http://dx.doi.org/10.5272/jimab.2013194.407

ISSN: 1312-773X (Online)

Journal of IMAB - *Annual Proceeding (Scientific Papers)* 2013, vol. 19, issue 4

SURGICAL TREATMENT AND RECONSTRUCTION FOR CENTRAL GIANT CELL GRANULOMA OF MANDIBLE- case report and literature review

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ABSTRACT:

Introduction: Central giant cell granuloma (CGCG) is a benign aggressive destructive osteolytic lesion of osteoclastic origin. The central giant cell granuloma is often found in the mandible, anterior to the first molars. It most commonly occurs in patients under the age of 30, with a clear female prevalence

Purpose: To present a case of CGCG of the lower jaw in Department of Oral and maxillofacial surgery, University Hospital 'St Anna'. Although en bloc resection provides the lowest recurrence rate, only a few single case reports describe the use of this technique followed by reconstruction with autogenous bone grafts.

Material and methods: The medical history of a 28 years patient with a large central giant cell granuloma in the mandible. Biopsy specimen taken from the lesion showed CGCG followed by curettage with peripheral ostectomy with preservation of the continuity of the mandible.

Result: At the 1-year clinical and radiological follow up there was no sign of recurrence.

Conclusion: After complete healing of the graft, prosthetic rehabilitation with implants will be perfomed. This allows the best functional and aesthetic results.

Key words: central giant cell granuloma, autogenous bone grafts, reconstruction

INTRODUCTION:

The central giant-cell reparative granuloma(CGCRG) has been defined as a localized benign but sometimes aggressive osteolytic proliferation consisting of fibrous tissue with hemorrhage and hemosiderin deposits, presence of osteoclast-like giant cells and reactive bone formation. CGCRG has been first described by Jaffe in 1953 [5] and accounts for approximately 7% of all benign tumours of the jaws.[6] It is usually appears as solitary, multilocular, radiolucencies, located in the mandible (anterior to the first molars) and maxilla. It occurs at least twice as often in the mandible than in the maxilla. CGCRG most commonly occurs in patients under the age of 30, with a clear female prevalence.[6] The aetiology of giant cell granuloma is undefined; some describe it as an inflammatory proliferation, some lesions behave as a neoplastic process in an aggressive fashion. Jaffe considered this tumour as a locally reparative reaction of the bone due to inflammation, local trauma or haemorrhage. [5] In the literature there is little evidence of any local reparative process.

The clinical behaviour of CGCG ranges from a slow growing asymptomatic swelling to an aggressive lesion that presents pain, local bone destruction, root resorption or tooth displacement .Currently, clinical signs and symptoms, radiological features and histological features are the main criteria to differentiate between non-aggressive (indolent) and aggressive lesions.

Aggressive lesions are characterized by one or more of the following features: pain, paraesthesia, root resorption, rapid growth, cortical perforation, and a high recurrence rate after surgical curettage- between 37.5% and 70% [4, 7, 10] and are mostly found in younger patients [2] are larger (over 5 cm)[1]. These aggressive type or recurrent lesions require wide en-bloc resection that leads to major defects in the jaws that can alter the facial contours [2, 3, 8] and necessitate major reconstruction. Some surgeons use autogenous bone grafts or vascularized fibula free flap for reconstruction of extensive CGCG.[3, 8] Histologically there is no strict criterion to differentiate between aggressive and non-aggressive lesions, however the number and volume of giant cells versus other components of the lesion might give an indication on its clinical behaviour.[1, 2, 6, 10]

Non-aggressive lesions are usually slow growing, symptom free and the treatment includes conservative surgical procedures.

Although the majority of cases were asymptomatic, the most common feature was a painless smooth swelling in the face or in the oral cavity. The lesion does not invade the perineural sheets so paresthesia is not usually observed in these patients. The other symptoms and signs are facial asymmetry, impaired nasal breathing, loosing or displacement of teeth, and pathologic fracture.[6, 9]

CGCRGs usually present as an expansile radiolucency (87.5%) in X-ray films, but radiologic features vary from ill-defined destructive lesions to a well-defined, multilocular or

unilocular appearance, with root resorption in 13.5% of the lesions and displacement of teeth in 18.0%.

Histologically, multinucleated giant cells, in a cellular vascular stroma, and often-new bone formation are demonstrated. The osteoclast-like giant cells have a patchy distribution and are usually associated with areas of hemorrhage. Ultrastructurally, the proliferating cells include spindle- shaped fibroblasts, myofibroblasts, and inflammatory mononuclear cells.[2, 6]

Differential diagnosis should be considered: Aneurysmal bone cyst, chondroblastoma, osteoblastoclastoma, ameloblastoma, fibroma non-ossificans, hyperparathyroidism, odontogenic cysts etc.

The treatment of CGCG of the jaws is performed according to the following factors: aggressive versus non-aggressive behaviour, location, size and radiographic appearance. Surgical options range from large (en bloc resection) to more conservative approaches (curettage).

