



Case Report

Surgical Management and Rehabilitation of Recurrent Central Giant Cells Granuloma (CGCG) of the Jaw: A Case Report

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Introduction

Central giant cell granuloma (CGCG) is an uncommon, benign but locally aggressive osteolytic lesion, which generally occurs in the craniofacial region, especially in jawbones. [1] It was firstly described by Jaffe in 1953 as a giant-cell reparative granuloma of the jawbones, resulting from a local reparative reaction, [2,3] suggesting an association between previous trauma and GCGC formation. World Health Organization (WHO) has defined GCGC as "*a localized benign but sometimes aggressive, osteolytic proliferation consisting of fibrous tissue with haemorrhage and hemosiderin deposits and presence of osteoclast-like giant cell with reactive bone formation*" [4].

GCGC accounts for <7% of all benign tumour of the jaw, affecting most frequently the mandible than the maxilla, especially occurring in the body and anterior portions of the mandible [5].

Usually, GCGC affects younger patients, with a predilection for females, generally between the 2nd and the 3rd decades of life [1].

Radiographically, GCGC may appear as small unilocular radiolucent area or larger multilocular radiolucency, with scalloped

margins and a honeycomb or soap bubble-like appearance; [6,7] occasionally, displacement of teeth and tooth germs, root resorption and cortical perforation are observed [8].

Clinically, GCGC can exhibit behaviour ranging from asymptomatic, indolent and slow growth to aggressive and rapid bone loss, with pain and sensory alterations. Currently, clinical signs and symptoms and radiological findings are the main criteria for distinguishing aggressive forms from non-aggressive ones [9].

Histologically, GCGC is characterized by fibroblast-like cells and osteoclast-like giant cells immersed in a fibrous connective tissue stroma containing cavernous or sinusoidal blood filled spaces [10].

Aetiology of GCGC is controversial: it has been suggested that it might be an inflammatory or reactive lesion, an endocrine lesion or a true tumour. Anyway, it seems that alteration in the haemostatic-vascular equilibrium of bone is the most recognised aetiological mechanism. Moreover, GCGC can be associated with genetic conditions, such as Noonan syndrome, neurofibromatosis type I and cherubism, often presenting as a multiple lesions [11,12].

First line treatment is surgical excision, ranging from simple curettage to en-bloc resection, basing on the extent of the lesion. However, surgical treatment is associated with varying degrees of morbidity, ranging from tooth loss to injury of the inferior alveolar nerve. Moreover, aggressive lesions require major surgical resection, with aesthetic and functional alteration [8,11,13].

After surgery, reported recurrence rate range from 11% to 49% and up to 72%, for the aggressive forms [7].

Therefore, considering the difficult management of extensive lesions, especially in young patients, in the past decades, a variety of non-surgical treatments have been proposed: intralesional corticosteroids, subcutaneous or nasal calcitonin, subcutaneous interferon alpha and, most recently, imatinib and denosumab [14,15].

We present a case of a 8-years-old patient with diagnosis of GCGC and a history of multiple recurrences, treated with surgical

resection of the lesion and curettage of residual cavity, followed by use of a distractor for prosthetic rehabilitation.

Case Report

An 8-year-old Italian male presented in August 2000 with an epulis in the region of the inferior vestibular fornix. Radiological imaging revealed a concomitant osteolytic lesion in the left mandibular parasymphiseal region in correspondence of the deciduous teeth 7.1, 7.2 and 7.3.

Extra-oral physical examination showed no mandibular asymmetries or deformities. The patient was asymptomatic.

In September 2000 dental elements were removed and a curettage of the residual bone cavity was performed.

Histological examination of the surgical specimen was consistent with Giant Cell Granuloma (GCGC) (Figure 1).

