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ORIGINAL ARTICLE

Giant cell lesion and traumatic bone cyst: pathologically related lesions?

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Abstract:

Introduction: Synchronous lesions of the jaws are reported in the literature and the etiopathogenesis can be similar in some cases. The oral surgeon must know the diseases that may occur simultaneously. **Objective:** The aim of this study was to report a case of synchronous lesions, your management, additionally discuss the etiopathogenesis correlation among these lesions and the possible evolution from one disease to the other in order to aid the professional about the management of this occurrence. **Methods:** This study reported a case of a 14-year-old female patient presenting a reddish sessile nodule in mandibular left premolars gingival region. **Results:** Radiographically, a well-defined radiolucent area involving left mandibular body was observed. An incisional biopsy of the extra-osseous lesion was performed and a giant cell lesion was histopathologically diagnosed. A surgical bone exploratory procedure revealed an empty cavity; therefore curettage to stimulate bleeding was performed in addition to the extra-osseous lesion excision. The final diagnosis was synergistic peripheral giant cell lesion and traumatic bone cyst. The 12 months follow-up did not show any relapse, moreover, the complete bone neoformation was observed. **Conclusions:** The pathogenesis of both lesions remains uncertain and a complete and detailed clinical and histopathological examination added to rigorous follow up with image exams resulted in a correct diagnosis and good prognosis of this case.

Keywords: Peripheral Giant Cell Granuloma; Bone Cysts; Jaw Cysts; Jaw Cysts/Etiology.

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INTRODUCTION

Peripheral giant cell lesion (PGCL) is a benign hyperplastic lesion with well-defined borders and is characterized by a nodular or pedicled nodule. It may affect individuals of any age, and it is more prevalent in female subjects. In addition, the most commonly affected site is the mandible[1]. Histologically PGCL presents a spindle cell proliferation with giant cells, interstitial hemorrhage, hemosiderin pigments, possibly with inflammation, mature bone or osteoid matrix[2]. The treatment of this condition is the surgical excision. Additional therapy (curettage or peripheral osteotomy) decreased significantly the recurrence rate. The recurrence rate reported for excision only is 16%, and 2.8% when associated with curettage or 0% when peripheral osteotomy was also performed[3].

Simple bone cysts (SBC) are lesions classified as a pseudocyst because it has no epithelial lining. Clinically, it is characterized by the presence of an intraosseous cavity which may be empty or contain blood or mucous fluid. It is more frequent in young individuals in the first decades of life, and usually affects the lower molar region. Radiographically, SBC presents as a radiolucent lesion with well-defined margins and may acquire a scalloped appearance. SBCs are often diagnosed accidentally since most of these lesions are asymptomatic[4]. Histologically, the specimen shows fibrovascular connective tissue, hemorrhage and vital bone fragments from the bone walls, cystic epithelium is not present[5]. The recommended SBC treatment is exploratory surgery and bone curettage; posterior bone healing was observed in the recommended SBC treatment is exploratory surgery and bone curettage; posterior bone healing was observed in most of the cases after this procedure[6].

Concomitant bone lesions may affect the jaws, and SBCs are most commonly associated with benign fibro-osseous lesions, especially fibrous dysplasia and bone cement dysplasia[7]. In the case of PGCL, a previous report has documented an association with florid cemento-osseous dysplasia in a patient with neurofibromatosis[8].

To our knowledge, no reports to date have documented an association between PGCL and SBC[9,10]. The aim of this paper is to report a case of concomitant PGCL and SBC lesions and discuss about their association and pathogenesis. The correct diagnosis is very important because treatment is different for each of these lesions. Therefore, inappropriate conduct will result in worse prognosis. This study was conducted in accordance with theDeclaration of Helsinki.

