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Journal of Oral Medicine, Oral Surgery, Oral Pathology and Oral Radiology

Journal homepage: www.joooo.org

Case Report

A central giant cell granuloma in anterior mandible- A case report

Varsha Patel G B^{1,*}, Sangeeth Siddabasappa¹, Shivaprasad S¹, Ashok L¹

¹Dept. of Oral Medicine and Radiology, Bapuji Dental College and Hospital, Davanagere, Karnataka, India



ARTICLE INFO

Article history:

Received 13-05-2022

Accepted 18-05-2022

Available online 18-06-2022

Keywords:

Central giant cell granuloma

Giant cell lesions

Osteolytic lesion

ABSTRACT

Central giant cell granuloma (CGCG) is an uncommon, benign but aggressive osteolytic neoplasm of the craniomaxillofacial region with an unknown etiology. The incidence in the general population is very low with age predilection generally younger than 30 years with a female predominance. CGCG is divided into rare aggressive variant and a common non aggressive variant based on clinicoradiographic features. It is usually unifocal and located at the anterior region of the mandible or maxilla, although are more frequently found in the mandible. A case of a large destructive CGCG involving the entire symphysis of mandible, causing extensive bony resorption, buccal and lingual cortical expansion in a female patient of age 32 years is presented.

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1. Introduction

Central giant cell granuloma (CGCG) is an uncommon, histologically benign but locally aggressive and destructive osteolytic lesion of osteoclastic origin that occurs in the craniofacial region, especially in jaw bones.¹ It was first described by Jaffe in 1953 as an idiopathic non-neoplastic proliferative lesion.²

According to World Health Organization classification, CGCG is defined as “an intraosseous lesion consisting of more or less fibrous tissue containing multiple foci of hemorrhage, aggregates of multinucleated giant cells, some amount of trabeculae of woven bone forming within the septa of more mature fibrous tissue that may traverse the lesion.”^{3,4}

Although the etiology and pathogenesis of CGCG is still unknown, it has been believed to be associated with local trauma, repair processes, inflammatory lesion or any development disorders.²

It accounts for <7% of all benign tumors of the jaws, the mandible being more frequently affected.¹

CGCG has been classified into two types based on its clinicoradiologic features into aggressive and non-aggressive form. Here, we report a case of CGCG on the anterior mandible.¹

2. Case Presentation

A 32-year-old female patient came with the complaint of painless swelling lower 1/3rd of the face since 8 months. Patient noticed the swelling 8 months ago which was gradual in onset, was initially smaller in size and gradually increased to present size. The swelling was not associated with pain. The medical, dental and family history were non-contributory.

Extra oral examination reveals gross facial asymmetry with solitary swelling present on symphysis region, measuring about 5X5cm, extending mediolaterally from the right retrocommisure area to left retrocommisure area and supero inferiorly from vermilion of lower lip and 2cm below the chin, roughly oval in shape, skin and the surface

* Corresponding author.

E-mail address: varsha1996jan@gmail.com (V. Patel G B).

over the swelling is stretched and is not associated with any other secondary signs and symptoms. On palpation, local rise temperature, non-tender, hard in consistency, non-compressible, non-fluctuant, non-mobile. (Figures 1 and 2)



Fig. 1: Clinical picture showing diffuse swelling in the symphysis region of the mandible

Intra oral examination (Figure 3) reveals a solitary swelling in relation to lower anterior vestibule, measuring 5x5cm in greatest diameter, extending mediolaterally from the distal surface of the right canine on to the distal surface of left first premolar. The mucosa over the swelling is stretched and shiny. On palpation, swelling is non tender, bony hard in consistency with buccal and lingual cortical plate expansion.

Based on history and clinical findings a provisional diagnosis of benign odontogenic lesion in lower anterior mandible and a differential diagnosis of central giant cell granuloma, ameloblastoma and central ossifying fibroma was considered.

Orthopantomogram showed the jaws in the permanent dentition stage typical for the age of the patient. It revealed a large multilocular radiolucency, with scalloped margins and a soap bubble and honeycomb appearance in the symphysis region, extending mediolaterally left 2nd molar to right 2nd premolar, which showed evidence of resorption. There was



Fig. 2: Bird view showing diffuse swelling extending beyond the lower border of the mandible



Fig. 3: Intra oral picture showing the swelling in the anterior mandible

noted diverging and expanding margins of the lesion, with cortical plate thinning as well as sclerotic margins at places. There was seen a pronounced expansion along the inferior border of the mandible in the symphysis region, causing its eccentric ballooning with periosteal new bone formation. Thin radiopaque septae separated the locules. Fine bony trabeculations in the lesion give a typical “soap bubble appearance.”(Figure 4)

The computed tomography of axial and coronal portion taken in contrast shows multicystic lesion involving mandible measuring 5.4X3.9X4.5 cm in midline and extending into left Parasymphyseal location which is measuring 2.7X2.1 cm is seen. Thinning of anterior cortex noted. No obvious resorption of the root of the tooth noted. No extraosseous soft tissue component. (Figure 5)

Surgical excision of the lesion was done. Excisional biopsy report revealed the presence of Multiple sections

