Florid cemento-osseous dysplasia: one of the few contraindications to osseointegrated implants

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Abstract: *Florid cemento-osseous dysplasia represents one of the few clinical contraindications to osseointegrated implant placement. As in any other surgical procedure, including biopsy, implant placement might open up the doors to bacteria access to the bone environment. Highly sclerosed irregular bone is appropriate for bacteria proliferation and formation of exuberant microbial biofilm, thereby hindering access not only of local cells and molecules of organic defense, but also of antibiotics potentially administered to treat secondary purulent chronic osteomyelitis commonly found in florid cemento-osseous dysplasia patients. The disease is a disorder of the maxilla, established during the process of bone remodeling; and despite its high frequency, its causes or associated factors remain unknown, except for its predominance among afrodescendents, especially middle-aged women.*

Keywords: *Bone dysplasia. Florid cemento-osseous dysplasia. Fiber-bone lesions.*

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INTRODUCTION: THE NAME!

Titanium-based dental implants are accepted by hard and soft tissues in such a way that, in their structural and molecular innermost, they integrate in all levels, including the molecular one. The inflammatory process along with its final repair phase act on and around titanium dental implants and do not result from the presence of an implant alone, but from surgical procedures necessary for implant placement.

Theoretically, should implant placement be possible without surgical procedures, there would be integration with adjacent tissues as a result of ongoing renewal processes typical of connective tissues, including bone tissues, happening at a lower or higher pace. Osseointegration results from the repair capacity of peri-implant bone tissues of which blood clot is invaded by osteoblastic cells that deposit a new bone matrix to be mineralized. This process might happen directly on titanium-based dental implants.

Meanwhile, once it is established, osseointegration is constantly renewed by the ongoing remodeling property of bone tissue. The mineralized portion is resorbed in certain sites while being neoformed in others. It is possible to assert that an adult's skeleton is renewed a few times over the years. For maintenance and renewal, normal bone requires osteoblastic and clastic cells to function properly, simultaneously and coordinately. A defect in one of all bone remodeling components implies structurally defective bone formation.

BONE DYSPLASIA

The term "dysplasia" refers to different, disorganized, shapeless tissue formation. In this case, the latter stands for bone tissue formation. In general, it results from changes in some osteoblasts or clasts genes.

Some diseases cause osteoblasts malfunction, that is, they fail to produce proper bone matrix or fail to mineralize it appropriately. Likewise, in other pathological pictures such as osteopetrosis, the clasts do not function properly and fail to perform the resorptive process. As a result, bones become denser and harder, acquiring marble-like characteristics.[1-9]

In other pathological pictures, bone dysplasia might result from osteoblasts defective genes producing immature, soft and disorganized bone, as it occurs in fibrous dysplasia of the maxilla. In this case, defect results from mutation of the GNAS-1 gene which plays an important role in differentiating and proliferating osteoblasts.

Some types of bone dysplasia rarely affect the jaws; while others are quite common, namely: mandibular florid cemento-osseous dysplasia and fibrous dysplasia of the maxilla.[1-9]

The present study aimed at discussing and warning clinicians about the diagnosis and implications of florid

cemento-osseous dysplasia when planning cases involving the potential need for osseointegrated implants.

FLORID CEMENTO-OSSEOUS DYSPLASIA

Florid cemento-osseous dysplasia is frequently found in the dental context. It is classically characterized by being found on both sides and in the form of shapeless irregular bone that resembles cotton wads placed at irregular radiolucent sites, especially in the posterior mandible (Fig 1). Nevertheless, it might occasionally occur in patient's maxilla.[1-9] Florid cemento-osseous dysplasia is the type of bone dysplasia most commonly found in the maxilla and in areas with or without teeth, although some authors suggest that it occurs in periodontal tissues, especially the periodontal ligament.

Florid cemento-osseous dysplasia might be considered a disorder of unknown etiology, occurring during maxillary bone tissue remodeling and associated with teeth support. Presently, it cannot be attributed to known causes with well-established methodological grounds. There are reports of family cases.