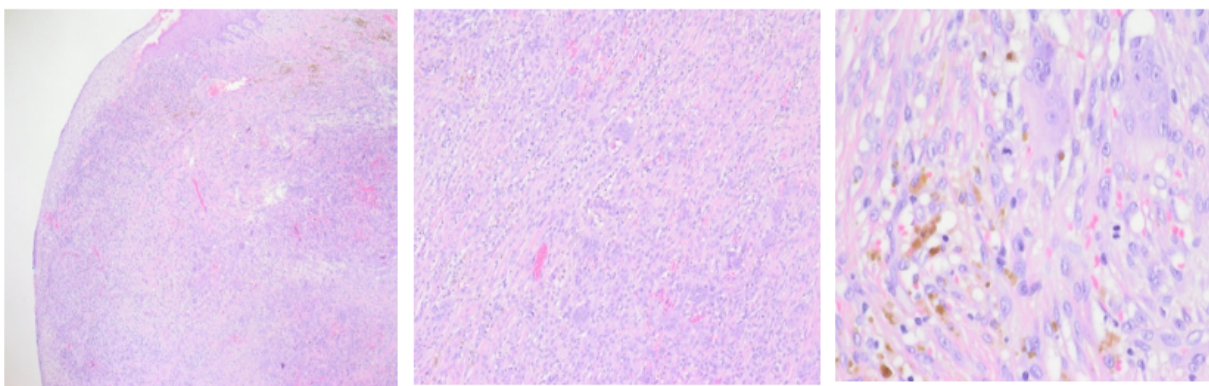


Figure 1: Histological features.

However, over the years, the patient underwent numerous relapses, always treated by surgical removing of the lesion and curettage of the residual cavity (Figures 2, 3).

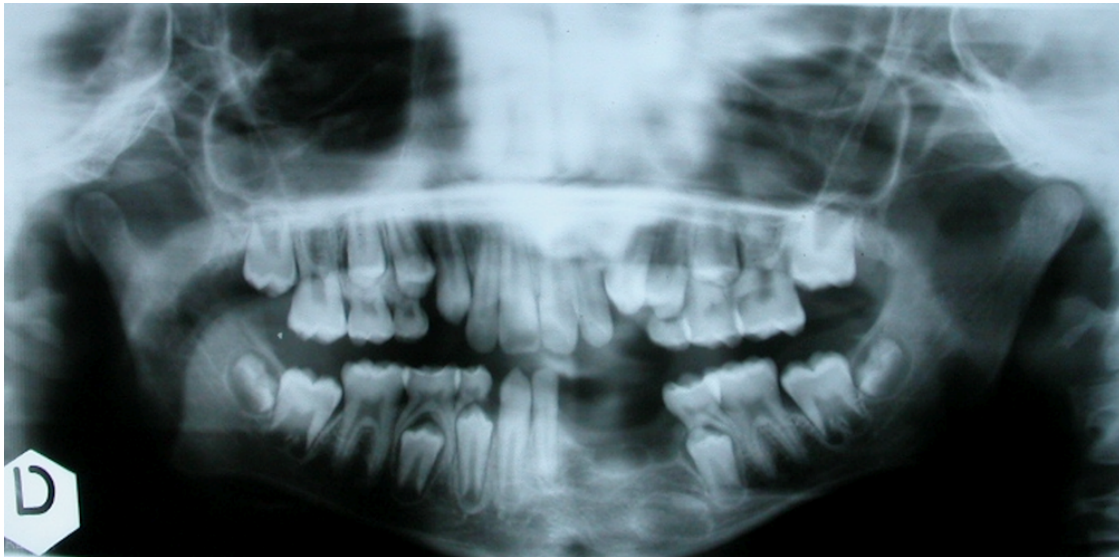


Figure 2: Radiographic image of the relapse after surgical treatment.



Figure 3: Oral cavity after XX surgical treatment.

After a 3 years relapse-free follow-up, considering the patient’s young age, mandibular reconstruction was performed to prevent further tissue loss. Reconstruction of the lower jaw was undertaken due to severe atrophy caused by prior surgical treatments.

Surgical access was achieved through an incision in the inferior vestibular fornix (from region 3.1 to 3.6), followed by the elevation of a trapezoidal mucoperiosteal flap. Transposition of the inferior alveolar nerve was carried out, after identification of the same, by means of piezo-surgery 0.5 mm inferiorly and 1 cm posteriorly. Trapezoidal mandibular osteotomies, with a major base superiorly, were performed by piezo surgery and a vertical bone distractor was applied in the vestibular region with a transmucosal activation screw (Figures 4-7). It was sutured after the application of a platelet rich fibrine (PRF) membrane.

