

Teeth with pulp vitality and apical periodontitis: periapical cemento-osseous dysplasia

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ABSTRACT

In spite of the dogmatic assertion according to which 'if a tooth has periapical lesion, there is no pulp vitality anymore', it does not correspond to reality, as there are periapical lesions in which the carrier teeth remain with pulp vitality. To provide a basis for this thought, the present paper discusses: 1) The concept of true periapical lesion, 2) The differences between apex and periapex, 3) The distinction between apical pericementum and apical periodontium,

and 4) Considerations about the true periapical lesions classification. Among the four clinical situations in which a tooth with periapical lesion keeps its pulp vitality, we selected, for this study, which is the first one of a series, the Periapical Cemento-Osseous Dysplasia, which does not require any treatment, but only controlling over time.

Keywords: Periapical cemento-osseous dysplasia. Periapical cemental dysplasia. Cementomas. Periapical lesions.

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Periapical lesions still represent the majority of jaw osteolytic lesions after the chronical periodontal inflammatory diseases. Its diagnosis is of great importance for clinical practice, in all specialties, and it involves from its right nomenclature to the establishment of a prognosis which should be as accurate as possible.

Clinical practice is occasionally permeated by dogmatic concepts, among them some affirmations such as 'if a tooth has periapical lesion, there is no pulp vitality anymore'. This does not correspond to reality, for there are periapical lesions in which the teeth show vitality. For instance, we have four clinical situations:

1. Acute or chronical apical periodontitis induced by physical factors, such as dental trauma characterized by concussion and occlusal trauma. These are inflammatory periapical lesions in which the tooth keeps its pulp vitality!

2. Deciduous teeth with root formation at its final stage and with extensive carious lesion and pulp exposure: in the tooth apexes, it is possible to detect a diffuse osteolytic area that corresponds to a periapical chronical inflammatory process. However, the pulp, even when it is inflamed in its coronary portion, shows vitality also in the radicular region. In many of these cases, when conservative treatment is applied by means of pulpotomy, the process goes backwards and the apex returns to its normal state.

3. Permanent teeth with Periapical Cemento-osseous Dysplasia.

4. Permanent or deciduous teeth with Benign Cementoblastoma.

Among these four situations of teeth with periapical lesions and pulp vitality, two of them are non-inflammatory lesions that only take place in the periapical region. For that reason, they should be considered true and primary periapical lesions. One of them, the Periapical Cemento-osseous Dysplasia – a pseudotumoral or, more accurately, pseudo-neoplastic disease – shows a self-limited growth and has no invasive connotation. The second, which is the Benign Cementoblastoma, represents a true neoplasia for it presents continuous growth and, in case it is not surgically approached in an adequate way, it may relapse.

The term 'lesion'

'Lesion' is the way to nominate any permanent or transitory anatomic alteration, regardless of its inflammatory, neoplastic or adaptive nature. The term 'lesion' has a wide coverage.

Lesions in any part of the body can be induced by physical, chemical and biological agents, and they may characterize certain situations or diseases. Some diseases cause certain typical lesions that allow their clinical, imaging and microscopic diagnosis.

In the periapical region, the 'diseases' or 'clinical-pathologic entities' that affect the mentioned region cause lesions also known as 'periapical lesions'.

The concept of true periapical lesion

Periapical lesions are those with etiopathogenic, clinical, imaging and microscopic characteristics which are specific and exclusive to the periapex.

Other diseases may occur in the tooth periapex. Nonetheless, they are not characterized by specificity or exclusivity of the region. Some examples are actinomycosis, tuberculosis, odontogenic tumors (such as the ameloblastoma), odontogenic and non-odontogenic cysts, such as the odontogenic keratocyst (odontogenic keratocystic tumor) and the nasopalatine cyst.

The concept of apex differs from that of periapex

The periapex, or periapical region, corresponds to the tissues that surround, directly or indirectly, the tooth apex, but it does not involve the apical mineralized dental tissues (Figures 1, 2 and 3). The terms 'apical' and 'periapical' are not synonyms. For example, Hypercementosis represents an apical alteration, whereas the Dentoalveolar Abscess is a periapical alteration.

