Florid cemento-osseous dysplasia: care and when to do the endodontic treatment

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ABSTRACT

Florid cemento-osseous dysplasia is a sclerosing bone disease that affects the alveolar process of the jawbones. In most cases, it involves the mandible bilaterally, but may affect three and even all four quadrants. If bacteria common in the oral microbiota and in the root canals reach the bone affected by florid cemento-osseous dysplasia, this disease may very likely progress to secondary osteomyelitis and result in extensive mandibular destruction and bone loss. Patients with florid cemento-osseous dysplasia should be referred to endodontists before or after a diagnosis is made. Initial periapical – and even panoramic – radiographs may suggest the presence of chronic periapical inflammation, which may lead to a misdiagnosis and to a decision to perform an inadequate endodontic intervention in a tooth whose pulp is, in fact, still vital. After a diagnosis is made, endodontic treatments should only be performed if there is no pulp vitality. All procedures should be performed under well-controlled systemic drug protection and rigorous aseptic conditions to avoid transient bacteremias and direct bacterial contamination of the periapical and periodontal bones, which may progress to secondary osteomyelitis of the mandible.

Keywords: Bone dysplasia. Florid cemento-osseous dysplasia. Osteomyelitis. Bone diseases.

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The human body is organized in tissues so that the internal environment and its vascularized connective tissues are not exposed to the external environment, which is contaminated by an exuberant microbial life composed of bacteria, fungi, viruses, parasites and other organisms.

On the body surfaces, such as the skin and gastrointestinal, genitourinary and respiratory mucosas in direct contact with the environment, 100 trillion bacteria and other similarly high numbers of the other microbial life forms compose the microbiota.

The internal environment should be seen as sacred, because the organism does not tolerate the invasion of microbial life; for this reason, the body is efficiently protected by skin and mucosas. In the gingiva, junctional epithelium has this protective function, and, in teeth, enamel isolates the dentin-pulp complex from the external environment.

When microorganisms reach connective tissues, very efficient defense mechanisms are triggered to eliminate them: inflammation and immunological responses. In systemically compromised patients, such as those with immunodepression, uncontrolled diabetes, anemia, cancer, alcoholism, undernourishment and debilitating systemic diseases, microorganism invasions that are apparently not very aggressive for healthy human beings may lead to severe fatal infections, because their defense mechanisms are not capable of eliminating the foreign invaders efficiently.

Before surgeries and other procedures that may break the barrier of the protective tissues and allow microbes to invade the organism of systemically debilitated patients, special efforts should be made to avoid the procedure or, whenever it is not possible to avoid it, to protect the organism with drugs and strict aseptic control.

Advances in Medicine have enabled patients with debilitating diseases to have longer lives among us and to have dental, and particularly endodontic, problems treated. Endodontic treatments, even when conducted under aseptic conditions, allow microorganisms to invade the organism in the periapical region, especially in cases of pulp necrosis followed by contamination of root canals.

In sclerosing bone diseases, microbial contamination by agents common in the oral microbiota may lead to severe osteomyelitis and osteoradionecrosis and expose patients to the risk of losing their mandible. Severely sclerotic bones and, in consequence, tissues disorganized by fibro-osseous diseases are frequently contaminated secondarily, leading to mandibular osteomyelitis.

In systemically compromised patients or those with sclerosing bone diseases, endodontic treatments should only be conducted when there is pulp necrosis and contamination. In these cases, treatments should be performed under strict aseptic conditions and control with medications. Under any other circumstances, endodontic treatments should be avoided, because the risk of osteomyelitis and sepsis is extremely high.

Objectives

Of all sclerosing bone diseases, florid cemento-osseous dysplasia has been diagnosed more frequently. The risk of endodontic treatments in patients with this condition should be very carefully evaluated. Treatments are often conducted without a clear indication in teeth whose pulp remains vital. This study reviews the characteristics of florid cemento-osseous dysplasia and the specificities of each clinical specialist contribution to building a knowledge base about this disease, as has already been done for Orthodontics.

In Endodontics, several special steps should be taken after the diagnosis of florid cemento-osseous dysplasia is made. The objective of this study is to discuss the special attention that should be paid to the accurate diagnosis of this disease, revisiting contents already presented elsewhere[1], but directing their applicability to clinical endodontists working in their private practices.