It is usually diagnosed in women aged between the fourth and sixth decade of life, in addition to being commonly associated with afrodescendents.[1-9] Importantly, in Brazil, 75% of the overall population have afrodescendent genes due to the miscegenation that features the Brazilian population. Nevertheless, it should be highlighted that European whites and Japanese are not immune to florid cemento-osseous dysplasia.[6]

Clinically: Clinical examinations do not reveal tumefacted internally compromised sites, expansion of corresponding cortical plates or inflammatory process primarily associated with the process. The overlying oral mucosa is completely normal. Teeth associated with florid cemento-osseous dysplasia are not displaced and do not present root resorption or alterations in pulp vitality. Florid cemento-osseous dysplasia is asymptomatic.

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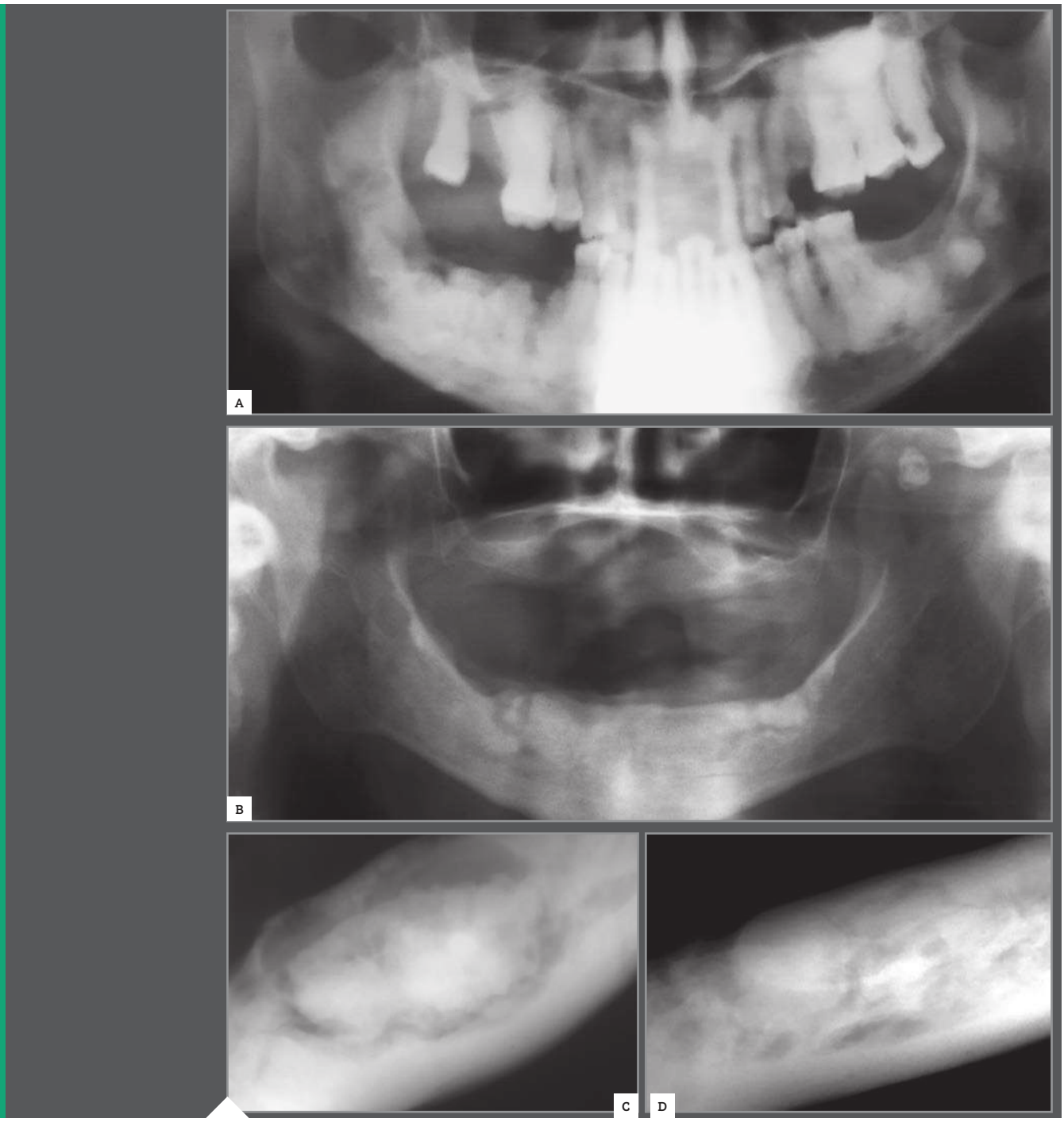