In the periapex there are the periodontal ligament, the bundle bone and the dental alveolus bone, with their medullary spaces filled by medullar tissue. In the apex of the teeth, there are the cementum, the dentine, the pulp and the periodontal stump in the cemental canal, in continuity with the ligament.

Apical pericementum and apical periodontium

The term 'pericementum' refers to the structures that are directly or indirectly related to the tooth

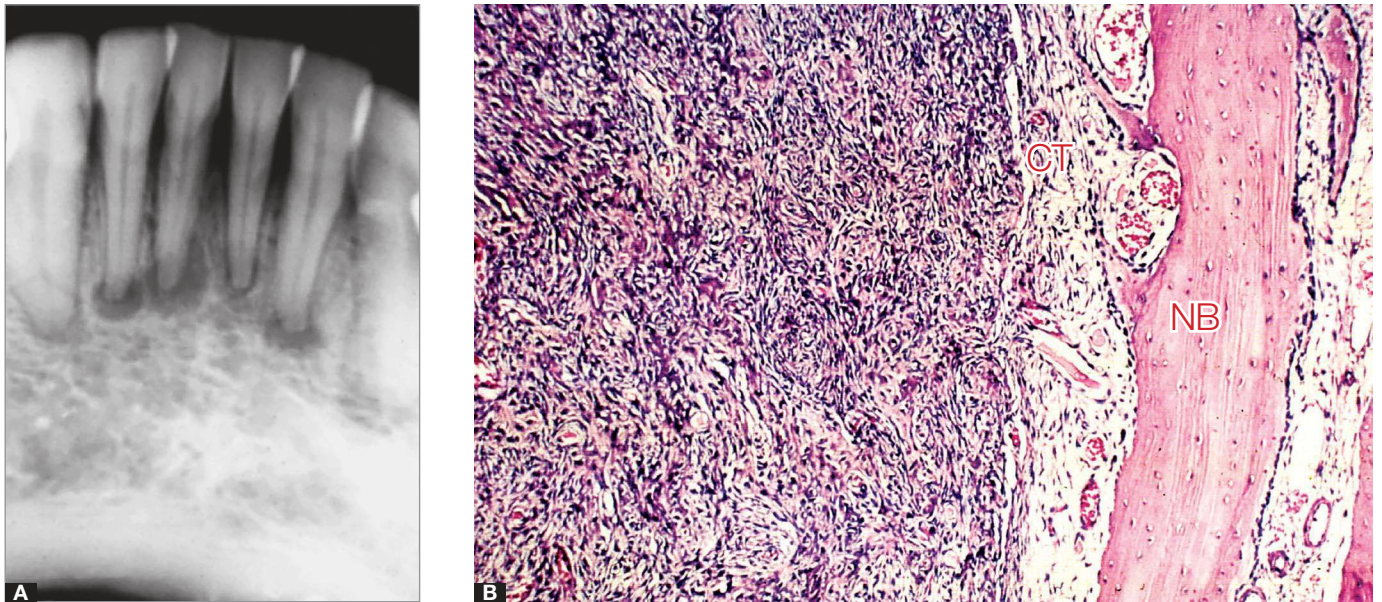


Figure 1. Periapical cemento-osseous dysplasia, at its initial stage. The teeth have pulp vitality and the periapical lesions are comprised of polyhedral fusiform cells tissues, outlined by discrete and poorly organized capsular connective tissue (CT). The adjacent bone is normal (NB) (HE, 10x).

