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Case Report

# Central Giant Cell Granuloma in Maxillary: Treatment and Literature Review

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## Abstract

**Objective:** We report the case of combined surgical-pharmacological treatment with local administration of intralesional corticosteroids (IIC) for the management of aggressive central giant cell granuloma (CGCG-A) in the upper jaw with a 4-year follow up.

**Methodology:** A 2-stage treatment is planned, surgical curettage and intralesional injections with 20 mg/ml triamcinolone diluted in a 2% lidocaine/epinephrine anesthetic solution of 1:200,000 in a 1:1 ratio. Use 1mL of infiltrate solution for every 1 cm3 of radiolucent lesion area applied every 2 weeks, for 6 weeks.

**Discussion:** At 6 months, complete clinical remission and decreased tooth mobility were observed. Radiologically, there were signs of bone apposition and a decrease in the volume of the lesion. At 3 years there was corticalization of the maxillary sinus without invasion, well-defined maxillary cortical and hyperdensity of the trabeculate. Clinically tooth mobility was not observed, healthy mucous membranes and normal eruption of teeth in root formation. Four years later no recurrence is noticed.

**Conclusion:** Combined treatment with local administration of IIC is a conservative alternative for the treatment of CGCG -A and can be considered as a therapeutic option to avoid high morbidity without affecting aesthetic and functionality, loss of teeth, and impair the quality of life of a young patient.

***Keywords:*** *Giant cell granuloma, Intralesional injections, Corticosteroid, Triamcinolone acetonide*

## Introduction

The World Health Organization (WHO)[1] defines central giant cell granuloma (CGCG) as ”an intraosseous lesion consisting of fibrous cellular tissue that contains multiple foci of hemorrhage, the incorporation of multinucleated giant cells (MNGC), and occasionally trabeculae of medullary bone [2]”. Although the term ’reparative giant cell granuloma’ has also been used, most pathologists have not preferred the word ’reparative’ because the lesion is usually destructive and sometimes aggressive [3].

This pathology occurs in a wide age range from 2 to 80 years, although it affects mainly children and young adults between 2-25 years of age with a female predilection. The estimated incidence is 1.1 in 1 million [4]. Seventy percent of cases occur in the mandible, generally in the anterior area of the jaws, crossing the midline [4,5].

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Chuong in 1986 [6] and Kaban in 1999 [7], classify the biological behavior of CGCG into aggressive and non-aggressive (CGCG-N) based on clinical and radiological characteristics, with the latter type being the most common. CGCG-N is distinguished by being small (less than 5cm), asymptomatic, slow growing and does not cause cortical perforation or rhizolysis; these injuries are generally detected in routine examinations. CGCG-A is characterized by pain, rapid growth, cortical perforation, rhizolysis, tooth displacement, and / or paresthesia. Externalization can cause mucosal ulceration. Malocclusions due to tooth displacement are also described [4]. CGCG-A, compared to non-aggressive, tends to have a high recurrence rate [2,4,6].

Radiographically, it is observed as a radiolucent lesion, either unilocular or multilocular, which is usually well delimited, presenting a type Ib or II radiographic pattern. The unilocular form is the most common. These lesions can vary in size from 5 mm to 10 cm and are usually associated with displacement and destruction of adjacent tissue. Depending on the stage or progression of the lesion, it should be suspected and differentiated from other small unilocular lesions such as periapical granulomas or cysts, while multilocular lesions can be confused with ameloblastoma [4,8].

This is why differential diagnoses include ameloblastoma, brown hyper- parathyroid tumor, fibrous dysplasia, cherubism, and aneurysmal bone cyst [4,9,10].

Histologically, CGCG exhibits few to many multinucleated giant cells, in a background of ovoid to spindle-shaped mononuclear stromal cells [4]. This lesion is microscopically indistinguishable from the brown tumor of hyperparathyroidism (TPHPT), which is why it must be ruled out with an appropriate biochemical and endocrinological analysis and evaluation of renal function. High levels of parathyroid hormone and alkaline phosphatase indicate hyperparathyroidism [11].

