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Central Giant Cell Granuloma: Case Report With Partial Mandibulectomy With Condylar Replacement

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Abstract

Central giant cell granuloma (CGCG) is a benign lesion located within the bone. Although the exact cause is unknown, it is believed to result from trauma, inflammatory processes, or genetic predispositions. This condition is more common in children and young adults, with a higher frequency among women. Symptoms can range from

asymptomatic and slow-growing to more aggressive manifestations, such as rapid bone destruction, cortical bone expansion, thinning and perforation, tooth root resorption, and displacement of adjacent structures, including nerves and teeth, accompanied by pain. In the clinical case in question, the 13-year-old patient M.I.A.S. had melamoderma. She had an expansive lesion on the left side of her mandible, which was growing aggressively but was asymptomatic, resulting in facial asymmetry. The suggested treatment was removal of the lesion. Given the severity of the lesion, it was not possible to preserve the mandibular angle and condyle. During reconstruction, we fixed a custom-made plate to the condyle using surgical resin and adjusted it in the mandibular fossa, followed by layered suturing, finishing with intradermal suturing using 4.0 nylon thread. The patient underwent surgery in a hospital setting under general anesthesia. She is still being monitored and, due to her age, may require further surgery. The goal of surgical treatment for more aggressive central giant cell granulomas is to remove the mass through appropriate surgery and perform the necessary repair with the lowest degree of morbidity and risk of recurrence.

Keywords: *Central Giant Cell Granuloma. Oral and Maxillofacial Pathology. Mandibulectomy and Oral and Maxillofacial Surgery and Traumatology.*

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I. Introduction

Central giant cell granuloma (CGCG) is a benign intraosseous lesion first described by Jafe.1 CGCG is also described as an intraosseous lesion consisting of cellular fibrous tissue containing multiple foci of hemorrhage, aggregations of multinucleated giant cells, and some trabeculae of bone tissue.[2] It is a non-neoplastic lesion found particularly in the maxilla and mandible. Although the etiology is uncertain, it is presumed to be caused by trauma, inflammatory processes, or genetic factors. CGCG occurs primarily in children and young adults, with a female predilection.[3,4,5,6]

The World Health Organization (WHO) defines CGCG as "an intraosseous lesion consisting of cellular fibrous tissue containing multiple foci of hemorrhage, aggregations of multinucleated giant cells (MNGCs), and occasionally, trabeculae of bone tissue."[1,7,8,9]

CGCG represents less than 7% of all benign tumors of the jaw. The mandible is more commonly affected than the maxilla, with a comparative ratio ranging from 2:1 to 11:9.[1,8,9]

It is typically observed in the region of the mandibular body, anterior to the first molar. The most commonly affected age group is under 30 years, with a distinct predilection for women.[8,9]

Clinically, CGCG can behave in a variable manner, exhibiting characteristics ranging from asymptomatic, indolent, and slow-growing to aggressive and rapid bone debulking, with cortical expansion, thinning and perforation, root resorption, and displacement of adjacent structures, including teeth and nerves, accompanied by pain. Perineural invasion and infiltration into adjacent soft tissues are usually not observed, and although the lesion is not encapsulated, it expands, pushing away and displacing adjacent structures rather than invading or infiltrating them. It is associated with a relatively high recurrence rate of 15% to 20%; the more aggressive the lesion, the greater the chances of recurrence.[9]

Imaging plays a crucial role in the diagnosis and characterization of CGCG. Typical findings include expansile, uni- or multiloculated lesions with wavy septations, moderate enhancement, and occasional evidence of cortical perforation and tooth root resorption. On computed tomography (CT), these lesions often present as expansile midline masses in the mandible, exhibiting multiloculated features with septations perpendicular to the cortex. Larger lesions may present with cortical dehiscence and scalloping, highlighting their potential for local bone destruction.[9,10]

Excision by direct curettage of the lesion and resection is considered an indirect approach to the lesion through en bloc surgical resection, including a 5-mm margin of healthy bone tissue as a pathological safety margin. Intralesional injections of corticosteroids, alpha-interferon, and calcitonin are also recommended. Radical surgical intervention in aggressive lesions is associated with low recurrence rates. Even if such a radical procedure is effective, tooth loss will inevitably occur, along with impaired inferior alveolar nerve function. Reconstruction of the resulting surgical defect is extremely important to restore aesthetics and function. [10,11]

This paper discusses the literature on GCCG and reports a clinical case with partial resection of the mandible and immediate production of a condylar prosthesis, fixed to a titanium plate with bone resin.