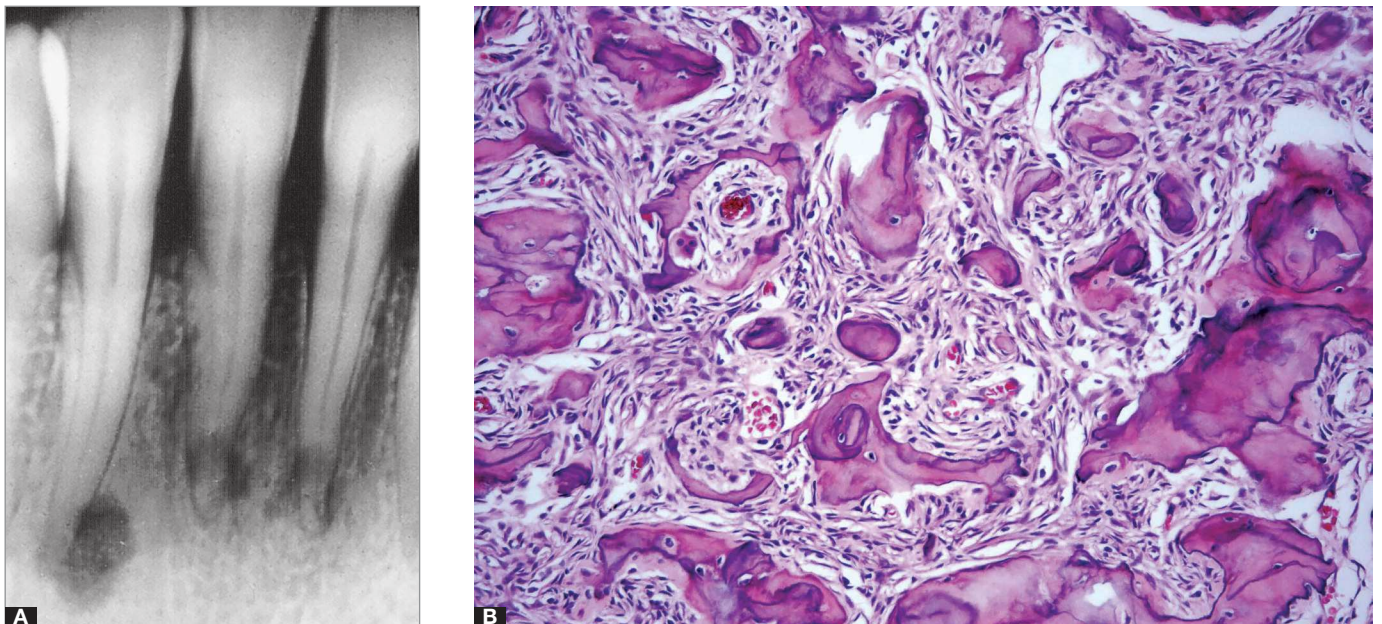


Figure 2. Periapical cemento-osseous dysplasia, at its intermediate stage. The teeth have pulp vitality and the periapical lesions consist of polyhedral fusiform cells tissues, which deposit poorly organized and randomly spread islets and 'trabeculae'. The pattern is irregular, with basophilic apposition lines, and it reminds, in a rough way, of cementum (HE, 25x).