Concept and reasons for the “apparent” increase in prevalence

From the moment Brazil adopted a national oral health plan and the social and economic conditions of populations with lower incomes allowed them to use health services, several diseases changed their prevalence in the general population. In Brazil, over 75% of the population has genes of African ethnic groups. At the same time, there was an increase in the knowledge about florid cemento-osseous dysplasia and in the number of cases diagnosed, particularly using panoramic radiographs.

Florid cemento-osseous dysplasia is not rare, and many cases are diagnosed in imaging documentation centers (Fig 1 to 4). It is more frequent among black women about 40 years old at the time of diagnosis, with a prevalence of 5.5% in this group.[2-6]

Although more common among black women, it does not affect them exclusively and may be found in white people. Florid cemento-osseous dysplasia is a sclerosing bone disease that affects the mandible bilaterally in most cases, but may also involve three and even all four quadrants. In this process of intense and abnormal bone sclerosis, normal bone is replaced with densely formed bone that is irregularly distributed and mottled with radiolucent soft tissue, and the alveolar process is specifically affected (Fig 1 to 4), including interdental and interradicular septa.

As the disease progresses, newly-formed bone invades the periodontal space and consolidates with tooth roots, without ever compromising pulp vitality or tooth position in the dental arch. These characteristics indicate the need of a precise diagnosis of the disease by an endodontist. Endodontic treatment is not necessary and is contraindicated when the pulp remains vital.

Bone and its characteristics: the basis to understand florid cemento-osseous dysplasia

The human skeleton is fully renewed every 2 to 4 years in children and in 4 to 10 years in adults. Bones are composed of soft and mineralized tissues. Mature bone cells are osteoblasts, osteocytes and clasts, which, in sync with other components, such as macrophages, promote bone remodeling, whose primary purpose is to control the levels of mineral ions in the blood and tissues.

Constant bone remodeling confers great adaptability to bone and, according to stimuli applied to its structure, may reorganize the shape and volume of bone design. The adaptive and reactional capability of bones is clinically very important in the daily practice in Dentistry.

Bone reactional capabilities and bone resistance to stimuli or aggressors depend on three fundamental factors, which may determine the types of lesions that a certain action produces in the affected bone:

1) Local bone morphology

More compact or dense cancellous bone has small marrow spaces and little room for the more abundant inflammatory exudates. Very early on, any inflammation may increase the pressure in the small marrow spaces, compressing vessels and slowing venous return, which leads the marrow tissue to necrosis more rapidly. An area of necrotized bone marrow tissue may be the ideal spot for bacteria to lodge and form microbial biofilm.

Bone with more sparse or loosely distributed trabeculae has, in case of an aggressive event, more space for inflammatory exudates and infiltrates, with longer times for local aggressive agents to be eliminated, which increases its defensive capability of eliminating aggressors.

We may logically conclude that bone that is more compact is physically stronger, but biologically weaker, because its inflammatory process affects it very rapidly. The contrary is true of less compact, more cancellous bone: it allows more time and room for inflammation tools to operate against aggressors.

2) Intensity and duration of aggressive events

Mild and constant aggression, referred to as chronic, promotes, as any other aggressive agent, an acute initial inflammation that in hours progresses to a mild or moderate chronic phase, with a limited accumulation of mediators locally.

Many mediators of the inflammatory exudate induce bone resorption, but have bipolar effects: when highly concentrated, they induce a predominantly clastic activity; but when found at low levels in the same osseous environment, they induce the osteoblastic action of synthesis, and new bone formation predominates on the trabecular and cortical surfaces.

Mediators that induce new bone formation on the trabecular and subperiosteal surfaces gradually change local bone morphology, which remains organized. As irritation increases its power to cause lesions, new bone formation may also occur, but not in such a well organized form.

Rapid and intense aggression, referred to as acute, promotes acute initial inflammation, but much more exudative and rich in mediators inducing bone resorption, They may, therefore, lead to the formation of areas of necrosis of the bone marrow and the endosteal tissue and of osteocytes.

Mild or chronic aggressive events may induce new bone formation and reactions that produce synthesis

predominantly, whereas severe or acute aggressive events lead to resorptive, osteolytic or destructive bone reactions.