The traditional therapy of CGCRG has been local curettage(this has been associated with a high success rate-80%), peripheral osteotomy, excision if needed reconstruction by using an autologous bone graft.[2, 6]

Surgical treatment of CGCRGs can be associated with recurrence and serious facial mutilation and loss of teeth and tooth germs. To avoid such disadvantages, a number of alternative nonsurgical herapies including interferon alpha-2a, calcitonin and intralesional corticosteroid injection have been advocated for the management of CGCRG . Non-surgical treatment of CGCRG is probably a good treatment option for small slowly enlarging lesions. Successful treatment of painful, large, and rapidly growing lesions is more likely achieved by surgical removal. In the literature, recurrence rates vary between 11% and 35%.[6]

CASE REPORT

We present 26 years old male with histopathologic examination of the lesion reported as 'giant cell reparative granuloma' of the mandible. On clinical examination the patient was without subjective complaints. Biopsy specimen taken from the lesion showed CGCG.

Radiografic imaging showed radiolucent lesion on right side of lower jaw, well- defined, with resorption of roots of tooth 46, unilocular in appearance (Fig. 1). The patient was operated under general anaesthesia. The tumour mass was removed through an intraoral approach and curettage with peripheral ostectomy with preservation of the continuity of the mandible with at least a 5 mm margin was performed. Teeth 45 and 47 were extracted. The inferior alveolar nerve was preserved. Immediate reconstruction was carried out for this case with with autogenous bone graft from mental region (Fig. 2). The bone graft was fixed with four screws. (Fig. 3) No complication was observed in terms of loss of teeth, wound dehiscence, infection of the surgical site, graft incorporation, fracture or loss of plates and screws and necrosis of bone segments. Prosthetic rehabilitation with implants will be perfomed.

RESULT:

At the 1-year clinical and radiological follow up there was no sign of recurrence. The postoperative defect is fully reconstructed. (Fig. 4)

DISCUSSION:

The true nature of CGCG remains speculative and considerable controversy exists in the literature. Normally,

it is not considered an odontogenic lesion.[8] It has been suggested that it might be an inflammatory lesion, a reactive lesion, a true tumour, or an endocrine lesion.[5, 8] One hypothesis suggests that CGCG belongs to the spectrum of mesenchymal proliferative vascular primary jaw lesions.[8] CGCG occurs predominantly in children or young adults, with approximately 75% of cases presenting before 30 years of age(our patient is 26 years of age), however it really can occur at any age. Females are affected more frequently than males, with a ratio of 2:1 [8] and more than 70% of CGCGs occur in the mandible and less than 30% in the maxilla with a preference for the anterior portions of both bones.[8] Due to the special anatomical characteristics of the maxilla, presentation, diagnosis, progress, management, and prognosis of maxillary CGCG are different from that of mandibular lesions: the cancellous nature of the maxilla and its thin cortical plates allow the lesion to expand much earlier than in the mandible.[8] The radiographic features of maxillary CGCGs are variable and may be confused with those of other lesions. They have been described as ranging from a unilocular to a multilocular radiolucent appearance with well- or illdefined borders.[10]

In the present case of CGCGs, there was a multilocular, radiolucent non perforating lesion which do not involved the cortical bone. Different authors [10] have classified CGCG into two types, based on clinical and radiographic features. The first is non-aggressive CGCG, which is characterized by a slow, almost asymptomatic growth that does not perforate the cortical bone or induce root resorption and has a low tendency to recur. The second is aggressive CGCG, which is characterized by pain, rapid growth, expansion, and perforation of the cortical bone, radicular resorption and a high tendency to recur. The aggressive lesions are mostly found in younger patients.[2] Aggressive lesions were also larger in size and from the histological point of view they showed a larger fractional surface area occupied by giant cells. In aggressive lesions also can be found a higher number of giant cells. The most reliable factors which relate to an increased risk of recurrence include clinical activity of lesions (72% of recurrence in the aggressive forms, 3% of recurrence in the non-aggressive forms), young age, presence of perforation of cortical bone and tumour size.[8]

CONCLUSION:

Currently, no biological markers are known to predict clinical behaviour, and standard histological techniques are not helpful for the clinician to determine the prognosis. Surgery has always been considered to be the traditional treatment and it is still the most accepted. If soft tissues and periosteum are preserved, and only the bony component is excised, then it is possible to reconstruct the surgical defect with autogenous bone grafts. By doing this bone continuity is maintained and prosthetic rehabilitation via implants can be safely performed.