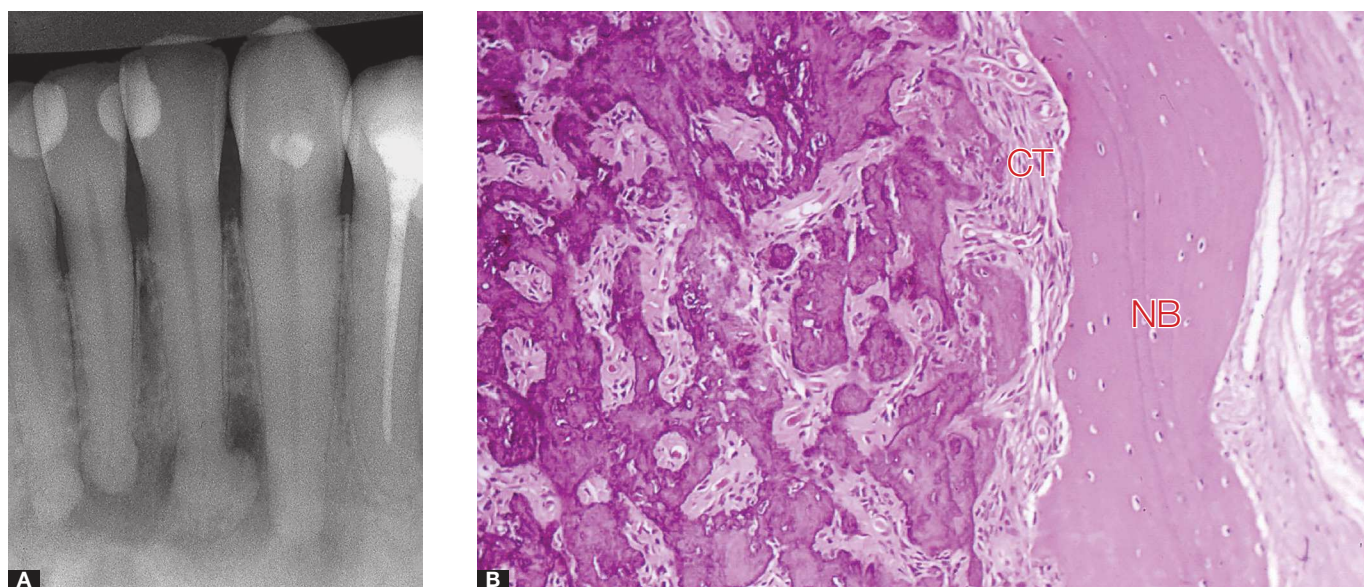


Figure 3. Periapical Cemento-Osseous Dysplasia, at its radiopaque final stage. The teeth have pulp vitality and the periapical lesions are predominantly formed by mineralized and irregularly deposited tissue of basophilic cementoid pattern, being fused to islets and 'trabeculae' and permeated by polyhedral fusiform cells. Discreetly, there is in the peripheral area, a discreet and poorly organized capsular connective tissue (CT). The adjacent bone has normal appearance (NB) (HE, 25x) (A: by Prof. Dr. Bitondi, Ribeirão Preto, SP).

apex, without involving it in the process, such as the terms 'periapex' and 'periapical region'. The term 'apical pericementitis' refers to an inflammation that may involve all the tissues in the apical region, including the mature bone tissue which is part of the alveolar process and the medullar tissues among its trabeculae.

For its turn, the term 'apical periodontium' encompasses the periodontal tissues of the apical portion of the tooth, and involves the cementum, the periodontal ligament and the bundle bone. The apical periodontium does not encompass the dentine and the alveolar tissue that is beyond the narrow layer of bundle bone which covers the periodontal surface of the alveolus.

As for the term 'apical periodontitis', it refers, exclusively, to an inflammation of the apical periodontal tissues: cementum, periodontal ligament and bundle bone. The bundle bone is also known as 'alveolar bone tissue strictly speaking'.

The difference between 'apical pericementum' and 'apical periodontium' is subtle and based on discussions about the use of the term 'apical pericementitis' or 'apical periodontitis'. Incredible as it may seem, the term 'apical pericementitis' is anatomically wider than the term 'apical periodontitis', which is limited to the apical periodontium (Fig 3).

In summary, the periapical region is comprised of three basic components:

1. The tooth, especially for the predominantly cellular cementum which is thicker than in the cervical and medium portions of the root, in which the periodontal fibers are inserted, as well as for delimitating the area of periodontal stump.

2. The periodontal ligament, formed by a thin strap of specialized and thoroughly organized fibrous connective tissue, which is 200 to 400 micrometers thick. Of its volume, 50% are represented by blood vessels, mainly venules. For that reason, it can be easily induced to an inflammatory process.

The portion that corresponds to the periodontal stump continues naturally with the pulp tissue on one side and, on the other, with the periodontal connective tissue. The periodontal ligament, for its turn, continues naturally with the medullar spaces of the alveolar bone. This way, in spite of the hard and inelastic walls, there are structures of communication with the soft tissues that surround the bone marrow.

3. The alveolar bone is comprised of its bundle cortical portion on the periodontal face, as if it were a kind of alveolar plaster, and the underlying common secondary bone. The other mineralized structures of the trabeculae outline the medullar spaces, resulting in drawing which, although apparently irregular, intercommunicate with the soft tissues of the region (Fig 3).

Classification and nomenclature of the periapical lesions

Among the bone alterations of the jaw, the periapical lesions are probably the most frequent group and most of them are related to pulpal alterations. Depending on the nature of the process in course, the true periapical lesions can be classified as follows:

1. Inflammatory;
2. Neoplastic;
3. Pseudotumoral.

The periapical lesions of inflammatory nature prevail and their causal agents are extremely variable, considering the multiple clinical situations to which they are associated. Among the clinical situations that induce the inflammatory periapical lesions, the following must be highlighted:

1. Generalized Purulent Acute Pulpitis and its extension to the apical periodontium.
2. Progressive Chronical Pulpitis with gradual necrosis eventually reaching total necrosis, including the periodontal stump in the cemental canal.
3. Aseptic Pulpal Necrosis from dental trauma.
4. Pulp Calcific Metamorphosis followed by necrosis, over years, induced by dental trauma.
5. Traumatic Occlusion inducing Chronical or Acute Apical Periodontitis, without pulpal necrosis, which may be part of the lesions called Occlusal Trauma.
6. Chemical and/or physical lesion of the periapical tissues during the operative procedures of biopulpectomy.