CASE REPORT

A 14-year-old female sought the Oral Medicine Department because of a lesion in the inferior alveolar ridge. The social and familiar history was impaired because this girl was confined in a young offender institution. This patient did not use any medication, however reported previous regular consumption of marijuana. Clinical examination revealed an asymptomatic, reddish, nodule with a smooth surface, firm consistency and measuring 2 cm. The patient reported an approximately 8-month history of this lesion located in the region of the left mandibular premolars, which were displaced (Figure 1A). The patient did not report traumatism in the affected zone. The thermal sensibility pulp test was positive in left inferior molars (36 and 37). Panoramic radiography revealed a well-delimited radiolucent lesion extending from tooth 33 to the ascending branch of the left jaw (Figure 1B). The patient did not present any systemic alterations.

On the basis of the clinical presentation, two possibilities were considered: a single lesion with an extraosseous and intraosseous component or two different synchronous entities. If the lesions are considered a single disease, the diagnostic hypothesis should consider

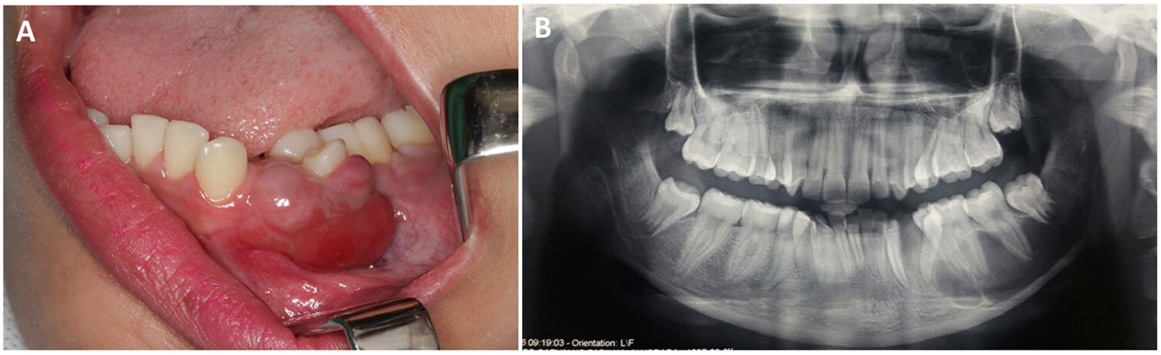

Figure 1. Initial aspect of the patient. A) Clinical image of extra-osseous nodule. B) Initial panoramic radiography.

intraosseous lesions that can perforate the cortical bone and result in extraosseous progression.

Initially, an incisional biopsy of the extraosseous lesion was performed. Analysis of the anatomopathological specimen revealed a proliferation of young spindle cells, presence of multinucleated foreign body type cells, and a richly vascularized stroma with areas of hemorrhage and mononuclear inflammatory cells. These features are compatible with those of giant cell lesions (Figure 2). In the intraosseous surgical exploration, after the mucoperiosteal flap, the cortical bone was removed with carbide bur, the aspiration was negative, and an empty cavity was found. Thus, bone walls were cured to stimulate bleeding for bone repair[11]. The extraosseous lesion was completely removed via excision and curettage; also the traumatic factors were eliminated and one premolar (34) involved inside the lesion was extracted. Thus, the final diagnosis was a SBC and a concomitant PGCL.

After 1 year of clinical and radiographic control, no recurrence of the extraosseous lesion was observed, and bone neoformation was observed in the curetted cavity (Figure 3). The patient remains under clinical and radiographic control.

DISCUSSION

There is no consensus regarding the etiopathogenesis of PGCL and SBC. Some authors suggest that PGCL may have a neoplastic character. However, others suggest a reactive origin, because these lesions are often associated with local traumatic factors such as biofilm accumulation, dental calculus, higher gingival bleeding index, and deep periodontal pockets[12]. In the present case, dental crowding and poor oral hygiene were local traumatic factors, and the patient was unable to confirm a previous trauma in the region.

Some hypotheses have been suggested about the etiopathogenesis of SBCs. These include calcium deficiency, degeneration of bone tumors, local alteration of bone growth, and intramedullary hemorrhage[10]. Among these hypotheses, intramedullary hemorrhage is currently the most accepted. It is caused by trauma, which results in the formation of an intraosseous hematoma and blood supply failure, subsequently resulting in necrosis and bone reabsorption, and finally the formation of a bone cavity[9].