Figure 1. Imagiological aspects typical of florid cemento-osseous dysplasia in three clinical cases characterized by mixed radiolucent and radiopaque sites resembling cotton wads or flower beds without precise limits on both sides. Images suggest advanced-stage disease due to formation of cemento-osseous mineralized tissue.

Process and phases: On both sides of the posterior mandible, normal bone tissue is simultaneously replaced by stellar fusiform cells of fibroblastic pattern, producing or organizing themselves into fibrous tissue with a bundle of collagen fibers and random distribution of cells. At this phase, images reveal multiple multilocular osteolytic areas of small dimensions.

These radiolucent areas gradually allow round or trabecular clusters of basophilic staining and strong apposition lines to form by deposition of cells differentiated into shapeless osteoblasts. Mineralized tissue tends to acquire the morphology of tooth cementum. Irregular mineralized tissue clusters and trabeculae increase in size and produce radiopaque areas vaguely resembling cotton wads or flowers placed on both sides of the posterior mandible and at the center of osteolytic sites.

Over the months, these radiolucent areas increasingly present with denser irregular cemento-osseous dysplastic tissue resembling cotton wads or flower beds, thereby producing an irregular mixture of radiolucent sites predominantly consisted of radiopaque hard tissue (Fig 1).

From an imagiological standpoint, florid cemento-osseous dysplasia might be classified according to three distinct sequential aspects: 1) radiolucent or fibrous; 2) mixed or fibrous cemento-osseous; and 3) radiopaque or cemento-osseous.

Disease variation:[1,3,4] the mildest forms of the disease are as follows:

1) **Periapical cemento-osseous dysplasia** mainly affecting the periapical region of anterior mandibular teeth, especially incisors. In this region, bone is equally and gradually replaced by an area limited by dense, cellularized, disorganized fibrous connective tissue, with subsequent formation of mineralized tissue shaped as clusters and trabeculae of cemento-osseous basophilic pattern, which ends up mineralizing almost the entire region. Radiographically, it presents with radiopaque areas resembling cotton wads and is limited to the apical and periapical surface of mandibular incisors. It does not compromise periodontal support or pulp vitality, and does not require treatment.

2) **Focal cemento-osseous dysplasia** mainly occurring in one or more isolated sites of the mandible. Its clinical biological behavior is self-limiting and, in the past, it used to be diagnosed as a less aggressive form of cemento-ossifying fibroma or ossifying fibroma, especially when microscopically analyzed.

HOW TO REACH A DIAGNOSIS OF FLORID CEMENTO-OSSEOUS DYSPLASIA?

In florid cemento-osseous dysplasia, the bone becomes sclerosed or irregu-

larly dense. Its irregular, little vascularized structure is the optimal stage for bacterial colonization and microbial biofilm installation to which defense agents, such as cells and molecules, antibodies, complements, other antimicrobial agents and antibiotics have limited access.

This means that, in case of florid cemento-osseous dysplasia, any microbial agent implies a great possibility of leading to secondary purulent chronic osteomyelitis, which is difficult to be controlled, whether by means of therapeutic, surgical or medical treatment.

Thus, in this region, teeth should be preserved and kept free of chronic inflammatory periodontal disease, pulpitis and periapical lesion, with rigid and thorough control of oral hygiene and mucosa integrity. Extraction and other types of surgery, including biopsy, open up the doors for microorganisms to enter and, for this reason, should be avoided. Diagnosis of florid cemento-osseous dysplasia should be based on clinical and imaging examination. Biopsies, other types of surgery and contamination by means of communication with the oral environment should be avoided at all costs. Once diagnosis of florid cemento-osseous dysplasia is confirmed, patients should be made aware of it, family members should be investigated and periodical control should be carried out.