The pseudotumoral and neoplastic periapical lesions are represented by only one lesion or disease each, with exclusive location limited to the tooth periapex. They are, respectively, benign cementoblastoma and periapical cemental dysplasia or periapical cemento-osseous dysplasia. There are two 'apical lesions' which may occur as a consequence of true periapical lesions: Hypercementosis and External Inflammatory Resorption.

CLASSIFICATION OF THE PRIMARY TRUE PERIAPICAL LESIONS

1. Inflammatory periapical lesions

1.1 – Apical Periodontitis (Pericementitis)

- 1.1.1 – Acute Apical Periodontitis (Pericementitis)
- 1.1.2 – Chronical Apical Periodontitis (Pericementitis)

1.2 – Periapical or Dentoalveolar Abscess

1.2.1 – Acute Dentoalveolar Abscess and its stages

1.2.2 – Chronical Dentoalveolar Abscess

1.3 – Periapical Granuloma

1.3.1 – Immunogenic

1.3.2 – Foreign Body type

1.4 – Periapical or Apical Periodontal Cyst

2. Tumoral Periapical Lesion

2.1 – Benign Cementoblastoma.

3. Pseudotumoral Periapical Lesion

3.1 – Periapical Cemento-Osseous Dysplasia.

In this paper, we will be addressing Periapical Cemento-Osseous Dysplasia based on clinical cases diagnosed by means of periapical radiography. In the periapical radiography, the diagnosis can be safe. However, in many cases it was not properly done. It is not rare to observe, in consultancies and evaluations, cases of Periapical Cemento-Osseous Dysplasia treated by an endodontic approach when, actually, it was never necessary.

Periapical Cemento-osseous Dysplasia

Its meaning and how it begins

Periapical Cemento-Osseous Dysplasia represents a disorder in the resorption and formation of the periapical tissues^{2,8,9,10} of the apical periodontal ligament, the cementum and the bundle bone. Its location is almost exclusively the lower incisors periapex, with the possibility of reaching canines and premolars (Fig 1 to 6).

Gradually, the periodontal ligament and the periapical bone are replaced by connective tissue richly cellularized. That results in radiolucent images that imitate immunogenic periapical granulomas, but the teeth show pulpal vitality and their radicular and coronary structure is in good conditions (Figs 1, 4 and 6).

The richly cellularized connective tissue is where the deposition of the cemento-osseous matrix starts in the form of islets and irregular trabeculae (Fig 2), whose mineralization and expansion fuse irregularly with the structure of the tooth apex, with an irregular external contour (Figs 3 and 5), making it totally different from hypercementosis, focal sclerosing osteitis and idiopathic periapical osteosclerosis.