II. Case Report

Patient M.I.A.S., 13 years old, with melamoderma, presented to the Surgery and Traumatology Clinic of the Specialization Course at UNIFIL – São Paulo, Brazil. He presented with an expansive, aggressively growing, asymptomatic lesion on the left side of the mandible, producing facial asymmetry. (Fig. 1 and 2). Imaging revealed a multilocular lesion, loss of the vestibular cortex, paresthesia, and slightly displaced teeth with

discreet root resorption. (Fig. 3, 4, and 5). Aspiration prior to the biopsy revealed the bloody nature of the lesion, which may contain purulence, multinucleated giant cells, and formed elements such as epithelioid cells, macrophages, and lymphocytes. In some cases, foci of necrosis. Even given the above, fine needle aspiration biopsy is not the first diagnostic approach. A biopsy was performed, with a final diagnosis of GCCG, followed by surgical resection.

Figures 1 and 2. Extraoral and intraoral clinical appearance.

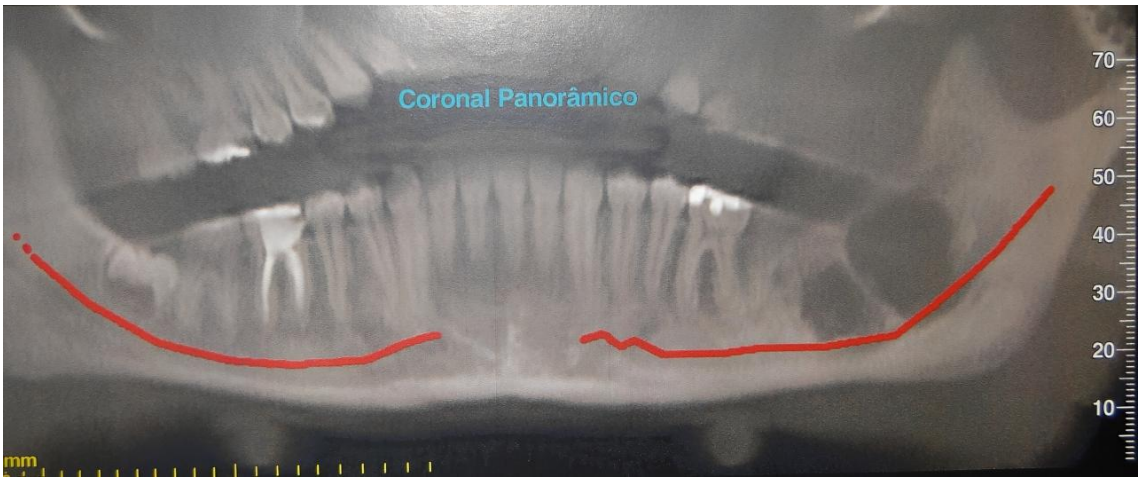


Figure 3. Tomography – Panoramic Coronal Section showing multilocular lesions, rupture of the vestibular cortex and its relationship with the inferior alveolar nerve.

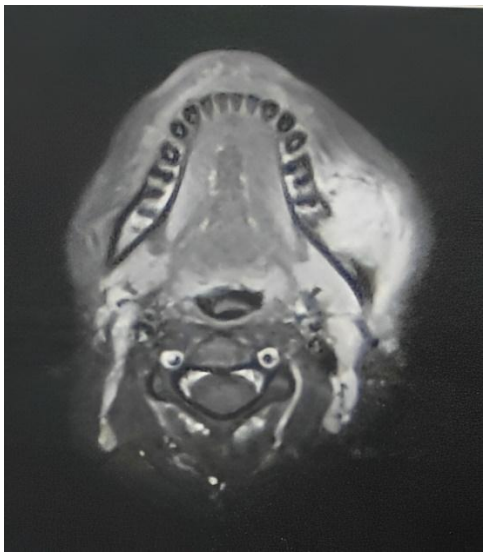


Figure 4. Tomography. Axial section. Showing the expansion of the lesion.

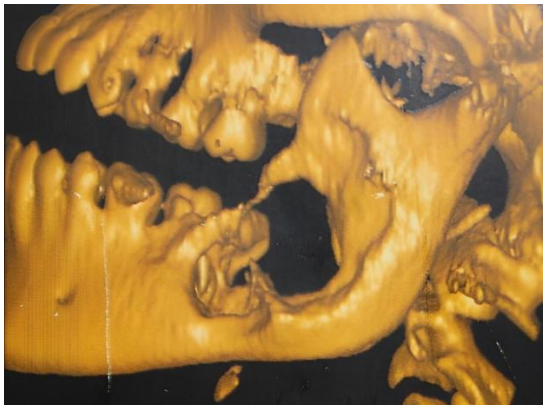


Figure 5. 3D reconstruction of the lesion.