FLORID CEMENTO-OSSEOUS DYSPLASIA AND OSSEOINTEGRATED IMPLANTS: ONE OF THE FEW CONTRAINDICATIONS!

Surgery and/or communication with the oral environment, including incisional biopsy, must be avoided. Thus, osseointegrated implant placement should also be avoided. Florid cemento-osseous dysplasia is one of the few contraindications to osseointegrated implants.

After exposure of bone with florid cemento-osseous dysplasia, it might take months to clinically identify secondary purulent chronic osteomyelitis. However, the latter will not take place only in rare cases. Should exposure to the oral environment occur as a result of implant placement, secondary purulent chronic osteomyelitis might not occur; however, florid cemento-osseous dysplasia patients are highly susceptible to it due to bacteria that might reach the site by blood as a result of transient bacteremia.

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Secondary purulent chronic osteomyelitis is difficult to be controlled, whether by means of therapeutic, surgical or medical treatment. Even after apparent clinical improvement, suppuration and tissue destruction usually resume. For this reason, communication with the oral environment must be avoided at all costs.

Osseointegrated implants per se do not cause inflammatory and infectious problems, given that cells and tissues, including dysplastic bone, integrate to the implant surface. Nevertheless, implant placement necessarily implies communication with the oral environment which is highly contaminated despite aseptic measures. During postoperative procedures and repair process, bacteria inevitably enter the site.

FINAL CONSIDERATIONS

Majoritarily, florid cemento-osseous dysplasia represents one of the few clinical contraindications to osseointegrated implant placement or any other surgical procedure, including biopsy.

Highly sclerosed irregular bone is appropriate for bacteria proliferation and formation of exuberant microbial biofilm, thereby hindering access not only of local cells and molecules of organic defense, but also of antibiotics potentially administered to treat secondary purulent chronic osteomyelitis commonly found in florid cemento-osseous dysplasia patients.

Should florid cemento-osseous dysplasia patients receive an osseointegrated implant and yield successful clinical results, it is paramount that judicious clinical control be performed for life, given that secondary purulent chronic osteomyelitis might develop at any time. This situation might be attributed to osseointegrated implants, even though contamination might have occurred by blood, a possibility that should not be completely discarded.

Lastly, osseointegrated implant placement in florid cemento-osseous dysplasia patients presents high biological risks and should be avoided.

REFERENCES:

1. Barnes L, Eveson JW, Reichard P, Sidransky D. Genética e patologia dos tumores de cabeça e pescoço. São Paulo: Ed. Santos; 2009.
2. Cawson RA, Odell WE. Cawson's: fundamentos básicos de patologia e medicina oral. 8ª ed. São Paulo: Ed. Santos; 2013.
3. Fletcher CDM, Unni KK, Mertens F. Pathology and genetics tumours of soft tissue and bone. World Health Organization Classificiation of Tumors. Lyon: IARC Press; 2002.
4. Neville BW, Damm DD, Allen GM, Bouquot JE. Patologia oral e maxilofacial. 3ª ed. Rio de Janeiro: Elsevier; 2009.
5. Regezi JA, Sciubba J, Jordan R. Patologia oral: correlações clinicopatológicas. 6ª ed. Rio de Janeiro: Elsevier; 2013.
6. Reichard PA, Philipsen HP. Odontogenic tumors and cysts. Berlin: Quintessence; 2004.
7. Sapp JP, Philip Wysocki GP. Patologia bucomaxilofacial contemporânea. 2ª ed. São Paulo: Ed. Santos; 2012.
8. Scully C. Medicina oral e maxilofacial: bases do diagnóstico e tratamento. 2ª ed. Rio de Janeiro: Elsevier; 2009.
9. Tommasi MHM. Diagnóstico em patologia bucal. 4ª ed. Rio de Janeiro: Elsevier; 2014.