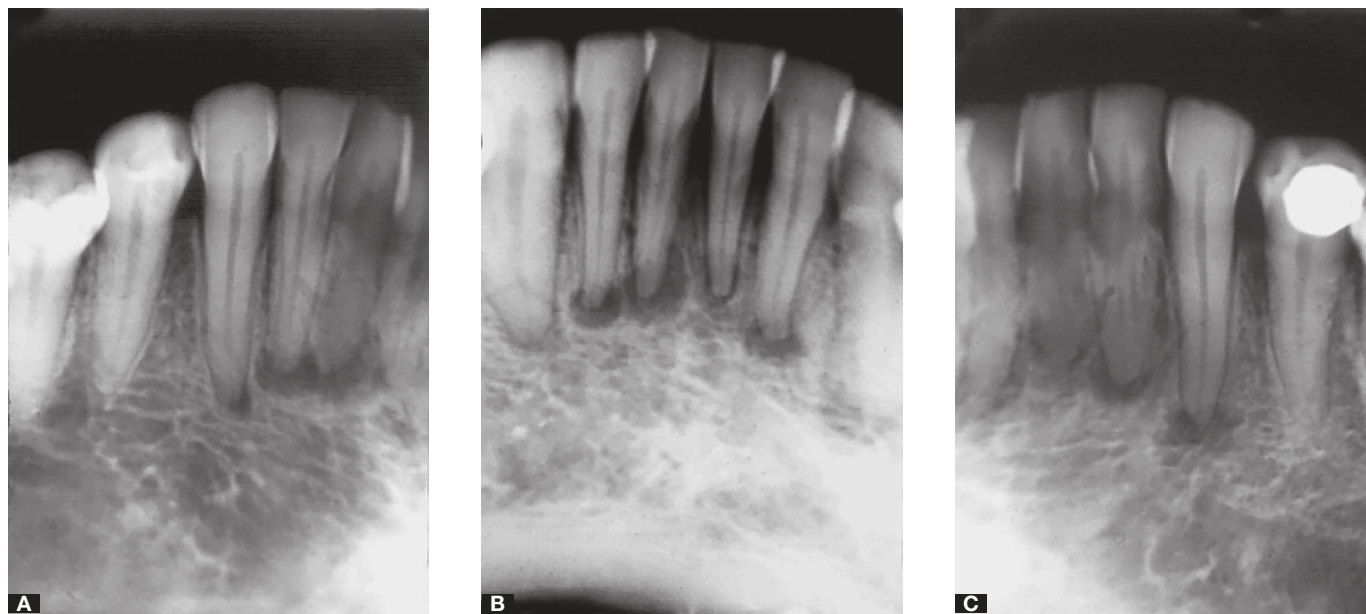


Figure 4. Periapical cemento-osseous dysplasia at its initial stage. The teeth show pulp vitality and the adjacent bone remains normal.

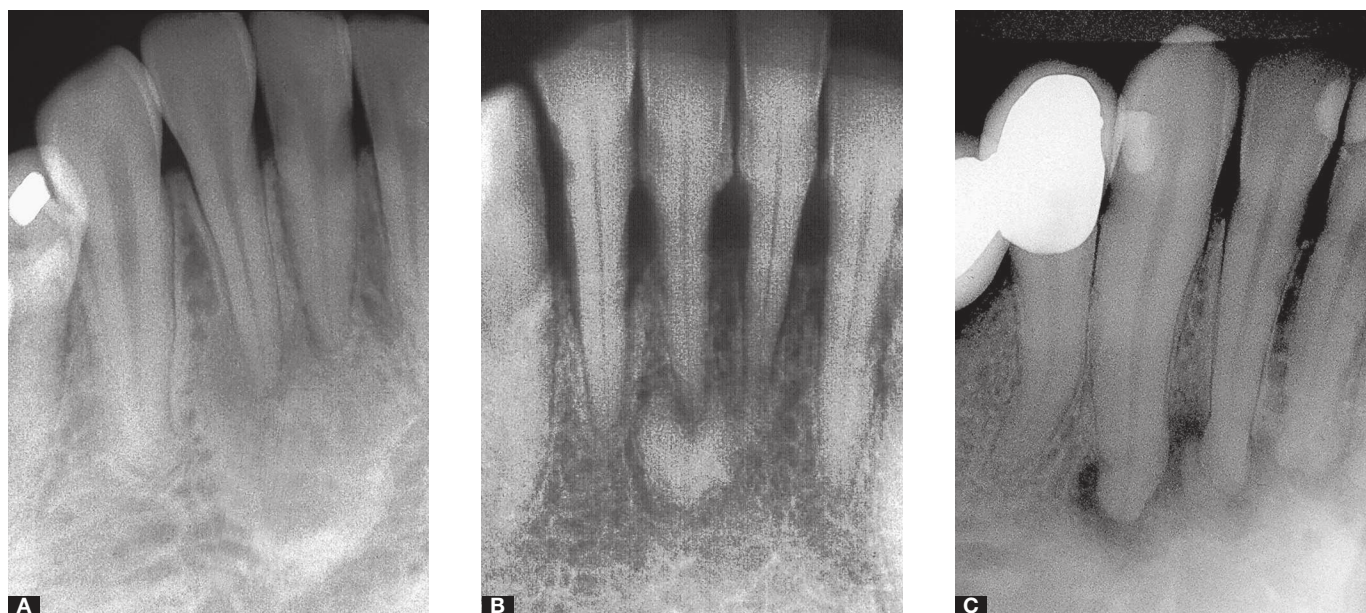


Figure 5. Clinical cases of periapical cemento-osseous dysplasia, which may impose difficulty to the imaging diagnosis. However, its association with clinical characteristics makes it possible to define the diagnosis, due to the fact that the teeth show pulp vitality (C: by Prof. Dr. Bitondi, Ribeirão Preto, SP).