3) Systemic state of the host

The systemic state of the host is determinant for bone reactions to aggressive events. Osteomyelitis occurs only in:

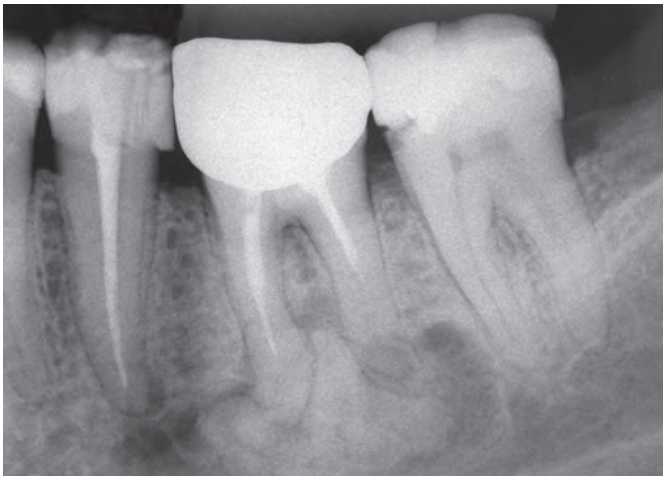

Figure 1. Single periapical lesions in the apical region, a finding that differs from those in benign cementoblastomas. The radiolucent areas are mixed and interspersed with radiopaque images of the lesion, composed of cemento-osseous tissue. The periapical region of the premolar is also affected. This presentation may also be found in cemento-ossifying fibromas and other fibro-osseous lesions. To diagnose florid cemento-osseous dysplasia, the four quadrants of the jawbones should be examined, because this disease is usually bilateral and affects the mandible primarily. In these lesions, the teeth have vital pulps and do not require endodontic treatment.

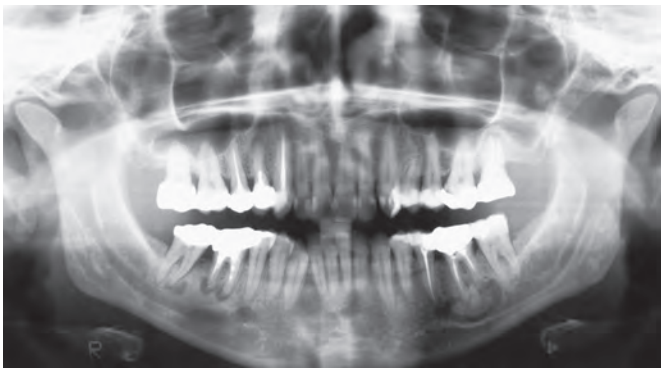

Figure 2. On the other side of the same mandible seen in Figure 1, the periapical region shows radiolucent areas, also seen in other underlying areas. The diagnosis of florid cemento-osseous dysplasia should be made based on clinical and imaging studies whenever possible. In these lesions, the teeth have vital pulps and do not require endodontic treatment.

1) Systemically compromised patients, such as those with uncontrolled diabetes, immunodepression, anemias, cancer, those undergoing chemotherapy for cancer, chronic alcoholics and those undernourished due to irregular diets or socioeconomic difficulties.

2) Patients with extensive local sclerosing bone diseases, such as florid cemento-osseous dysplasia and Paget's disease.

The areas of bone that undergo irradiation develop osteomyelitis and, when common bacteria affect these sites, osteoradionecrosis. This may be the consequence of: a) chronic hypoxia due to endarteritis obliterans, which hinders the flow of blood into the cells; b) hypovascularization; c) hypocellularity in the irradiated area because of a very low mitotic index, which reduces reparative and reactional capabilities; and d) the death of osteocytes, cells that are essential for bone histophysiology.

When a patient is healthy systemically, the same causes that may induce osteomyelitis promote osteitis, also a type of inflammation, but local and focal and with less serious consequences, because the osteolytic areas remain restricted and small, and there is a predominance of areas of bone sclerosis with very few symptoms. The prognosis of osteitis is very good.

The diseases referred to as odontogenic fibro-osseous lesions

Since 1971, the WHO, as well as Pindborg[7] and other authors, have attempted to unify the nomenclature and classification of odontogenic tumors, among which are the fibro-osseous lesions. In their first publication, six lesions were defined, four of which were included in the group called "cementomas": cementifying fibroma, periapical cement dysplasia, benign cementoblastoma and gigantiform cementoma. The other two diseases listed as odontogenic fibro-osseous lesions, although much more closely associated with bone, were ossifying fibroma and fibrous dysplasia.