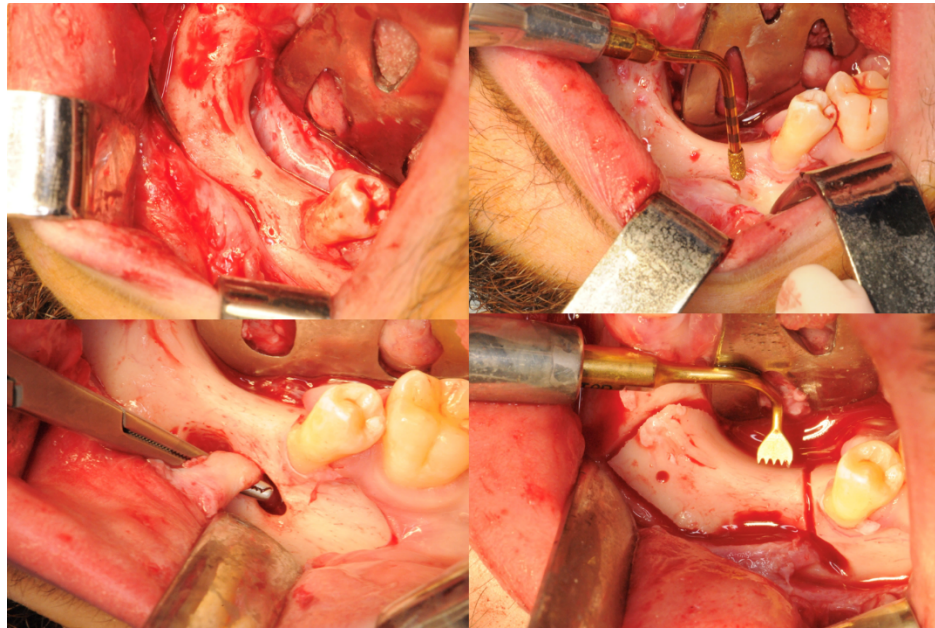


Figure 4: Intraoperative images.

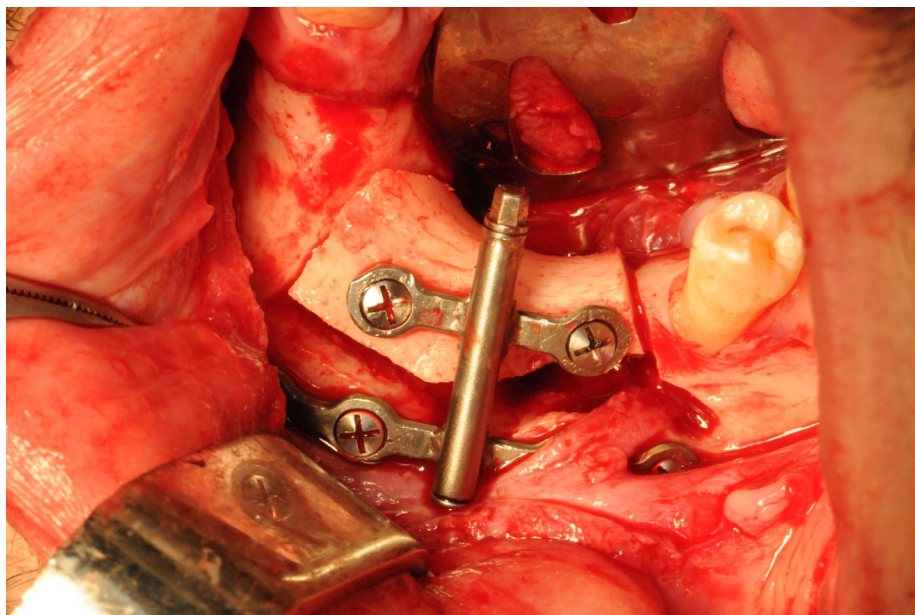


Figure 5: Vertical bone distractor applied after osteotomies.



Figure 6: 3D Image of a vertical bone distractor applied after osteotomies.