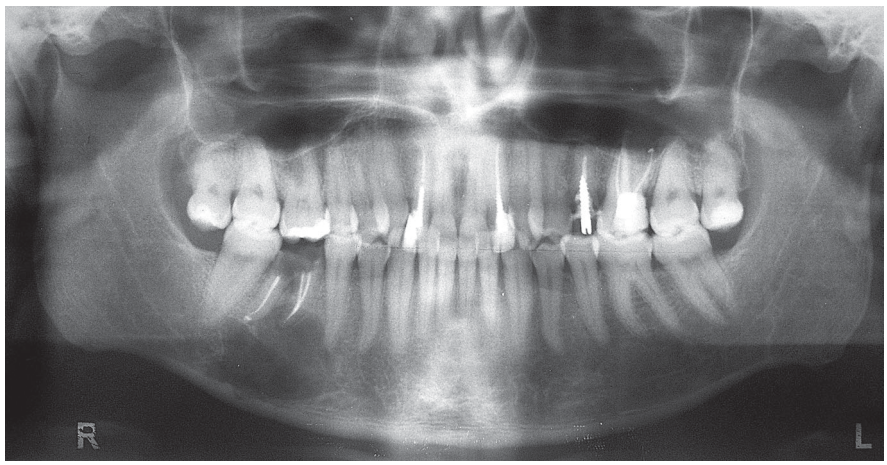

Fig. 1. Radiografic imaging showed radiolucent lesion on right side of lower jaw, well- defined, with resorption of roots of tooth 46

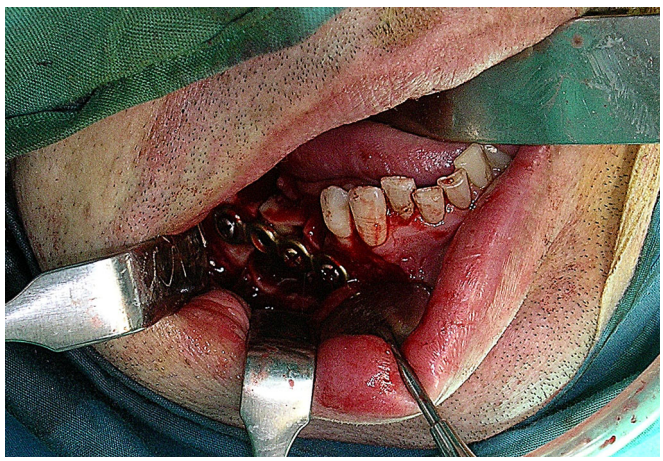

Fig. 2. Intraoperative fixation of the autogenic graft with titanium plate and four screws

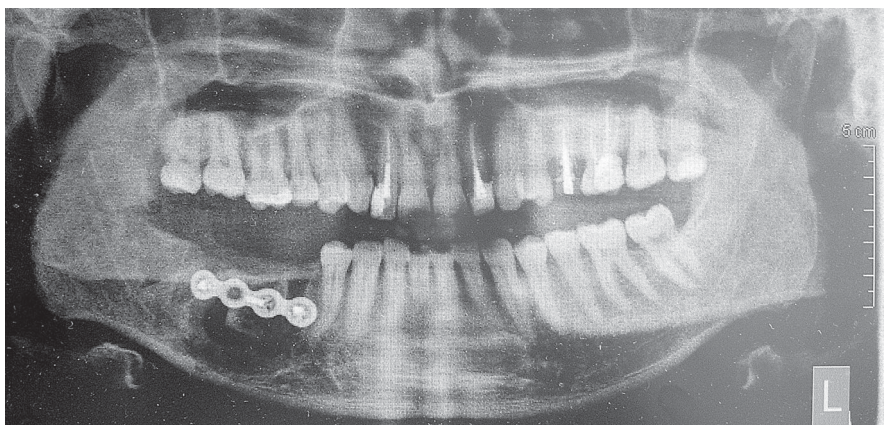

Fig. 3. Postoperative X ray

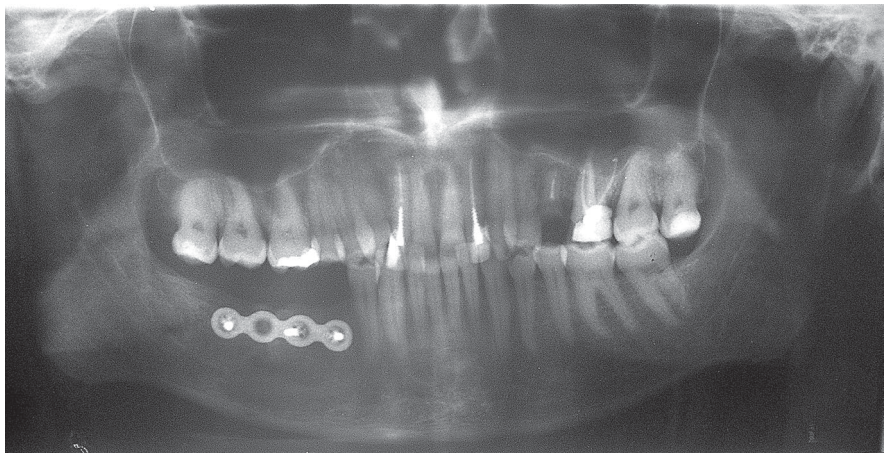

Fig. 4. Postoperative result after 1 year.

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