Internal Dental Root Resorption: A Case Report and Literature Review

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Abstract
Root resorption is the loss of hard dental structures under the action of elastic cells, which can also be accompanied by bone tissue loss. Root resorption can be external or internal. In both situations, root resorption requires the presence of blood supply allowing migration of elastic cells. In internal root resorption, pulp vitality is required at least in the apical third of the root canal.

Case presentation: A 25-year-old female patient was referred to dentist for a vestibular sinus tract distal to upper lateral incisor. A root canal filling was performed 18 months ago for dental pulp necrosis. The periapical radiography of upper lateral incisor revealed internal root resorption and lateral granuloma. The histopathological exam showed highly vascularized internal and external granuloma. But how can the development and progression of internal root resorption in a non-vital pulp in which endodontic filling was performed be explained? Where were elastic cells recruited from? Diagnostic hypotheses and possible mechanisms involved are discussed by reviewing the literature. The interdisciplinary approach takes into account local and systemic etiopathogenetic factors. Conclusions: Internal root resorption remains an incompletely understood process and its onset and atypical evolution require differential diagnosis with many diseases including general disorders.

Keywords: Internal root resorption, Osteoclasts, Odontoclasts, Plasma cells, Accessory root canal granulomas

Introduction
Bone tissue is subjected during the course of life to continuous physiological remodeling, characterized by bone resorption (under the action of osteoclasts) and bone apposition (through production of osteoid matrix by osteoblasts)[1]. This continuous bone remodeling is also present in alveolar bone, whose functions are maintained by the presence of teeth in the alveoli and the phospho-calcium metabolism balance under hormonal control.

The same cannot be said about teeth. External root resorption can be a physiological event for the primary teeth during the eruption of permanent teeth. However, this process can be pathological when it is extensive and develops much earlier than the physiological rate of permanent teeth eruption.

Root resorption in permanent teeth is pathological whether it is external or internal. Root resorption is induced by the elastic action of cells present in teeth and adjacent tissues (odontoclasts, dentinoclasts, cementoclasts and osteoclasts), being characterized by loss of dentin, cementum and/or bone[2].

External root resorption (periodontally derived) develops on the external surface of the tooth root, is accompanied by concomitant alveolar bone resorption, and its causes can be periapical inflammatory processes, excessive pressure forces exerted on the tooth root during orthodontic treatment or the rapid development of a tumor.
process\cite{3}. In contrast, internal root resorption (pulpally derived) starts in the root canal and extends to the internal dentin wall, being caused by pulp inflammation secondary to caries, trauma, thermal or chemical injuries. In both situations, blood supply ensuring the recruitment and viability of clastic cells involved in resorption is required. This means that in internal root resorption, viable dental pulp, even if infected, situated at least in the apical third of the root canal is needed. Once the pulp necrosis has developed in the entire root canal (which means cessation of blood supply and of the possibility of clastic cell recruitment), internal root resorption does no longer progress\cite{3}.

But how can the development and progression of internal root resorption in a non-vital pulp in which endodontic filling was performed, be explained? Where were clastic cells recruited from? What differential diagnoses should be considered in such situations? These are some questions that will be addressed in the following subsections.

**CASE PRESENTATION**

A 25-year-old female patient was referred to our department for a vestibular sinus tract distal to upper lateral incisor in whom a root canal filling was performed 18 months ago for dental pulp necrosis. The periapical radiography of upper lateral incisor revealed an inadequate root canal filling with internal root resorption around the gutta-percha in the upper half of the root canal and lateral granuloma. The diagnostic challenge was related to how an internal granuloma can develop in a non-vital root canal. The patient did not agree with the proposed endodontic retreatment and an apical surgery with retrograde filling was performed – see Figure 1. The histopathological exam revealed highly vascularized internal and external granuloma with many plasma cells – see Figures 2 and 3. Evolution was favorable over the 18 months follow-up period.

**Figure 1:** Clinical and paraclinical diagnosis, treatment and follow-up stages in atypical internal root resorption – illustration by the case of a 25-year-old female patient; it was obtained informed consent of the patient.