Some studies have investigated possible etiopathogenic correlations between SBC, Aneurysmal Bone Cyst (ABC), and Central Giant Cell Lesion (CGCL). These lesions are caused by mechanical factors that produce an intraosseous vascular lesion with a hematoma. Three possible outcomes have been proposed for this

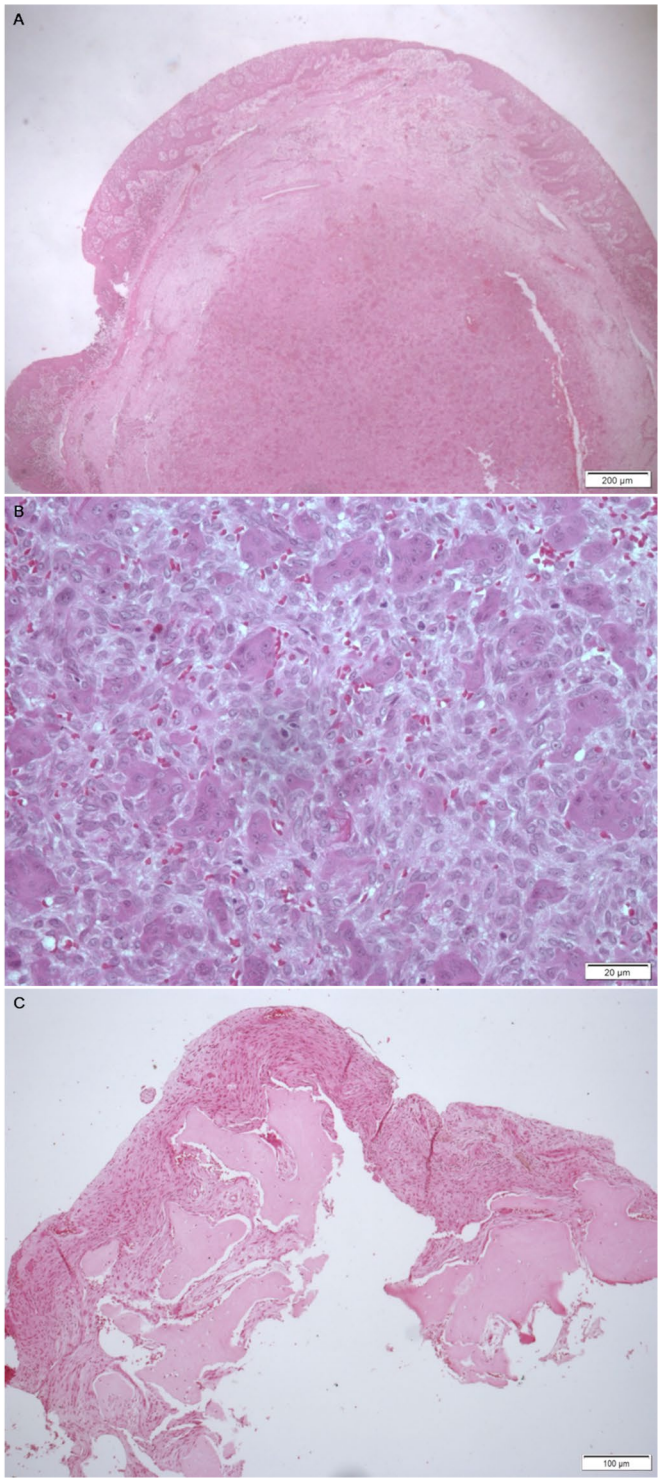

Figure 2. Photomicrographs of excisional biopsy. A) Well circumscribed proliferation recovered by stratified epithelium (HE, 4x). B) Multiple giant cell dispersed in a fibrous connective tissue (HE, 20x). C) Intra-osseous material after curettage: fibrous connective tissue and vital normal bone fragments.