Currently, treatment is varied due to its still unknown origin, which is why various treatment options are described in the literature. There is talk of an inflammatory origin because giant cells are found in foreign body reaction granulomas and sarcoidosis, which is why intralesional injections of corticosteroids are promoted as treatment [12,13]. It is also described as an endocrine lesion, due to its similarity to TPHPT, which can respond to calcitonin [3,14]. Another hypothesis is developed based on a vascular lesion due to its dense vascularity, treated with interferon, an antiangiogenic agent [3,15]. Although the lesion is named for the abundant number of multinucleated giant cells, neoplastic cells are found primarily in the cellular stroma [14]. These stromal cells recruit and activate multinucleated giant cells, which develop the osteo- clast phenotype [11], which is why resorptive drugs such as denosumab have been tried, which interrupt osteoclast-mediated resorption.

CGCG is a benign neoplasm, exclusive to the jaws, potentially and locally aggressive with high rates of recurrence, so surgical therapeutic conduct will be guided by the aggressiveness, location, extension, involvement of noble structures, systemic condition of the patient, among others. The reported recurrence is greater than 72%, after curettage and surgical resection [16]. Lange reports a recurrence of 49-72% for aggressive lesions [5].

Multiple treatments are proposed in the literature, some agreeing that the treatment of choice for CGCG-A is enbloc surgical resection, which, although it may be effective, loss of teeth or associated tooth germs is inevitable, resulting in malocclusion and possibly damage to the inferior alveolar nerve [5]. In addition to this, reconstruction can lead to large defects that can be a challenge to recover functionality and aesthetics [2].

For CGCG-N, conservative treatments, such as intralesional corticosteroid injections (IIC), have shown favorable success rates and low recurrence between 15 and 35% [3,17,23], however, no cases have been reported in CGCG-A. This study shows the case of a 12-year-old patient diagnosed with CGCG-A with a notable response to combined surgical-pharmacological treatment with intralesional administration and her follow-up over 4 years.

## Case Presentation

A 12-year-old female patient, with no morbid history, was referred to the Maxillofacial Surgery Service (SCMF) in October 2020 due to a maxillary injury that had been on for one month, after being treated extra systemically by puncture. aspiration and localized periodontal treatment, without favorable results.

On extraoral examination, there was no facial asymmetry, edema, or palpable regional lymphadenopathy. In the intraoral examination, an increase in the volume of the left maxillary was observed, asymptomatic, ulcerated, firm consistency and well delimited, with respect to the left posterior-upper teeth from the first premolar to tuberosity, which presented increased mobility.

All teeth were vital to the EndoIce© pupal test. A study is complemented by cone beam computed tomography (CBCT), which shows a non-iloculated osteolytic lesion in continuity with the sinus floor, inflammatory reaction of the maxillary sinus mucosa, and rhizolysis of adjacent teeth. (Fig.1) Laboratory tests were requested, which were within normal ranges (PTH 43.7pg/ml, alkaline phosphatase 222 U / L, calcium 9.3 mg / dL, creatinine 0.52 mg / dL, etc.).

Under local anesthesia, an incisional biopsy was performed, the results of which report a multistratified keratinized squamous epithelium, fibrous connective tissue with abundant islands of calcified material in a stroma with mononuclear inflammatory cells. Focus of multinucleated giant cells is observed, without signs of cellular atypia (Fig. 2). Diagnostic confirmation of central giant cell granuloma. This aggressive lesion was subsequently treated initially with enucleation and surgical curettage under general anesthesia in December 2020.