1. Presence of a vestibular sinus tract distal to 1.2; 2. Excision of the sinus tract, detachment of a mucoperiosteal flap with exposure of the vestibular bone plate at the level of root 1.2 – the fenestration located distally to the root of 1.2 is seen; 3. Surgical exposure of ½ the root of 1.2 – the gutta-percha cone can be observed through the thinned dentin walls due to internal root resorption; 4. Retrograde filling cement after apical resection below the internal granuloma, and removal of pathological processes (the patient did not agree with the proposed endodontic retreatment); 5. Anatomopathological appearance of the excised specimen – view from the palatal side (5a), with visualization of internal root resorption, the gutta-percha cone during the pathological process, which exceeds the apical constriction by 1.5 mm, and the external granuloma; image 5b shows the pedicle connecting the two pathological processes; 6. Histopathological aspects of the highly vascularized internal granuloma (6a) and of the external granuloma with many plasma cells (6b); 7, 8 and 9. Clinical and imaging follow-up stages at 3, 6 and 18 months; in images 7b and 8b, the Hammer “residual shadow” is still present, which is no longer seen in image 9b (adequate periapical ossification).

**Figure 2:** Light microscopy images of the internal granuloma.

1. Large zone of resorption with a tendency to extend deeply into the dentin; odontoblast degeneration and loss of predentin; irregular dentin surface due to numerous resorptive bays; the resorbed dentin was replaced by chronic inflammatory infiltrate and dilated blood vessels. 2. The gutta-percha cone in the center of the root canal surrounded by chronic inflammatory infiltrate mainly consisting of plasma cells, lymphocytes and numerous dilated capillaries. 3. On the dentin surface, in the resorptive lacunae, numerous odontoclast precursors were recruited and formed a continuous barrier between the dentin and the chronic inflammatory infiltrate extending towards the center of the root canal. 4. Hemorrhage and fragments of necrotic dentin; precursors of odontoclasts were attracted to the site of resorption, stimulated by necrotic dentin. 5. Superficial dentin necrosis and large fragments of necrotic dentin surrounded by a dense chronic inflammatory infiltrate. 6. Site of chronic inflammation analogous to the external granuloma, in the proximity of the apical foramen; polymorphonuclear neutrophils, macrophages, plasma cells and dilated blood vessels. Goldner’s trichrome stain.
Figure 3: Photomicrograph of the external granuloma.
1. Chronic inflammatory infiltrate consisting predominantly of lymphocytes and plasma cells.
2. Dilated, congestive capillaries surrounded by a heterogeneous inflammatory infiltrate containing polymorphonuclear neutrophils, macrophages, plasma cells and lymphocytes.
3. Areas of fibrous connective tissue with active fibroblasts and well-defined bundles of collagen fibers alternating with chronic inflammatory areas.
4. Zone of necrosis containing degenerated extracellular matrix associated with numerous active macrophages.
5. Surface epithelium invaginated into the granuloma, showing various degrees of cell degeneration; inflammatory infiltrate with lymphocytes and extravagated erythrocytes in the adjacent connective tissue.

Discussion

Pulp necrosis is most frequently a complication of untreated or incorrectly treated dental caries. Cariogenic bacteria cause pulp inflammatory and infectious processes (pulpitis) which, untreated or in case of intense injury, induce pulp necrosis. In this situation the tooth loses its vitality, the entire pulp is disintegrated, cellular tissue structures and odontoblasts are no longer seen, blood vessels are destructured, and inflammatory cells are in the process of disintegration[4]. The classic treatment of pulp necrosis involves isolation of the tooth with a rubber dam, mechanical-antiseptic treatment, drug treatment and subsequently, long-lasting endodontic sealing. But what are the pathogenic mechanisms involved in the development of an atypical internal root resorption? The possible pathogenic mechanisms are shown in Figure 4.

Figure 4: Pathogenic mechanisms involved in atypical internal root resorption:
1. Lateral accessory root canal;
2. Lateral incisor with two root canals;
3. Periapical granuloma extending to the middle third of the tooth;
4. Solitary plasmacytoma of the maxilla; On the intraoral radiograph, in tooth 1.2, an inadequate root canal filling is seen (inhomogeneous, exceeding the apex by 1.5 mm), with internal root resorption (interrupted white arrow) and lateral granuloma (white arrow).