The difficulty of nomenclature and classification uniformity for these lesions lies on the impossibility to distinguish whether the mineralized tissue in them is cement or bone, even when under light or scanning electronic microscopy or immunohistochemistry. In 1989, Burkhardt[8] analyzed two fibro-osseous lesions using light microscopy, electron microscopy and immunohistochemistry and found that some mineralized tissues retained a dentine nature.

This study focuses on one of these lesions, previously called cementomas, and also gigantiform cementoma for a long time, that are now universally known as florid cemento-osseous dysplasia.

Is the gigantiform cementoma the same as the florid cemento-osseous dysplasia?

The gigantiform cementoma was initially characterized as a benign odontogenic lesion that affects middle-aged black women and is typically multiple and symmetrically distributed. Radiographically, it appears as dense and lobulated masses that are microscopically composed of a cement-like tissue.

However, in 1976 the term florid cemento-osseous dysplasia was coined by Melrose et al.[9] to describe an exuberant multiquadrant fibro-osseous lesion in which the bone was gradually replaced with a fibro-cemental tissue. These characteristics are exactly the same as those found in the original description of a gigantiform cementoma.

The term florid cemento-osseous dysplasia was largely accepted also to describe entities previously reported on as multiple cemento-ossifying fibroma, multiple sclerosing osteomyelitis and sclerotic cemental masses. The term florid cemento-osseous dysplasia was also adopted for the cases of gigantiform cementoma.

The need to maintain the term gigantiform cementoma or familial gigantiform cementoma, however, was demonstrated in 1989, when Young et al.[10] reported on five generations of the same family with familial gigantiform cementoma compatible with an autosomal dominant mode of inheritance. Although the two entities were indistinguishable under imaging and microscopic examination, the familiar gigantiform cementoma has a familial or hereditary character, whereas the florid cemento-osseous dysplasia does not.

Florid cemento-osseous dysplasia has its greatest prevalence among black women, but familial gigantiform cementoma does not have this predilection. Familial gigantiform cementomas produce large expansive and asymmetrical masses that are not found in florid cemento-osseous dysplasia. Several authors recommend that these two lesions, although the points in common, should be classified separately as distinct entities.[3]

Florid cemento-osseous dysplasia may be focal and generalized

The term florid cemento-osseous dysplasia was used in 1976 for the first time, when Melrose et al.[9] described an exuberant multiquadrant fibro-osseous disease characterized by a process of bone replacement with a type of fibro-cemental tissue in the jawbones. The term florid cemento-osseous dysplasia was largely accepted also to describe entities previously called multiple cemento-ossifying fibroma, multiple sclerosing osteomyelitis and sclerotic cemental masses.

Summerlin and Tomich[11] reanalyzed cases of more localized lesions, such as cementifying and central ossifying fibromas, fibrous dysplasia of the jawbones, chronic sclerosing myelitis, sclerotic cemental masses and fibro-osseous lesions, under several aspects. They concluded that the clinical entity called focal cemento-osseous dysplasia was the initial phase of florid cemento-osseous dysplasia, which gradually extends to other regions to form the full or generalized stage of the disease.

This initial stage of florid cemento-osseous dysplasia, called focal cemento-osseous dysplasia, may mimic several other fibro-osseous lesions of the jawbones, particularly central cementifying or ossifying fibromas. Therefore, special attention is required to establish a differential diagnosis, as this is a benign tumor with variable degrees of local aggressive potential. Focal florid cemento-osseous dysplasia is only the localized and still initial form of florid cemento-osseous dysplasia, but it is not a different clinical entity or disease.

In turn, cemental or typical periapical cemento-osseous dysplasia of the mandibular incisors region may be only a uniform manifestation of the same focal cemento-osseous dysplasia, but this has not been definitely established. Does cemental or periapical cemento-osseous dysplasia progress to florid cemento-osseous dysplasia? Clinical experience and the literature reveal that such progression is not the case. When together, in the same patient, they seem to be a fortuitous and rare association.

The inception of florid cemento-osseous dysplasia

Florid cemento-osseous dysplasia begins its development when bone is reabsorbed and replaced with fusiform and polyhedral cells that form a discretely fi-

brous connective tissue, probably bilaterally and simultaneously, at a certain point of the mandible, in the region of molars and premolars. These areas, when visible on imaging studies, are radiolucent or hypodense because they are osteolytic and irregular.

After a certain time, these polyhedral cells begin depositing a disorganized, irregular and randomly distributed collagen matrix upon which the foci of basophil mineralization initiates. At this point, its structural and organizational pattern resembles immature bone, a disorganized form of mature bone, or a cementoid tissue.