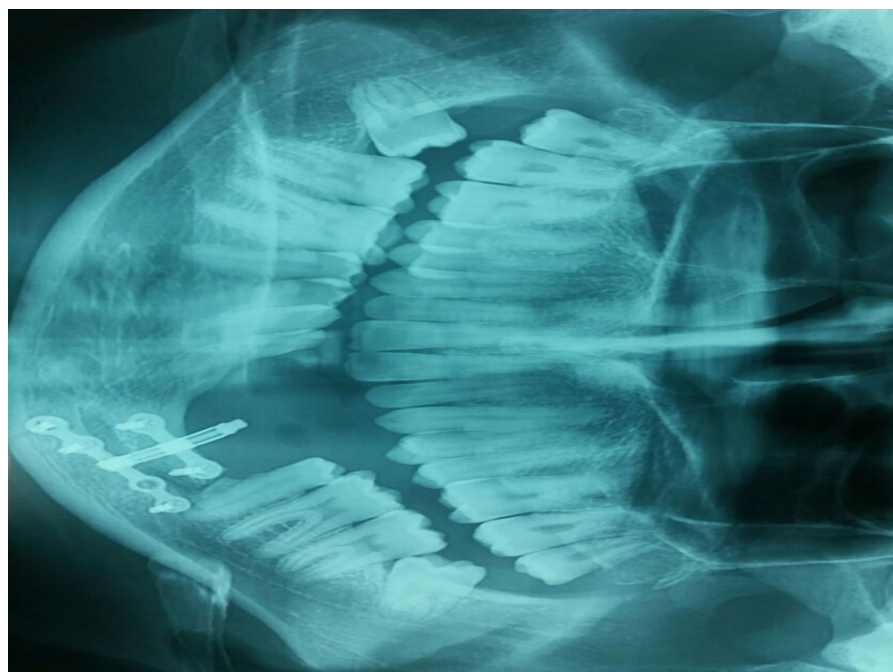


Figure 7: Radiographic image of the vertical bone distractor applied after osteotomies.

Over the first post-operative month, the distractor was activated at a rate of 0.5 mm per day, achieving a total distraction of 15 mm. The Consolidation phase for the newly formed bone occurred during the second month. At the beginning of the third month, the bone distractor was removed and the prosthetic implant rehabilitation was carried out using 3 titanium implants with the aid of bone PRF. Three DENTSPLY xive fixture diameter implant (fixture diameter 3.8 mm, length 13 mm) were placed within the distracted segment. After 3 months healings abutment were used to obtain a transmucosal healing. One month later impression was carried out by a customized tray with polyether impressions material. A prettau bridge was performed with cad cam techniques, the middle structure reproduces the pink aesthetic simulating soft tissue loss, and a cemented metal ceramic bridge replay miss teetees achieving an outcome satisfying for our young patient (Figures 7-9).



Figure 8: Prosthetic implant rehabilitation.



Figure 9: Prettau bridge applied on implants' site.

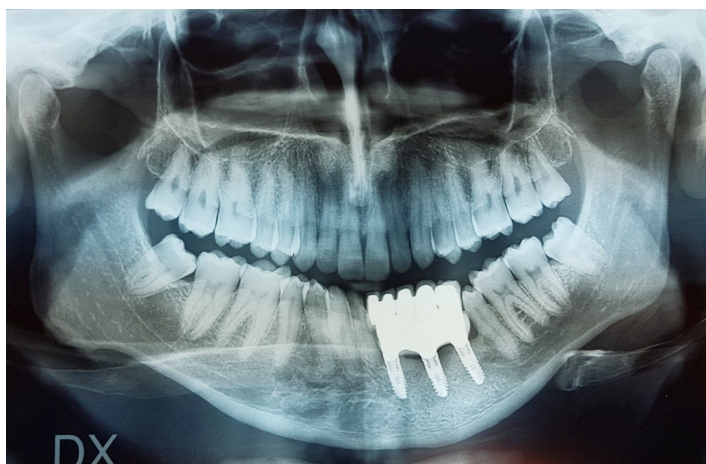


Figure 10: Radiographic image of the final rehabilitation.

Discussion

GCGC, originally termed as “*giant cell reparative granuloma*” by Jaffe, is a rare, benign but locally aggressive osteolytic lesion, which generally affects the jawbones. [2] WHO defined GCGC as “an intraosseous lesion consisting of cellular fibrous tissue that contains multiple foci of haemorrhage, aggregations of multinucleated giant cells, and occasionally trabeculae of woven bone”, classifying it as non-odontogenic tumour [4].

GCGC accounts for approximately 7% of all benign tumours of jawbones, [16] affecting predominantly young patients under 30 years of age, with 80% of cases occurring before 20 years of age. Females are usually more affected than males, with a ratio of 2:1, even if some authors reported a clear female predilection of 62% [6,17].

According to literature, it has been reported that more than 70% of GCGC develops in mandible and less of 30% in maxilla, generally affecting the anterior region of both bones [18-20].

Radiographically, GCGC shows different features, varying from unilocular to multilocular radiolucent areas with well- or ill-defined borders. As reported by Tosco et al., there is a strong prevalence of multilocular, radiolucent and perforating lesions, with an involvement of cortical bone [11,16].