Curiously, this lesion was described for the first time in a horse, by Forget,³ in 1860. In humans, it was firstly described by Brophy,¹ in his surgery book, in 1915, as periapical cemental dysplasia.

Clinical aspects: the teeth have pulp vitality!

The affected teeth present, invariably, no painful symptoms and the disease is discovered by chance by means of routine radiography exams (Fig 6).

Its great prevalence is among black women aged between 30 and 40, and may reach 5% in some population groups.⁷ The most affected teeth are lower incisors and canines with pulp vitality (Fig 6).^{4,7} As for Eastern people, the higher prevalence is in the lower canines and premolars.¹¹

Despite its unknown etiopathogeny, its evolution is self-limiting. Besides, it has no neoplastic character and does not require any treatment. Clarification of the situation and the patient's awareness are enough.

Imaging aspects are fundamental!

At its initial stage, comprised predominantly of richly cellularized soft tissue, the periapical cemento-osseous dysplasia presents itself as partially regular radiolucent areas in the apex of one or more teeth, which may be at the same or different stage of evolution (Figs 1, 4 and 6).

At the intermediate stage, the images are mixed and the lesions are characterized by irregular and small radiopaque areas, which permeate the initially radiolucent lesions (Figs 2 and 5).

At the final stage, which has not time predictability to be shown by means of imaging, the lesions become predominantly opaque, with an irregular radiolucent halo. Generally, there is direct connection of the mineralized areas with the image of the tooth apexes involved (Figs 3 and 5). The images are very distinctive compared to the ones of hypercementosis, focal sclerosing osteitis or idiopathic periapical osteosclerosis.

A diagnostic difficulty which may be faced refers to the situation in which there is still one of the lesions (Fig 5). At the radiolucent initial stage, it may be confused with a chronical periapical lesion, but the tooth will reveal pulp vitality when tested.

It is important to highlight that the endodontic treatment always represents a therapeutic measure to be adopted after a safe diagnosis. The endodon-

tic treatment should not be applied as a diagnosis resource for making a diagnosis by process of elimination based on the evolution of what may happen. The safe diagnosis of periapical cemento-osseous dysplasia can be made from radiographic and/or tomographic imaging.

Microscopic aspects: biopsies do not have to be performed!

At its initial stage, it is possible to notice a tissue of connective nature which is richly cellularized by cells that are apparently fibroplastic (Fig 1).

At some points, these fusiform and star-shaped cells become polyhedral, with a morphologic aspect of osteoblasts and/or cementoblasts, under which we can notice the eosinophilic deposition of a cemento-osseous matrix to be mineralized (Fig 2).

The cemento-osseous matrix is subjected to mineralization, going through an intermediate stage characterized by the formation of areas with varying degrees of organization. The mineralization pattern has a strong basophilic or dark purplish coloration, with apposition lines that are very similar to those of the cementum (Fig 3).

At the final and more mature stages, just a few areas of the richly mineralized connective tissue can be observed. The predominant scenario is of large areas of cementum-ossification, involved more peripherally by a connective tissue with rough capsular organization (Figs 3 and 5).

Important points of the diagnosis and clinical conduct

It is not rare to find cases of periapical cemento-osseous dysplasia in which the six lower anterior teeth have been treated by an endodontist. Periapical cemento-osseous dysplasia does not demand any treatment itself: it is asymptomatic and self-limited. At the latest stages, microbial contamination of the cemento-osseous densely mineralized tissue may contribute with microbial dissemination over a great area of the adjacent osseous tissue. The endodontic treatment would be advisable due to other reasons, such as the pulp necrosis caused by caries or in case of dental trauma. Nonetheless, it is not recommended for periapical cemento-osseous dysplasia⁶ itself.