At 45 days after surgery, intralesional administration of 1 cc of a solution composed of 0.5 cc of triamcinolone acetonide 20 mg/ml and 0.5cc of 2% lidocaine/epinephrine anesthetic solution of 1: 00,000 in a 1:1 ratio begins a protocol of 1 ml of solution for every 1 cm3 of the radiolucent lesion area applied every 2 weeks, for 6 weeks. (Fig. 3)

Periodic clinical and radiographic monitoring was performed. The first month after application, we observed significant clinical changes. Four months later, imaging showed a decrease in the size of the lesion, signs of bone apposition, progress in root development of the associated teeth, and continuity of the maxillary sinus floor. At the beginning of 2022, the patient begins orthodontic treatment because the increased mobility generated in the left maxillary molars generates malocclusion. In clinical control in 2023, complete remission of the lesion was maintained and through panoramic radio- graphic imaging control and CBCT, trabeculate hyperdensity was observed with normal eruption of a third molar in advanced root development. (Fig. 4.)

Four years after the start of treatment, no clinical or imaging recurrence was observed. Clinically, healthy mucosa is observed, without tissue loss, teeth within their arch without occlusion alterations. Radiographically, there is normal bone trabeculate, the maxillary sinus floor is in continuity, and there is no anomaly of dental development. (Fig. 5)

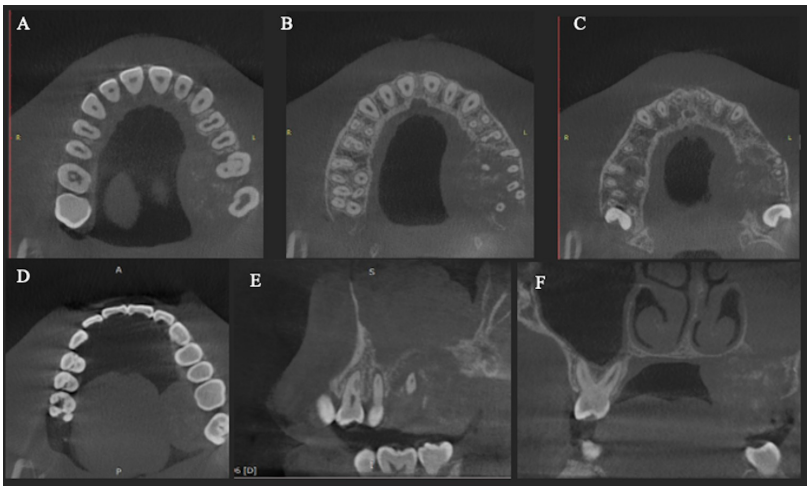


***Fig 1.*** *(A-C) Axial image shows hyperdense areas in relation to left maxillary molars, displacement of dental roots. (D) Increase in palatal volume in relation to maxillary molars. (E) Sagittal section showing mixed lesion. (F) Coronal section shows mixed lesion in relation to the left maxillary sinus, inflammatory reaction of the ipsilateral mucosa.*

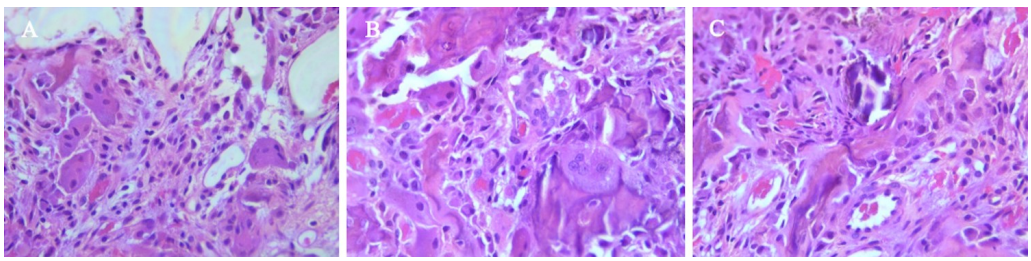


***Fig 2.*** *Histopathological images. [A-C (HE 40x)] Proliferation of giant cells, macrophages and fibroblasts. Extravasated blood and hemosiderin.*