On radiographs, radiolucent areas have irregular and localized radiopaque areas that gradually unite to form radiopaque masses, usually surrounded by irregular radiolucent areas that have an interface with the normal adjacent bone and are filled with soft tissues not yet mineralized. In this initial stage, there are no clinical symptoms and, up to some time later, no signs on CT scans or radiographs (Fig 1 to 4). This process tends to be bilateral, and asymptomatic, and it gradually progresses up to the moment when it is diagnosed on imaging studies performed for other clinical reasons.

What factors may trigger this pathological process? Florid cemento-osseous dysplasia probably originates in a disruption of cement formation in the periodontal ligament resulting from inadequate differentiation of periodontal ligament stem cells. Under normal conditions, these cells differentiate daily into cementoblasts, fibroblasts and osteoblasts of the cancellous bone. A disruption in the differentiation of periodontal ligament stem cells may explain why the mineralized material in these cases resembles cement and bone interspersed with fibrous tissue, similar to that of the periodontal ligament. It may also explain why the mineralized masses are not encapsulated.

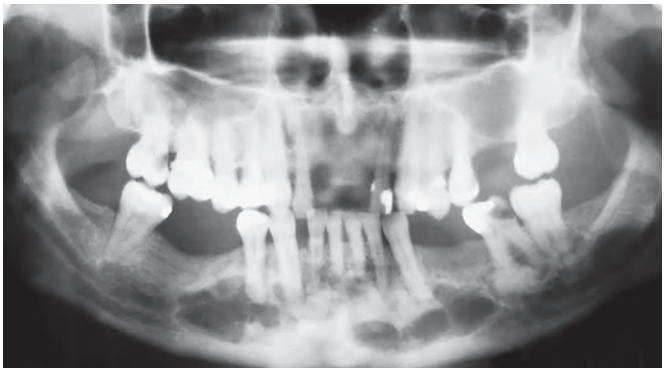

Figure 3. Initial stage of radiolucent lesions of florid cemento-osseous dysplasia, not yet mineralized. The analysis of isolated periapical images may lead to a misdiagnosis of periapical inflammatory lesions, such as periapical periodontal cyst, periapical granulomas, periapical cemento-osseous dysplasia, benign cementoblastoma, cemento-ossifying fibroma, or other fibro-osseous lesions of the jawbones. In these lesions, the teeth have vital pulps and do not require endodontic treatment.

Clinical and imaging characteristics of fully established florid cemento-osseous dysplasia

Florid cemento-osseous dysplasia has no symptoms or clinical signs. The pulp of the affected teeth is vital, and their color is normal. The cortical plates do not buckle and, therefore, there is no increase in volume of the jawbones, nor any asymmetries. The areas affected are normal at palpation, and the underlying mucosa is also normal (Fig 1 to 4).

Radiographically, there are radiopaque masses that resemble cotton wool or irregular flowerbeds and that are randomly distributed in the region of mandibular premolars and molars, particularly in the periapical region of these teeth. Their appearance on imaging studies is mixed, with radiopaque areas interspersed with radiolucent areas (Fig 1 to 4).

In the space limited by the cortical plates of the jawbones, florid cemento-osseous dysplasia gradually involves the alveolar bone of the interdental and interradicular spaces. In these dental regions, it is not possible to see a radiolucent periodontal space or a radiopaque lamina dura, but there is no root resorption by replacement, no matter how advanced the disease is, which is a sign that the periodontal ligament is still there.

Radiopaque areas filled with soft tissue not yet mineralized are seen around the "cotton wool" formed by the radiopaque areas of the cementoid or osteoid tissue (Fig 1 to 4). The superposition of images reveals that these radiopaque and radiolucent findings appear randomly distributed on various radiographic views.

The characteristics described here are especially detailed on CT scans and tridimensional reconstructions. Between these masses and the teeth, there are points where no periodontal spaces are visible, not even on thinner CT cuts, but there is no replacement resorption. These masses extend from one cortical plate to the other on CT cuts.

As time progresses, the degree of compromise of the jawbones, especially of the mandible, increases because the disease extends to the region of the third molars and canines and may even reach the midline, and lesions may be found in two quadrants. In some cases, these radiopaque areas surround and involve unerupted teeth found in the region.

In most cases, only two quadrants are affected, but the posterior regions of the maxilla may be compromised, usually at a lesser degree than the mandible.

