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External multiple invasive cervical resorption with subsequent arrest of the resorption

CASE REPORT

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¹Director posgraduate clinical endodontics, University of Uberaba, Uberaba, Minas Gerais, Brazil ²Molecular Biology Laboratory, University of Uberaba, Uberaba, Minas Gerais, Brazil **Abstract** – A rare, the uncommon, case of multiple invasive cervical resorption (MICR) with subsequent arrest of the resorption involving eight teeth of the mandible (tooth 46 to tooth 32) is presented. This case is noteworthy because of the characteristics of its clinical evolution. There have only been a few cases in the literature were found presenting involvement of such a large number of teeth. Furthermore, noreports on cases of MICR with arrest of the resorption, as described in this report, were found.

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Case report

A 23 year-old dark-skinned female patient sought the dentistry services of the University of Uberaba (Universidade de Uberaba) complaining of multiple fractures in the crowns of her lower anterior teeth. During radiological evaluation, images suggestive of invasive cervical root resorption were observed for all affected teeth (Figs 1 and 2), without evidence of a decay process. When the patient's history was taken, she did not report any dental trauma in this region. Clinical and laboratory tests were requested, but these did not indicate any systemic disturbance. No such cases were reported in the family history. The clinical examination detected pulp vitality in the teeth that still had a clinical crown. Probing and periodontal examination for microbial activity did not reveal any significant disorder. The most important clinical evidence for the situation faced was the type of occlusion: the patient presented a deep bite, thus suggesting excessive loading of the lower anterior battery. The development of this process had led to fracturing and burial of the roots of four teeth: 44, 43, 42 and 41 (Fig. 3).

Additionally, teeth 46, 45, 32 and 31 were affected and in a progression phase, such that teeth 45 and 31 were indicated for extraction because of the advanced stage of the lesion. On the other hand, teeth 46 and 32 were in the initial stages of the process. During the extraction of teeth 45 and 31, material for histopathological examination was collected (Fig. 4).

It was decided to carry out curettage and restoration of the initial lesion on tooth 46, but only curettage and smoothing of the cavity of the mesial surface of tooth 31. When removing the resorption lesion by means of curettage, tissue that did not present the clinical macroscopic characteristics of invasive cervical resorption was found. This resorption generally presented a unique superimposed appearance of invasive tissue surrounding the cervical body of the root, thus making its removal difficult. Nevertheless, during surgical removal, a smooth and regular resorptive cavity was found, and the lodged tissue could be removed very easily.

The sample removed for anatomical/pathological examination was composed of the crown from tooth 45, along with the surrounding periodontal and gingival tissues (Fig. 4). The evaluation was performed on serial thin sections cut from the resorption region, which presented the characteristic histological appearance of scar tissue, as if the resorption had been arrested, with a layer of fibrous tissue lining the resorption cavity. This was demonstrated by thin sections stained using the haematoxylin–eosin method (Fig. 5) and Masson's trichrome method (Fig. 6). Furthermore, the evaluation showed absence of clastic cells and Howship's lacunae on the tooth surface (Fig. 7). Immunohistochemical analysis was negative for clastic cells (CD68).

During the five-year clinical and radiological follow up performed subsequent to the intervention, the patient did not present any further progression in the teeth that had been treated, or any new areas of resorption (Fig. 8, 9), despite experiencing tooth movement resulting from orthodontic treatment.

Discussion

External resorption may occur at any point on the external surface of the root of completely formed teeth (1). This resorption results from the activities of multinuclear cells in the periodontal ligament, because of instability and imbalance caused by aggression or



Fig. 1. Radiographic aspects suggesting invasive cervical root resorption, observed in all affected teeth (46, 43).



Fig. 2. Radiographic aspects suggesting invasive cervical root resorption, observed in all affected teeth (42, 32).

the absence or removal of periodontal or cementum tissue (2).

The present report describes the clinical appearance of external root resorption with the characteristics of invasive cervical resorption because of the location and clinical and radiological features, in accordance with the Frank & Bakland classification (3). These authors created the term 'extra-canal invasive resorption' (EIR) to describe this phenomenon. Moreover, the classification of EIR was subdivided, depending on the location of the resorption process: infraosseous or supraosseous (4–6). Supraosseous resorption occurs most frequently at the cervical level and is named external cervical resorption. The preference for this resorption location is because of its greater fragility due to failure of the protection from the cementum tissue (7). The pulp vitality observed in the present report is in line with the



Fig. 3. Clinic aspect of affected teeth in the mandibular (46, 32).



Fig. 4. Sample removed for the pathological examination was composed of the crown from tooth 45, along with the surrounding periodontal and gingival tissue.

findings by Yaacob (8), who described the protective dentine around the pulp chamber as the agent responsible for maintaining the vitality.