Basing on clinical and radiological features, two entities can be distinguished: non-aggressive and aggressive forms. According to Chuong et al., six criteria can be used to recognize a lesion as aggressive or not: pain, rate of growth, swelling, tooth root resorption, cortical perforation and recurrences [9].

Clinically, less aggressive forms are characterized by indolent, asymptomatic slow growth, while aggressive lesions are

associated with pain, cortical bone disruption, root resorption and displacement of teeth; when located in maxilla, GCGC can invade the floor of maxillary sinus, the orbit or the nasal fossa [11,21].

Rate of recurrence varies from 13-22% to 49%, even if some authors report values of up to 70% for the aggressive types [5].

Aetiology of GCGC is still controversial. The clinical behaviour of these lesions excludes the hypothesis of a reparative process, as initially suggested by Jaffe, and it’s not considered an odontogenic lesion. Several authors have been suggested GCGC might be an inflammatory lesions, a reactive lesion, an endocrine lesion or a true tumour. Hillerup and Hjorting-Hansen proposed alteration in the haemostatic-vascular equilibrium of bone as possible aetiological factor [22,23].

Histologically, GCGC is characterized by numerous multinucleated giant cells (MGCs) in a prominent fibrous stroma; foci of haemorrhage with liberation of hemosiderin pigment and newly formed bone are evidenced. GCGC shows histological similarities with giant cell tumour of bone (GCTB), both being characterized by mononucleated, spindle-shaped neoplastic cells and over-express receptor activator of nuclear factor κ B-ligand (RANKL). However, GCTB typically affect metaphysis of long bones and may metastasise or undergo malignant transformation in 1.8% of cases. [5,24,25].

The differential diagnosis must exclude other conditions, such as cherubism, an autosomal dominant disorder characterized by bilateral expansion of the jawbones, brown tumours of hyperparathyroidism and multilocular cystic lesion of the jaws. Moreover, GCGC can be associated with genetic syndromes, such as Noonan syndrome and Neurofibromatosis type 1 [11,12].

Furthermore, the mononuclear cell population in GCGCs shows proliferative activity, indicated by the expression of cell cycle protein Ki-67. It also has been demonstrated that giant cells in GCGC express calcitonin receptors [26].

The management of GCGC remains debated. First line treatment is represented by surgical excision, whose extension depends on various factors, such as aggressive versus non-aggressive behaviour, size, location and radiographic appearance of the lesion. Several authors proposed excision via curettage with or without peripheral osteotomy; however, this choice seems not to be the safest treatment for GCGCs, especially in aggressive type. On the other hand, en-bloc resection, although it is the most effective treatment and has the lowest recurrence rate, is not always suitable for young patients and can result in deformities, nerve injuries and dental loss [8].

Therefore, in recent years, alternative non-surgical treatments have been proposed: calcitonin, intralesional corticosteroid, subcutaneous interferon and denosumab. Particularly, a-IFN seems capable to stop rapid growth of the lesions, thanks to its role as a mediator in increasing bone apposition. However, additional surgery remains necessary to remove lesion: in fact, literature reports one case of complete remission of GCGC with a-IFN exclusive therapy [8,22].

Giant cells of GCGC express receptor activator of nuclear factor-kappa B (RANK) and stromal cells express its ligand (RANKL), promoting the maturation of osteoclast-like giant cells and potentiating the osteolytic process of this lesion. Denosumab, a monoclonal antibody that bind RANKL, has reported to prevent tumour progression, reform mineralised bone and reduce tumour size in adults and skeletally mature adolescents with GCGC. On the other hand, possible side effects of treatment with Denosumab, especially in children before skeletal maturity, include hypocalcaemia, hypophosphatemia, growth impairment and osteonecrosis of the jaw [14, 27-29].

In our case, due to patient’s young age and the history of multiple relapses, a radical surgical treatment was preferred, followed by a bone distraction protocol. This approach successfully achieved adequate bone height and soft tissue adaptation, enabling subsequent implant-prosthetic rehabilitation.

Conclusion

The treatment and management of GCGC remains a challenge. Although alternative non-surgical treatments are now well established, the treatment of choice remains a surgical approach.

In our young patient with a history of multiple relapses and surgical treatment with subsequential soft tissue loss, the use of a bone distractor to treat mandibular atrophy has proven to be a valuable option: it allowed the soft tissues to gradually adapt to the expansion. bone, resulting in an excellent implant-prosthetic rehabilitation and a high quality of life (QoL).

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