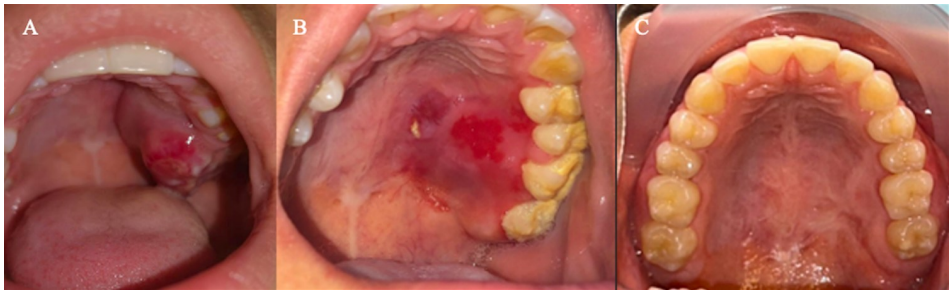


***Fig 3.*** *(A) Left maxillary ulcerated tumor lesion. (B) First administration of intralesional triamcinolone 45 days after surgery. (C) Control year 2024, healthy mucosa, without increase in palatal volume.*

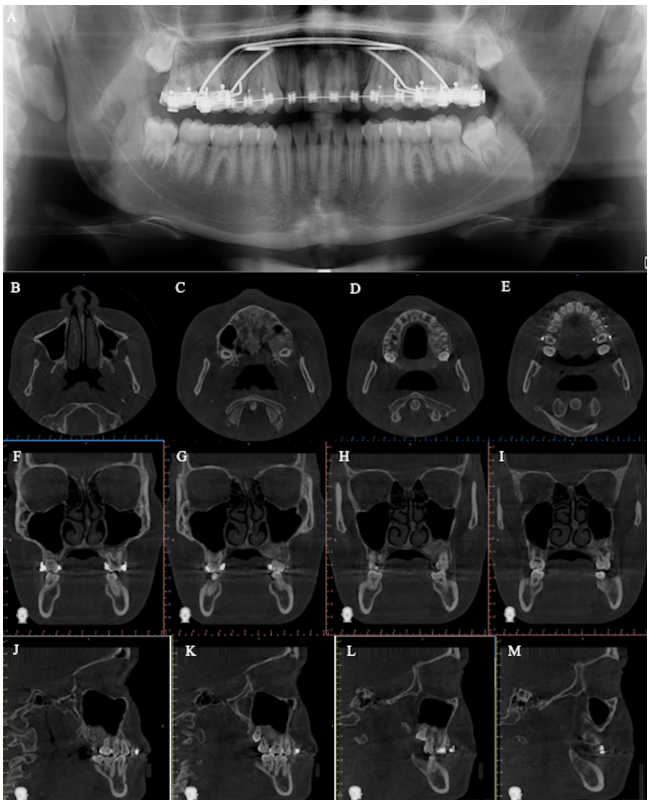


***Fig 4.*** *(A) 2023 panoramic radiograph in orthodontic treatment. Bone tissue without alterations. Maxillary sinus mucosa without alterations. CBCT images. (B-E) Axial section shows hyperdense areas with slight asymmetry in the palate in relation to the opposite side. (F-M) Coronal and sagittal section, left maxillary hyperdense areas, without maxillary sinus alterations.*

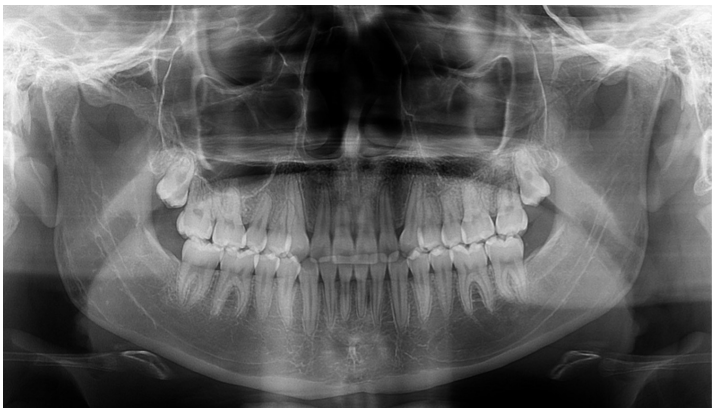

***Fig 5.*** *Panoramic radiograph year 2024, condition remains in remission, same radiographic characteristics as control year 2023. Bone tissue without alterations. Maxillary sinus mucosa without alterations.*