Surgical resection was planned to preserve the condyle. However, the lesion was extremely aggressive, expansive, and beyond the reach of preservation.

The proposed treatment was excision of the lesion. The patient underwent surgery in a hospital setting under general anesthesia. The surgical approach was the Risdon approach, with an incision (marking) 1.5 to 2 cm from the inferior margin of the mandible (however, the large tumor volume required a variation in the approach). Local vasoconstriction was performed with infiltration of 1:140,000 epinephrine into the subcutaneous tissue for local hemostasis. (Fig. 6).

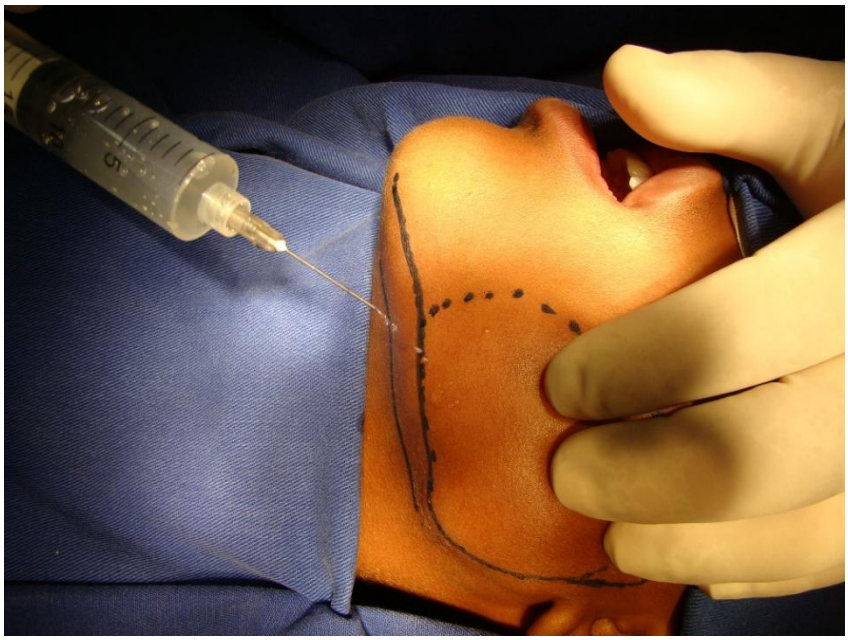


Figure 6. Left submandibular marking (Risdon access) and adrenaline infiltration. Source: the author.

During surgery, the patient underwent an Intermaxillary Block (IMB) with screws and steel wires for maxillomandibular stabilization. The incision was made in layers, followed by tissue divulsion and exposure of the lesion. (Figs. 7, 8 and 9).

Figures 7, 8 and 9: Incision, Divulsion and Exposure of the already resected lesion.

Given the extent of the lesion, preservation of the mandibular angle and condyle was impossible. (Fig. 10). During surgery, we chose to mold the condyle with surgical bone resin, fixing it to the titanium plate, system 2.4. (Fig. 11, 12, 13).

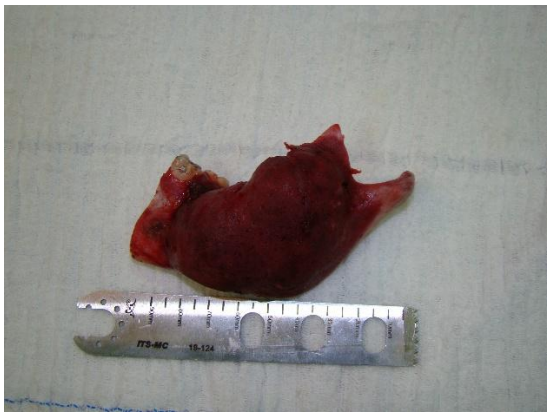
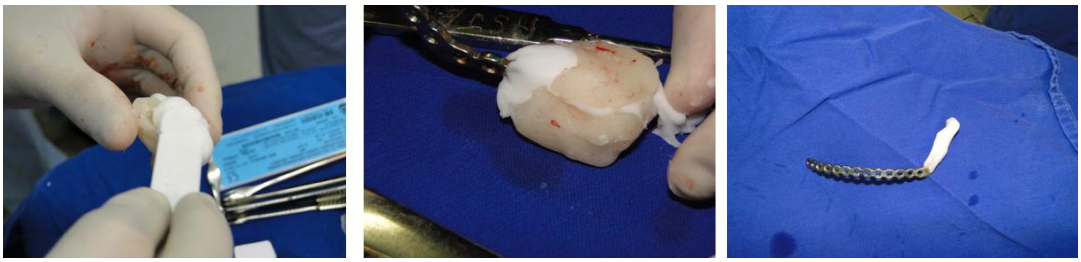


Figure 10. Resected anatomical specimen.