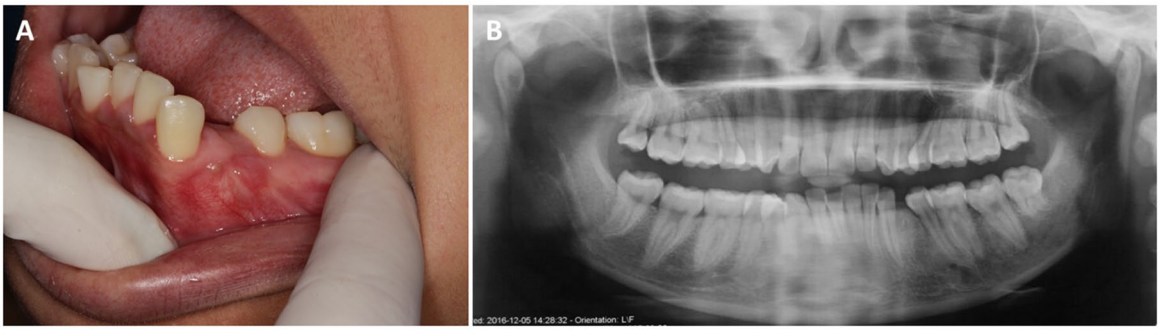

Figure 3. Follow-up after one year. A) Oral mucosa without signals of recurrence. B) Panoramic radiography showing complete bone healing.

hematoma. If the hematoma is connected to a vascular enlargement, blood pressure could result in expansion and ABC development. If the hematoma is supplied by small blood vessels, a CGCL composed of soft tissue and endothelial-lined capillaries, proliferating fibroblasts, and multinucleated giant cells can develop. If the blood supply to the hematoma is interrupted or blocked, it may result in the development of a SBC[9,13].

Moreover, transformations from one lesion to another have already been reported in the literature and appear to occur in several ways. In 2002, Chiba et al. published a case report of SBC that transformed to a CGCL 8 months after intervention[10]. Biesecker et al. reported two cases of ABCs that formed after curettage of a preexisting lesion; one developed after a CGCL and one evolved from a SBC[14]. Therefore, it is appealing to consider that all these pathological entities may be related, as different manifestations of the same disease. These lesions could be caused by an intramedullary hematoma, and ABCs and CGCLs might be formed from the connective tissue as part of hematoma repair; however, a failure of hematoma repair results in a SBC.

On the basis of these findings, we proposed the hypothesis that our patient could have had a CGCL, which perforated the cortical bone and externalized during its progression. Subsequently, the local blood supply would have been compromised, thereby causing the degeneration or involution of the intraosseous lesion and resulting in an empty cavity, which was diagnosed as a SBC (Figure 4). The features that support this hypothesis are the proximity of both lesions, the characteristics of tooth displacement that are uncommon in PGCL and more frequent in CGCL, and previous reports of total spontaneous regression of CGCLs[15].

In conclusion, when a lesion is detected in the gingiva or alveolar ridge, complementary imaging examination is imperative to evaluate bone involvement.

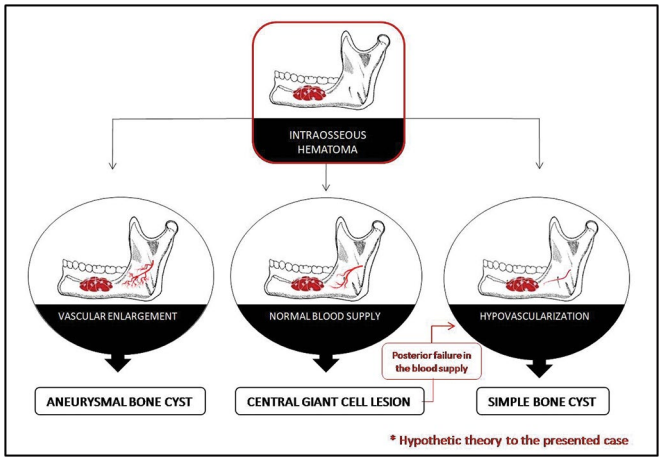

Figure 4. Etiopathogenic correlations between the Simple Bone Cyst (SBC), Aneurysmal Bone Cyst (ABC), and Central Giant Cell Lesion (CGCL). Hypothetical theory to the presented case.

The diagnostic process underlying lesions of the gnathic bones should consider the presence of simultaneous lesions, and the combination of SBC and PGCL should be listed among the possible differential diagnoses.

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