Lateral accessory root canal

The most plausible pathway for the development of internal root resorption, in a tooth with pulp necrosis treated by endodontic filling (lateral cold condensation of gutta-percha), during the course of 18 months after treatment, is the presence of a lateral accessory canal[5]. Lateral canals may develop in tooth roots when during odontogenesis there is a localized fragmentation of the epithelial root sheath, or when blood vessels (originating from the follicular sac and supplying the dental papilla) persist after the end of odontogenesis[6-7]. They are most frequently missed by imaging examination because of their small size (the lateral canal foramen is 2-3 times smaller than the apical foramen), and are mainly discovered when they cause pathological processes[8].

The possibility of the presence of viable pulp (even if infected) in this lateral accessory canal is almost null. In this lateral accessory canal, infectious pathogens probably survived, contributing to the formation of the external granuloma and releasing chemotactic factors for the cells involved in internal root resorption[9,10].

The presence of the pedicle connecting the root to the external granuloma can explain the lack of external root resorption, as well as the connection with the lateral accessory canal. In order for internal root resorption to occur, elastic cells had to be recruited from the alveolar process (osteoclasts from the osteolysis area or from hematogenous bone marrow), and then, through a newly formed blood vessel, they had to penetrate through the lateral accessory canal into the root canal. The inhomogeneous endodontic filling might have favored this process. This blood vessel might have formed as a neovessel detached from the interalveolar network under the action of angiogenic factors.
Lateral incisor with two root canals

In the majority of the cases, according to the literature, the upper lateral incisor has one root and one root canal\(^{11}\). However, the literature also reports cases with upper lateral incisors with: two roots and two root canals; one root and two root canals (the second canal being most frequently situated towards the palatal side), or even 3 root canals; one root and 4 root canals or dens invaginatus of various types\(^{12-17}\). These variations seem to be race-dependent (more frequent in the Turkish population)\(^{18}\).

The presence of a second narrower root canal was excluded by surgery, which evidenced only one central root canal on the root resection line.

Periapical granuloma extending to the middle third of the tooth

Another hypothesis of the mechanism of internal root resorption in this case is the development of a periapical granuloma due to canal and apical delta infection (a consequence of pulp necrosis in 1.2 at presentation) or/and the displacement of pathogen-infected debris beyond the apical constriction, during endodontic treatment. This hypothesis was refuted by the radiological aspect and intraoperative examination (no periapical granulation tissue was found).

Solitary plasmacytoma of the maxilla

Plasma cell neoplasms can take several clinical forms: multiple myeloma, solitary plasmacytoma and extramedullary plasmacytoma.

Solitary plasmacytoma of the maxilla is a rare disease, with a frequently favorable evolution after excision, but cases with aggressive local evolution, post-excision recurrence or evolution towards multiple myeloma have also been reported\(^{19}\). The dominant symptom is pain, and if the tumor process invades adjacent tissues, tooth mobility can be observed\(^{20,21}\). Imaging usually evidences a radiolucent area with well-delimited margins, most frequently unilocular, located in a single bone (osteolysis being induced by clonal plasma cells)\(^{22}\).

The multitude of plasma cells in both the external and internal granuloma, some plasma cells being bi- or trimuculate, it was necessary to clarify whether the radiolucent mass situated distally to 1.2 could be a solitary plasmacytoma of the maxilla. The literature reports no cases of dental onset of a solitary plasmacytoma, only peri-dental invasion\(^{23,24}\). In this respect, the patient was examined by a hematologist, without detection of positive clinical signs for solitary plasmacytoma or multiple myeloma. Full blood count, erythrocyte sedimentation rate, total protein, immunoglobulins A, G and M (Ig-A, Ig-G and Ig-M), and blood smear were within normal limits.

Conclusion

Internal root resorption is still a challenge for doctors from several points of view: in early stages it can remain undiscovered, its onset and atypical evolution require differential diagnosis with many diseases including general disorders, therapeutic approach involves balancing conservative treatment (when tooth resistance is not severely affected) against radical treatment – tooth extraction.

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Consent: Written consent has been obtained from the patient for publication of this case report and any accompanying images.

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