Occurrence of Multiple Giant Cell Lesions in the Jaw: Case Report

Ana Rita Pinheiro Barcessat1*, André Caroli Rocha2, Fernando Simões Morando2, Décio Pinto dos Santos Jr3, and Maria Rozeli de Souza Quirino4

1Department of health and Biological Sciences, Federal University of Amapá, Brazil
2Buco-Maxillo-Facial Surgery of Southern Regional Hospital, Brazil
3College Faculty of Dentistry, University of São Paulo, Brazil
4College Faculty of Dentistry University dade of de Taubaté, Brazil

Abstract

Simultaneous occurrence of Giant cell lesions in the four quadrants of the jaws is not a common situation. Multinucleated giant cells are structures of osteoclast behavior, confirmed by immunohistochemical studies, probably derived from a cell common to the macrophage, but there is no precision, however, as to the exact mechanism, and the etiology remains to be clarified. It has been stated that this is a reactive lesion, a developmental anomaly or a benign neoplasm, commonly associated with inflammation, hemorrhage and local trauma. This paper proposes to report the case of a 43-year-old male patient, 43 years old, who presented lesions such as like exophytic masses in the maxilla and mandible, in the four quadrants associated with residual root outbreaks with a 3-year evolution of 3 years.

INTRODUCTION

Giant cell lesions are injuries that may affect the maxillary bones centrally, or the alveolar and gingival mucosa in its peripheral variant, according to their behavior, they can be classified as aggressive and nonaggressive. [1-2]. It represents a common commitment in oral cavity and many lesions present the histological formation of giant cells permeating their stoma, however the simultaneous occurrence in the four quadrants of the jaws is an uncommon clinical situation. Multinucleated giant cells are structures of osteoclast behavior, confirmed by immunohistochemical studies, probably derived from a cell common to the macrophage but there is no precision, however, as to the exact mechanism, and the etiology remains yet to be clarified [2]. It has been stated that this is a reactive lesion, a developmental anomaly or a benign neoplasm, commonly associated with inflammation, hemorrhage and local trauma [1-4]. The aim at this paper is reporting the case of a 43-year-old male patient, 43 years old, who presented lesions such as like exophytic masses in the maxilla and mandible, in the four quadrants associated to residual pieces of dental roots with a 3-year evolution of 3 years.

CASE PRESENTATION

A 43-year-old male patient, leucoderma, complaining of an intra-oral mass and bad breath, underwent radiographic exams. He presented painless lesions such as like exophytic masses in the maxilla and mandible, in the four mouth quadrants associated to residual pieces of dental roots with a 3-year evolution of 3 years (Figure 1). Radiographic examination revealed areas of bone rarefaction, multiple residual roots and mixed-aspect lesions, lithic defects, delineated by halo of sclerosis in, reactive bone (Figure 2). Indices of Ca (calcium) P (phosphorus) and parathyroid hormone (PTH) were normal and there was no clinical evidence of systemic disorders such as cherubism neither or compatible with any syndrome. The clinical diagnostic hypotheses were of non-neoplastic proliferative processes, peripheral lesion of giant cells and fibro-osseous lesion. The lesions were removed together with the residual roots under general anesthesia (Figure 3). Histopathological examination showed strongly cellular fibrovascular tissue, with areas of erythrocytic extravasation, large giant multinucleated cells and innumerable foci of reactive bone (Figure 4). An observed a repeated pattern that was repeated in the lesions in all quadrants, confirming the radiographic examination. Histopathological diagnosis was giant cell lesion.
cell lesion. One month after surgery, the patient was well, without evidence of relapse. After one year there was no recurrence of injury.

**DISCUSSION**

Peripheral Giant Cells Lesion (PGCL) is a reactive process induced by irritant local factors and Central Giant Cells Lesion (CGCL) is an intra-osseous process of unknown etiology whose diagnosis is done through clinical and radiographic exams. Diagnosticated clinically and radio graphically.

CGCL and PGCL features are histological indistinguishable. Histological, the features of CGCL or PGCL are indistinguishable from the brown tumor of hyperparathyroidism brown tumor and from giant cell lesions of systemic disorders such as cherubism, Noonan syndrome and neurofibromatosis Type 1 [2-4].

The autosomal dominant hereditary disease called cherubism is characterized by a progressive expansion of the jaws, with replacement of the bone by a fibrous tissue with some giant cells, this expansion associated with to the eyes upward is are similar to the cherubim angels seen in the renaissance paintings, an condition incompatible condition with the patient’s clinical presentation [5-6].

Multiple giant cell lesions in the jaws have also been also associated to Noonan syndrome, as a phenotypic variation within the syndromes of the as/MAPK pathway, in which a large range of symptoms have been reported, including unusual facial characteristics and congenital heart abnormalities, however, those unusual facial characteristics and it’s wide spectrum of congenital heart defects were not identified in the present case [7-9].

Neurofibromatosis type 1 (NF1) is a neurogenic disorder attributed to second hit mutations in the NF1 gene. The development of tumors in the nervous tissues is one of its characteristic and Giant cell granuloma (GCG) is related as a clinical manifestation, however in order to diagnose as to be given the diagnosis of NF1, individuals must present have at least two features of the condition including birthmarks, skin fold and neuro fibromas, none of those characteristics could beware seen by clinical examination in the present patient [10-11].

Hyperparathyroidism is another condition in which an occurrence called brown tumor appears as a focal giant cell lesion, occurring due to increased secretion of parathyroid hormone (PTH). PTH works by increasing the release of calcium and phosphorus from bones and its secretion is stimulated by calcium and pentagastrin. However but, the patient had normal Ca (calcium) P (phosphorus) and parathyroid hormone (PTH) indices, therefore so, hyperparathyroidism was disregarded [12,13].

Clinical diagnostic hypotheses, for the present case, were non-neoplastic proliferative processes, peripheral lesion of giant cells and fibro-osseous lesion, once the masses were associated to radicular pieces areas and the patient had no family history or clinical appearance of syndromes or genetic disorders.

Aneurismal bone cyst, considered a pseudocyst with aggressive clinical behavior, was also considered for differential diagnoses.
diagnosis but there were no histories of trauma and vascular disorders [14].

Considering the patient’s poor oral condition, the radiographic aspect showing the residual roots with lesion, dental crowns with destruction and the aspect of deposited bone, the diagnostic was defined as PGC, as Since it is considered a reactive process that can be initiated at periodontal ligament, gingival or mucoperiosteum and, CGCL is an intra-osseous lesion, only the right mandibular lesion resembles a little more like a central lesion, due to the presence of a subtle granular bone pattern at the periphery of the expanded bone, in the histological and radiographic exam [15].

The surgical treatment was proposed considering the peripheral lesion of giant cells, opting for removing all lesions by curettage together with the residual roots under general anesthesia. The choice of treatment and the frequency of recurrence depend on factors such factors as the patient’s age, and heat was a young man, location, extent and clinical behavior of the lesion. The most common therapy is surgery, but non-invasive treatments with calcitonin, intraregional corticoid and interferon-α have been widely used as adjuvant especially for CGCL, but not in this case [16,17].

Central lesions are in general more aggressive and have correlation between with greater expression of RANKL and NF-κB than peripheral lesions induced by local factors and, presenting low rates of bone resorption and low recurrence, which that can explain the follow up without recurrences after one year [18,19].

CONCLUSION

The true nature of this lesion is unknown, even considering the controversy in the literature about whether it is a true neoplasm or a reactive response, the present case is clearly a reactive response to the patient’s poor oral health condition and a nonaggressive lesion, peripheral giant cells lesion, PGCL.

REFERENCES