, without any clinical symptoms, the resorption cavity will be filled with MTA. MTA is also very well tolerated by the tissues. MTA carriers, ultrasound, inverted paper points used as pluggers, and radiographic control of the MTA filling at the early phase of condensation are all crucial factors for success and to ensure a high quality filling. In teeth with a large resorption cavity in the coronal third of the root canal, use of composite materials must be taken into count in order to strengthen the tooth and to increase its resistance to tooth fracture [(60)].

Surgical Treatment of Internal Root Resorption

Surgical method is essential when it is impossible to get access to the lesion through the canal. Surgical treatment should always be performed in a second intention, after orthograde treatment has been performed, the coronal part of the canal being filled. In these cases, because of the shape of the lesion, surgical approach allows to get direct access to the lesion and to do a mechanical cleaning of the resorbed defect. The conventional guidelines of the endodontic surgery procedure must be respected [(61)]. Following local anesthesia a mucoperiosteal flap is raised. The cortical bone plate is removed to provide access to the root area. The soft tissue lesion is curetted and the intra radicular dentin cavity is prepared with the aid of an operative microscope, cleaned, and dried. The filling materials (like mineral trioxide aggregate (MTA) or Biodentine) are placed and smoothed on its external surface. The surgical procedure is finished with thorough cleaning of the wound area. The flap is relocated and sutured.

Prognosis of the Treatment of Internal Inflammatory Resorption

In light of the rare occurrence of internal inflammatory root resorption, it is not surprising that there are very few studies on the outcome and prognosis of the treatment of the resorption. Although the deficiency of follow-up studies on the long term prognosis of the treatment of teeth with internal root resorption is obvious, there is a general consensus based on clinical experience and case reports that the prognosis of the treatment is fairly good or even excellent for cases which have not perforated and where the tooth has not been weakened too much by the loss of tooth structure [(62)].

5. Conclusion

Internal inflammatory resorption is an uncommon resorption of the tooth which starts from the root canal and destroys the surrounding tooth structure. Odontoclast cells which are responsible for the resorption are structurally and functionally similar to bone osteoclasts [(39)]. Internal inflammatory root resorption is a particular category of pulp disease, which can be diagnosed by clinical and radiographic examination of teeth in daily practice [(2)]. The fact that patients mostly do not mention in their anamnesis neither clinical symptoms not signs of periodontal disease, there is a small number of the cases of internal resorption in the clinical practice to suggest the inclusion of immunologic specific reactions as the etiopathogenetic factors in the internal resorption. However these considerations require additional immunologic research. It could be concluded that internal resorption is a rare form of resorption of the hard dental tissue which etiology is most frequently unknown, or it is trauma, inflammation - either unspecified or in the combination with specific immunologic reaction. The reciprocal activity between the newly formed granular tissue and dentinoclasts induces and proceeds the resorption process inside the endodontic space which could be compared to pathogenetic changes in the periapical region. The initial diagnosis and management is very important in order to arrest the resorption process. The success or failure of therapy should be followed clinically and by Rtg control. Naturally, if the resorption is arrested actually is not progressing, we trust that our treatment is successful, we saved a tooth and the objective of our therapy has been accomplished [(42)]. Today, the diagnosis of internal root resorption is considerably enhanced by the three-dimensional imaging. Furthermore, the CBCT's superior diagnosis accuracy resulted in an improved management of the resorptive defects and a effective results of conservative management of teeth with internal resorption. Modern endodontic techniques including optical aids, ultrasonic improvement of chemical debridement, and thermoplastic filling methods should be applied during the root canal treatment of internally resorbed teeth. Alternative materials such as calcium silicate cements offer new prospects for the recovery of resorbed teeth. In these conditions, the prognosis

of the treatment of internal resorptions, even if root walls are perforated, is good [(2)].

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