The exact aetiology of cervical resorption is still unknown. Gold & Hasselgren (9) suggested that there are three environmental factors that may contribute towards this destructive process: absence of protection for the root surface, presence of vascular conjunctive tissue and an inflammatory stimulus.

The clinical evidence points towards occlusion trauma as the most likely cause for the onset and development of resorption in the clinical case described here. Nevertheless, several causes have been deemed responsible for causing such resorption: orthodontic movement (2, 10– 12), tooth whitening (13), neoplasia (10), periodontal diseases, poor occlusion, pressure exerted by supernumerary teeth and systemic conditions (11).

Goldman (14) classified resorption as inactive, active and very active, depending on the quantities of inflammatory cells, osteoclasts and defective bone tissue, by making comparisons with the stromal collagen and calcification of the adjacent tissues.

In the present clinical case, the resorption was classified by means of histochemical analysis as inactive,



Fig. 5. Characteristic histological aspect of scar tissue, with a layer of fibrous tissue lining the resorption cavity of tooth 45 (Haematoxylin and eosin, magnification $\times 200$).



Fig. 6. Fibrous tissues lining the resorption cavity of tooth 45 (arrow) (Masson's trichrome methods, magnification ×800).



Fig. 7. Detail of Fig. 5. Absence of clastic cells and Howship's lacunae on the dental surface (Haematoxylin and eosin, magnification $\times 1600$).



Fig. 8. Radiographic aspects 5 years later (46).



Fig. 9. Radiographic aspects did not any further progression resorption 5 years later (32).

i.e. in a phase of arrested resorption. This was characterized by the presence of scar tissue in this phase of arrested resorption, absence of inflammatory cells and negative CD68 immunostaining. It is important to note that the classification of the resorption as inactive was established without any curative intervention.

This development suggests that the patient may have undergone two distinct phases during the resorption process. Initially, there may have been a period close to the time of peak growth, when the pace of the onset and development of resorption was influenced by modulating factors from the bone metabolism, such as growth or sex hormones acting as activation factors for resorption in the periodontal region, in accordance with the study by Mascarenhas (15). Subsequently, during a period when these factors attained a balance, there was a decrease in the pace of onset of resorption. It is not common to observe a self-limiting arrest of the resorption mode and, in the radiological follow up over the subsequent 5 years, no further progress or appearance of new resorption points was detected. In this present study, the patient presented involvement of eight teeth, thus characterizing multiple cervical external resorption, which was diagnosed at the age of 23. Clinical reports of cervical resorption involving more than one tooth are, in most cases, in patients of more than 20 years of age, of both genders (16). Consequently, multiple resorption cases diagnosed at this stage, at a young adult age, may have begun and accelerated at a younger age. Such cases, therefore, deserving more accurate investigation, aimed at obtaining more efficient preventive diagnosis.

References

- 1. Henry JL, Weinmann JP. The pattern of resorption and repair of human cementum. J Am Dent Assoc 1951;42:270–90.
- Yusof WZ, Ghazali MN. Multiple external root resorption. J Am Dent Assoc 1989;118:453–5.
- Frank AL, Bakland LK. Nonendodontic therapy for supraosseous extracanal invasive resorption. J Endod 1987;13:348–55.
- 4. Frank AL. Invasive resorption: an update. Compend Colin Educ Dent 1995;16:250–62.

- 5. Hovland EJ, Bakland LK. Endodontics. Dent Clin North Am 1992; 36:501–4.
- Rubeinstein LK, Byrne BE. Supraosseous extracanal invasive resorption. Gen Dent 1993;41:430–3.
- Choquet J. Note sur les repports anatomiques existant chez l'homme entre l'émail et le cément. L'Odontologie 1989;8:115– 25.
- 8. Yaacob HB. The resistant dentine shell of teeth suffering from idiopathic external resorption. Aust Den J 1980;25:73–5.
- 9. Gold SI, Hasselgren G. Peripheral inflammatory root resorption. J Clin Periodontol 1992;19:523–34.
- Hammarström L, Lindskog S. General morphological aspects of resorption of teeth and alveolar bone. Int Endod J 1985;18:93–108.
- Pierce AM. Cellular mechanisms in tooth and bone resorption. Morphological studies in rats and monkeys. PhD thesis, Karolinska Institutet, Stockholm, 1989.
- 12. Carter LC. Resorption of tooth substance: diagnosis and management. Compendium 1992; 13: 1008-10, 1012-16.
- Consolaro A. Reabsorções dentárias nas especialidades clínicas. ed. Maringá: Dental Press; 2002. p. 87–102.
- Goldman H. Spontaneous intermittent resorption of teeth. J Am Dent Assoc 1954;49:522–32.
- Mascarenhas P, Gaspski R, Al-Shammari K, Wang HL. Influence of sex hormones on the periodontium. J Clin Periodontol 2003;30:671.
- 16. George DI, Miller RL. Idiopathic resorption of teeth a case report of three cases. Am J Orthod 1986;89:13–20.

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