All teeth were vital to the EndoIce© pupal test. A study is complemented by cone beam computed tomography (CBCT), which shows a non-iloculated osteolytic lesion in continuity with the sinus floor, inflammatory reaction of the maxillary sinus mucosa, and rhizolysis of adjacent teeth. (Fig.1) Laboratory tests were requested, which were within normal ranges (PTH 43.7pg/ml, alkaline phosphatase 222 U / L, calcium 9.3 mg / dL, creatinine 0.52 mg / dL, etc.).

## Discussion

Conventional treatment of CGCG involves surgical interventions such as curettage and en bloc resection. Even if it is performed meticulously, recurrence has been reported in 70% of cases when using this technique [6]. In the mentioned case, carrying out an en bloc surgical resection would have resulted in the loss of all the teeth affected by the lesion, as well as a decrease in facial volume. Furthermore, additional surgical interventions would have been required to restore the affected areas, and even then, there would be a persistent risk of recurrence due to the aggressive behavior of this lesion.

This pathology usually presents unilaterally, asymptomatic and expansive. In most cases it occurs in the 3rd decade, with a 2:1 predilection for women. Some more aggressive lesions tend to have a greater recurrence. Due to the similarity with other pathologies such as brown tumor of hyperparathyroidism, Paget's disease, neurofibromatosis, cherubism and Noonan Syndrome, all of these must be ruled out clinically and radiographically, in addition to including the following complementary laboratory tests for calcium and parathyroid hormone [6,18]. The aggressive form of CGCG, presenting high recurrence, is generally treated with surgical procedures, which leads to a lack of aesthetics and functionality, there is damage to the dental follicles and bone loss, frequent complications in young patients [5,19,20].

A search conducted in English revealed 13 case reports [2,9,9,21,22,24-31] ,and 4 case series[3,13,17,23] which used intralesional corticosteroids for the treatment of CGCG (Table 1). Twelve of these cases resorted to combined treatment (IIC-surgical) for aggressive GCCG with resolution in follow-up of up to 8 years. Two treatment failures were reported, of which calcitonin was used in nasal and intralesional spray [26,31], in which the treatment was changed to triamcinolone acetonide, obtaining favorable results and no recurrence in a 3-year follow-up.

However, it appears that the success rate is high in combination treatments or with triamcinolone acetonide alone [8,21,22,29].

Various authors in recent years have used triamcinolone acetonide, a synthetic corticosteroid 8 times more powerful than prednisone [31]. It is a simple technique therapy, low cost, fast acting and prevents both aesthetic and functional unwanted effects [3].

Hirayama et al [33] reported that dexamethasone, a glucocorticoid, has a direct effect on inhibiting bone resorption due to the activity of mature osteoclasts. On the basis of experimental evidence, it is possible to hypothesize that the effect of intralesional injections with corticosteroids on CGCG is produced by the inhibition of extracellular production of lysosomal proteases and steroidal apoptosis of osteoclast-like cells [3,17].

According to the literature, intralesional injections are effective as a treatment for CGCG, however, a positive result cannot always be obtained in multilocular or aggressive lesions [20]. For this reason, it was decided to apply combined ICC-surgical treatment.