A very important clinical sign for the diagnosis of florid cemento-osseous dysplasia is the appearance of a yellowish material, similar to bone, that is exposed through the oral mucosa due to perforations, unexplained at first. When it occurs, it may indicate the presence of low virulence contamination of the lesion, not yet diagnosed and unknown to the patient.

What should be the clinical management of cases of florid cemento-osseous dysplasia?

The reactional and reparative capability of bone with florid cemento-osseous dysplasia is severely limited. Bone marrow spaces are few and very small. Any amount of inflammatory exudate (liquid) and infiltrate (cells) compress the vessels early on and lead to necrosis.

Any bacteria common in the oral microbiome may reach the sclerotic bone affected by florid cemento-osseous dysplasia and lead to inflammation, which rapidly progresses to purulent or chronic suppurative osteomyelitis forming multiple fistulae and having mutilating consequences for the patient.[2,3,14,15,16]

These bacteria rapidly form a mixed microbiota, which colonizes the internal sclerotic surfaces of the bone and hide in the necrotic areas, which makes it difficult for medication to reach them and be effective. When this occurs, the treatment should be surgical, with debridement and significant loss of mandibular structures, as in the case of primary chronic suppurative osteomyelitis.

The main strategy for the treatment of florid cemento-osseous dysplasia is to avoid that the compromised bone gets in contact with the oral microbiome. Such measure should be similar to those adopted in the case of chronic inflammatory periodontal disease associated with bacterial plaque on teeth, caries followed by pulp necrosis, chronic periapical diseases, and dental and maxillary trauma and surgery, such as extractions and dental implant installations.

After a definitive diagnosis of florid cemento-osseous dysplasia, the endodontic treatment should be performed only when there is confirmed loss of pulp vitality and under preventive antibiotic therapy.[17] Aseptic conditions should be extreme. The potential for a tooth with infected pulp necrosis with or without periapical lesion to lead to osteomyelitis is high. As soon as a tooth in these conditions is detected, endodontic treatment should be performed urgently.

The oral mucosa should not be ulcerated or operated on, not even to obtain an incisional biopsy for a microscopic diagnosis of florid cemento-osseous dysplasia. The definitive diagnosis of florid cemento-osseous dysplasia should be based exclusively on clinical and imaging findings.

A simple periodontal scaling procedure, the installation of dental implants or an extraction may suffice to induce suppurative chronic osteomyelitis secondary to florid cemento-osseous dysplasia.[14] In case of an urgency, emergency or other inevitable events that may expose the compromised maxillary bone, severe preventive antibiotic therapy should be followed according to strict parameters of time, dose and choice of drug.

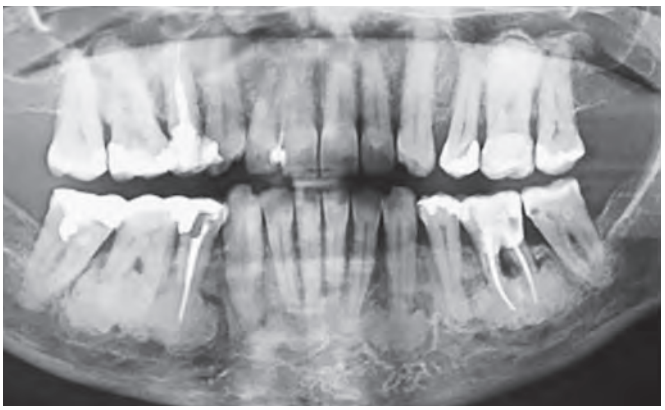

Figure 4. Final stage of florid cemento-osseous dysplasia, with the predominantly mineralized lesions surrounded by small and irregular radiolucent halos. When examined separately, the periapical images may suggest a misdiagnosis of periapical cemento-osseous dysplasia, benign cementoblastoma, cemento-ossifying fibroma, or other fibro-osseous lesions of the jawbones. In these lesions, the teeth have vital pulps and do not require endodontic treatment.

Despite all the care taken, the patient with florid cemento-osseous dysplasia may develop secondary chronic suppurative osteomyelitis because of the contamination of the compromised bone by transient bacteremias, which the human body has almost every day. These transient bacteremias may occur during personal hygiene, mastication or other very simple everyday actions.