Figure 6. Female patient, at the age of 40, melanodermic, with anterior teeth revealing periapical cemento-osseous dysplasia at its initial stage. The patient's teeth were almost exposed to endodontic treatment by a procedure mistake. The endodontic treatment must not be applied aiming at diagnostic by process of elimination or according to the process evolution. It can only be applied after a safe and accurate diagnosis. The safe diagnosis of periapical cemento-osseous dysplasia may be performed by means of imaging, with lesions typically found in teeth that show preserved pulp vitality and adjacent bone in normal conditions.

Something else that should be considered in the diagnosis of periapical cemento-osseous dysplasia has to do with investigating other areas of the jawbone and the maxilla with other cemento-osseous lesions. The melanodermic subjects are highly predisposed to having cemento-osseous lesions.

Maxillary cemento-osseous dysplasia^{9,10} may appear in three different ways:

- a) Periapical cemento-osseous dysplasia.
- b) Focal cemento-osseous dysplasia, with aleatory and isolated areas – especially the jawbone.
- c) Florid cemento-osseous dysplasia, which affects the two or four quadrants of the jawbone or the maxilla, respectively.

Whenever one of these dysplasias are identified in a patient, the occurrence of the other types should not be rejected, as they are sometimes associated. Thus, it is part of the protocol searching for other areas of the cemento-osseous dysplasia in the maxilla of periapical cemento-osseous dysplasia carriers.

The prognosis of the teeth with periapical cemento-osseous dysplasia is good and they will be hardly lost due to this disease. When tooth loss happens, it is generally due to the inflammatory periodontal disease at advanced stage, associated to bacterial plate and subgingival calculus.

The diagnosis of periapical cemento-osseous dysplasia must be made from the interaction of the clinical and imaging data and it does not require biopsy for microscopic analysis.^{4,5} The patient must be monitored every year, with clinical and imaging exams, for evolution control and necessary care.

Differential diagnosis: considerations

The images of periapical cemento-osseous dysplasia are very different if compared to the likely situations verified in its differential diagnosis: hypercementosis, focal sclerosing osteitis and idiopathic periapical osteosclerosis.

Hypercementosis: Teeth affected by hypercementosis have cudgel-shaped roots, which are also capacious due to the continuous and slow deposition of

new cementum layers, especially in the apical third. The radicular limits are uniformly preserved, as well as the periodontal space. In periapical cemento-osseous dysplasia, the roots are fused, and the lesions continue towards the irregular and mineralized structures of the lesion, and it is impossible to individualize them in relation to the lesion. The capsular peripheral tissue is irregular and its thickness varies.

Focal Sclerosing Osteitis related to Occlusal Trauma

– Among the signs of occlusal trauma lesions induced by occlusal interferences, traumatic occlusion or parafunctional events may result in focal sclerosing osteitis in the apical region. The periodontal space, in these cases, is preserved and the radicular limits are precisely distinct, even in cases in which the apical resorption takes place. The sclerotic region does not show a radiolucent halo, as it happens in the periapical cemento-osseous dysplasia. In its images, the sclerotic area continues naturally within the trabeculae of the adjacent bone.

Idiopathic Osteosclerosis – of unknown cause, some sclerotic areas persist in the jaw bones, especially the posterior region of the jaw, without the pos-

sibility of defining in a direct way a cause and effect relationship. In many cases, idiopathic osteosclerosis is periapical and should be part of the differential diagnosis of periapical cemento-osseous dysplasia. When it comes to periapical idiopathic osteosclerosis, the radicular limits are preserved, and so is the periodontal space. Its image goes along with the adjacent trabeculae, without a radiolucent halo.

Final considerations

Despite the dogmatic information according to which ‘if a tooth has periapical lesion, there is no longer pulp vitality’, it does not correspond to reality. There are periapical lesions in which the teeth show pulp vitality. Among four clinical situations where it occurs, this paper analyzed periapical cemento-osseous dysplasia, which does not require any treatment, unless for monitoring over time.

Endodontic treatment is not advisable for this disease, even though many cases are found with canals previously exposed to obturation, which is an attempt to diagnose or treat, even without a safe and definite diagnosis.

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