Figures 11, 12 and 13: molding and fabrication of the condylar head in the fixation and reconstruction system.

To complete the reconstruction, we fixed the plate with the prefabricated mandibular head during surgery using surgical resin and adjusted it in the mandibular fossa, suturing it in layers, and finishing with intradermal suturing using 4.0 nylon thread. (Fig. 14, 15 and 16).

Figures 14, 15 and 16. Final sequence of fixation to the intradermal suture.

Thirty days after surgery, we evaluated the patient's facial features from an anteroposterior view, revealing slight asymmetry. The oral cavity was evaluated, and all structures were normal, with no inflammatory processes or plaque exposure. We evaluated mouth opening and closing and laterality, which were within normal limits, considering the aggressiveness of the operated lesion, the complexity of the surgery, and the patient's physiological and age characteristics. Finally, the smile, which motivates and drives us in our work (Fig. 17, 18, 19, 20, and 21).

Figures 17, 18, 19 and 20. Final postoperative. Anterior, Oral Cavity. Opening and Smile.

Figure 21. Control panoramic X-ray, 30 days, showing the adaptation of the condylar prosthesis.

As this is a young patient, monitoring is necessary for subsequent reconstructive surgery, changing the plate and possibly using condylar prostheses (head of the mandible and mandibular fossa).

III. Discussion

GCCG is a benign osteolytic lesion, accounting for almost 7% of all benign tumors of the jaw, mainly in young adults in their second and third decades of life. It has a predilection for the anterior jaw and is more prevalent in females. [1,6,8,12,13]

Radiographically, it presents as a radiolucent area, which may be well-defined, ill-defined, or multilocular, and may involve tooth displacement and/or resorption. It can be classified as aggressive or non-aggressive based on its clinical behavior. [14,15,16]

The distribution of lesions in the jaws can be seen in Fig. 21. In the mandible, most lesions are concentrated in the anterior and left part of the maxilla.[12]

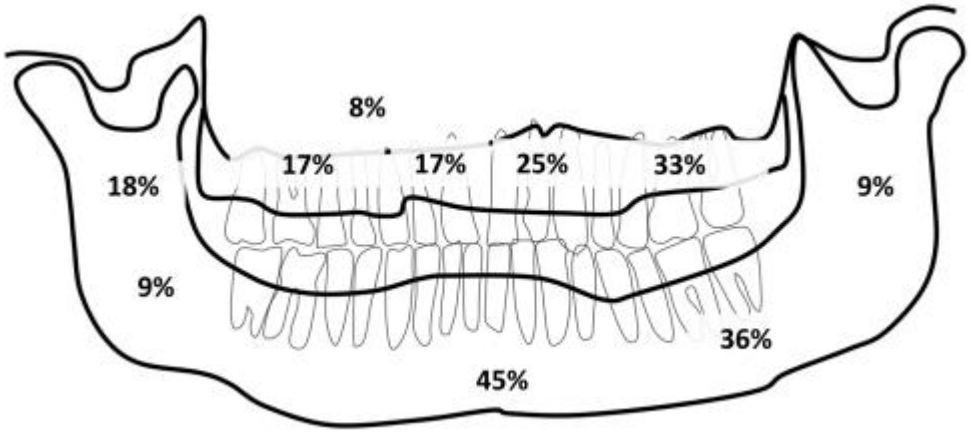


Figure 21. Distribution of central giant cell lesions in the maxillomandibular complex.[12]

The treatment modalities for CGCG are widely debated in the literature, ranging from more conservative drug treatments to surgery. Oral and intralesional medications such as corticosteroid injections, calcitonin spray, denosumab, and IFN-α administration are available. Surgery, however, can be accompanied by lesion recurrence, which, according to current literature, can reach approximately 24%. This treatment modality causes aesthetic and functional impairment, requiring extensive surgical rehabilitation to restore aesthetics and an acceptable quality of life to the patient. [17,18,19,20,21,22,23]

The aggressiveness of giant cell granuloma (GCGC) lesions in the jaw can determine the treatment model. More aggressive lesions, which tend to be larger, resorb tooth roots, disrupt cortical bone, and exhibit rapid growth, generally require more radical treatments, such as en bloc resection or supplemental radiotherapy, while less aggressive lesions can be treated with enucleation, curettage, and oral drug therapy and intralesional injections. [6,17,18,19,20, 21,22,23]

IV. Conclusions

The goal of surgical treatment for aggressive central giant cell granuloma is to remove the mass with appropriate surgery and repair it adequately with the least possible morbidity and risk of recurrence.

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