Although well known in the literature, many dentists still struggle with the diagnosis of florid cemento-osseous dysplasia[4,5,18,19]. Sometimes the signs and symptoms of undiagnosed florid cemento-osseous dysplasia are assigned to the action of bisphosphonates on bone remodeling, which is a serious conceptual and practical mistake.[20]

It is fundamental that the patients and their families, after a clear diagnosis of florid cemento-osseous dysplasia, understand the disease and avoid equivocal procedures suggested by other professionals. They should take all the measures necessary to block the access of bacteria to the compromised area by adopting a healthy and self-directed lifestyle.

What are the dental treatment contraindications in the cases of florid cemento-osseous dysplasia?

Surgical procedures should be avoided to prevent the access of bacteria from the microbiota to bone, as they may lead to the development of chronic suppurative osteomyelitis secondary to florid cemento-osseous dysplasia.

Extractions should only be performed when extremely necessary. The extraction of unerupted teeth should be avoided as much as possible, and these teeth should be controlled with follow-up radiographs taken yearly.

The installation of osseointegrated implants in the compromised areas is strictly contraindicated, because the risk of developing secondary chronic suppurative osteomyelitis is very high.

All surgical procedures should be avoided, including those for orthodontic purposes, such as forced orthodontic eruptions and installation of mini-implants and mini-plates. Orthodontic treatments are contraindicated because it is not possible to move teeth in areas affected by florid cemento-osseous dysplasia. Some microscopic views of the disease show the absence of full segments of the periodontal ligament, the structure responsible for orthodontic movements.

The contraindications of procedures in the jawbones of patients with florid cemento-osseous dysplasia have some important details. If the disease affects only the mandible at the time of the procedure, tooth movements and dental implants may be performed in the areas not compromised in the maxilla. The same applies to endodontic treatments, endodontic surgeries and extractions, even of unerupted teeth. The installment of mini-implants and mini-plates should always be avoided, because they offer access to bacteria during all the time that they are in the mouth, which may result in daily events of bacteremia.

Final considerations

In Endodontics, numerous special measures should be taken when treating patients with florid cemento-osseous dysplasia.

1. Before a final diagnosis is made

In its initial stage, radiographs of florid cemento-osseous dysplasia have radiolucent areas, which may be periapical. At this point, an equivocal diagnosis of chronic periapical inflammation may be made, and inadequate endodontic treatments may have serious consequences because of the transient bacteremia to which they may lead.

The diagnosis of florid cemento-osseous dysplasia should always be made using panoramic radiographs or CT scans, or both, so that the two sides of both jawbones are examined. This lesion is almost always mandibular and bilateral, affecting the region of premolars and molars more frequently.

Lesions seen on single periapical radiographs of only some teeth may very likely induce diagnostic errors and a misdiagnosis of chronic periapical inflammation. In these cases, however, the pulp of the teeth affected is vital. Therefore, a panoramic radiograph or a CT scan, or both, should be indicated.

To avoid this type of misdiagnosis, we should keep in mind that teeth with vital pulps should never be treated endodontically before there is a safe and definitive diagnosis of the lesion that they may have. The teeth affected by florid cemento-osseous dysplasia have vital pulps even at advanced stages of the disease.

In teeth with vital pulps and periapical or lateral periodontal lesions, endodontic treatment should never be used as a diagnostic tool, as it is a treatment, and not a diagnostic procedure. Sometimes the patient reports that "I had the treatment to see what would happen later, to see if the problem would be solved", which is a serious management error.

2. After a final diagnosis is made

After the diagnosis of florid cemento-osseous dysplasia is made, the therapeutic management of the case consists of lifelong patient follow-up, as there is no treatment for this disease, but complications should be avoided, as mentioned above.

Endodontists, in these cases, should be consulted when a tooth has pulp or periapical changes and requires endodontic treatment. The same should apply to decisions about medications to protect the tooth from transient bacteremia, and extremely aseptic measures should be taken. The most common bacteria in the oral microbiota and in the infected canal in the sclerotic bone may be responsible for suppurative osteomyelitis secondary to florid cemento-osseous dysplasia.

In patients with florid cemento-osseous dysplasia, the internal environment should be extremely well isolated to avoid contamination of the bone affected. The patient and the professionals involved in the treatment should create the context to avoid periodontal diseases, caries and pulp diseases, as well as mucosa ulcerations, extractions and other surgical procedures. Patients and professionals should be aware of the characteristics of this disease, so that they may avoid its serious complications when treatment for these conditions is needed.

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