Usually, the aggressive treatment of CGCG is en bloc resection, which has low recurrence, but requires greater reconstruction. In our presented case of an aggressive CGCG, the management was a presurgical combination of osteotomy and curettage with subsequent application of intralesional corticosteroid. En bloc resection was avoided, developing teeth were preserved and, above all, the quality of life of the patient who did not have to undergo major interventions was maintained thanks to this therapeutic alternative. After a 4-year clinical and radiographic follow-up, there are no signs of recurrence.

These results are consistent with the literature, since cases treated with IIC and surgical curettage have obtained successful treatments of up to 5 years without recurrence [3,14,23,24,26,27], without subjecting the patient to major surgeries.

**Table 1.** Cases reported in English. Studies of intralesional corticosteroids for the treatment of central giant cell granuloma.

| Author, Year | Age, Sex | Location | Tipe of treatment | Results | Follow up | Complementary treatment |
|---|---|---|---|---|---|---|
| **Kermer et al. 1994** | 40M | Mandibular | TA+lidocaine | Resolved | 3 years | |
| **Rajeevan et al. 1998** | 17F | Mandibular | TA+lidocaine | Resolved | 10 months | |
| **Marshall et al. 2001** | 10F | Mandibular | TA+lidocaine | Resolved | 1 year | IIC+surgery |
| **Crestanello et al. 2004** | 46F | Mandibular | TA+lidocaine | Resolved | 3 years | IIC+surgery |
| | 38F | Maxillary | TA+lidocaine | Resolved | 2 years | IIC+surgery |
| | 22F | Maxillary | TA+lidocaine | Resolved | 2 years | IIC+surgery |
| **Comert et al. 2006** | 4M | Maxillary | Predniso-ne+lidocaine | Resolved | 3 years | IIC+surgery |
| **Muñoz et al. 2010** | 13F | Maxillary | TA+lidocaine | Resolved | | IIC+surgery |
| **Schütz et al. 2010** | 11M | Mandibular | Calcitonin | Failure | | |
| | | | TA+lidocaine | Resolved | | IIC+surgery |
| **Nogueira et al. 2010** | 5-25 (11M.10 M) | Maxillary (8), Mandibular (13) | TA+lidocaine | Resolved | 3-8 years | 2 surgery, 4 IIC+surgery |
| **Ferretti et al. 2011** | 16F | Mandibular | TA+lidocaine | Resolved | 1 years | |
| **Rachmiel et al. 2011** | 24F | Mandibular | TA+lidocaine+Calcito nin | Resolved | 5 years | IIC+surgery |
| **da Silva et al. 2013** | 9M | Mandibular | Calcitonin | Failure | | |
| | | | TA+lidocaine | Resolved | 3 years | |
| | | | | Resolved | 5 years | |
| **Dolanmaz et al. 2015** | 11-48 (4M-3F) | Mandibular (4), Maxillary (3) | TA+lidocaine | Resolved | 5 years | 3 IIC+surgery |
| **de Mendonça et al. 2019** | 12F | Maxillary | TA+lidocaine | | | |
| | | | Aledronate | Resolved | 3 1/2 years , 6 years | |
| **Nilesh et al. 2020** | 27F | Mandibular | TA+lidocaine | Resolved | 10 years | |
| **Nogueira et al. 2020** | 11 (4M-7F) | Maxillary (3) Mandibula (8) | corticoesteroid (2), denosumab (2), com-bined (7) | Resolved (8) | 1-6 years | |
| **Kumar et al. 2022** | 22F | Mandibular | TA+lidocaine | Resolved | 2 years | |
| **Joshi et al. 2023** | 11F | Mandibular | TA+lidocaine | | 6 months | |

## Conclusion

The combined treatment of IIC and curettage is advantageous for large aggressive lesions to reduce the size of the lesion and minimize the need for bone resection which could result in aesthetic and functional defects, loss of teeth and impair the quality of life of a young patient.

## Ethical Approval

Yes, ethical approval was obtained.

## Consent

Consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

## Conflict of Interest

None declared.

## Acknowledgment

None.

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