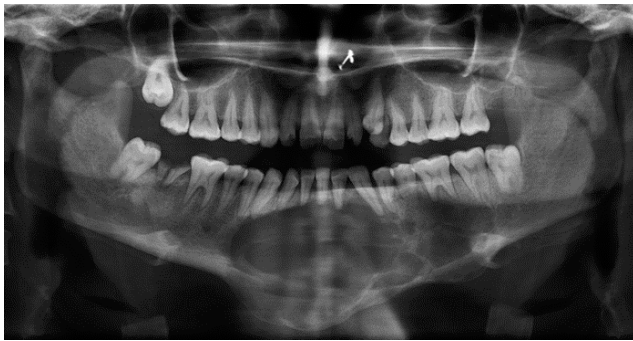


Fig. 4: Orthopantomogram revealed a large multilocular radiolucency, with scalloped margins and a soap bubble and honeycomb appearance in the region of the symphysis mandible

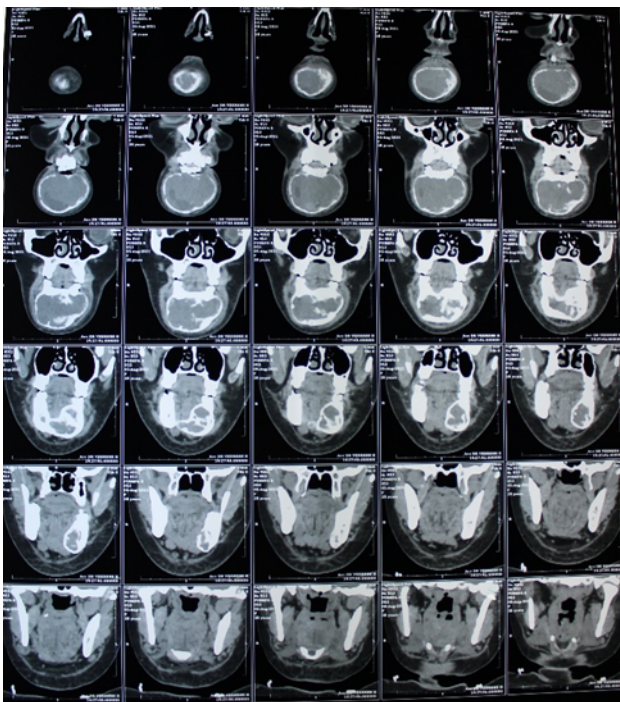


Fig. 5: Computed tomographic scan revealed a multiloculated expansile cystic lesion with bony septae within, measuring 5.4X3.9X4.5 cm in midline and extending into left Parasymphyseal location which is measuring 2.7X2.1 cm is seen. Overlying cortex was thinned out with breaches of its integrity at places

show a capsulated lesion with interrupted bone trabeculae and a diffuse lesion composed of numerous scattered multinucleate giant cells in a background of spindle cell stroma with oval nuclei, vascularised tissue with haemorrhagic areas and fibrous areas, no cellular atypia seen. Features are consistent with central giant cell lesion -mandible.

3. Discussion

Giant cell granuloma and its related lesions in the jaw are grouped under single umbrella but with varied clinical behavior ranging from simple reactive to neoplasm sometimes even manifesting as aggressive malignant neoplasm. The central giant cell granuloma (CGCG) was once thought to represent reactive lesion, however the unpredictable and sometimes occasionally aggressive behavior and because of its possible relationship to tumors of long bone and some syndromes, it is best classified as benign neoplasm.⁵ The incidence of CGCG in the general population is estimated to be 0.0001% with 60% of cases occurring before the age of 30. Gender predilection reports are variable, but the majority of them occur in females with a female:male ratio 2:1.^{6,7} It has been noted that the development of CGCG occasionally coincides with the onset of pregnancy or menarche which is similar to our case. CGCG is more prevalent in the anterior than the posterior jaws, often crossing the midline (50%), and the mandible is more commonly affected than the maxilla and confined to the tooth-bearing areas of the jaws.⁶

Clinically, CGCG may behave variably, exhibiting characteristics ranging from asymptomatic, indolent, and slow growth to aggressive and rapid hollowing out of bone, with cortical expansion, thinning and perforation, root resorption, displacement of adjacent structures including teeth and nerves, accompanied by pain. Perineural invasion and infiltration into adjacent soft tissues are generally not seen, and although the lesion is unencapsulated, it expands, pushing away and displacing adjacent structures, rather than invading or infiltrating into them. It is associated with a relatively high recurrence rate of 15%–20%; the more aggressive the lesion, the higher the chances of its recurrence.¹

Radiographically, CGCG appearance ranges from unilocular to multilocular radiolucent well-defined to ill-defined margins. The lesion bony defects size and nature varies according to the aggressiveness of the lesion.²

Chuong et al. and Ficarra et al. has classified CGCG into aggressive and non-aggressive types on the basis of six criteria like pain, growth rate, swelling, tooth root resorption, cortical perforation and recurrences. Aggressive lesions exhibit pain and rapid growth and usually more than 5cm in size with the features of swelling and cortical bone perforation and teeth displacement and root resorption. And this type of lesion has high chances of recurrence. Whereas, non-aggressive lesions are low growing and have no or less symptoms and may be without associated features.^{2,8}

The differential diagnosis of CGCG includes aneurysmal bone cyst, benign chondroblastoma, brown tumor of hyperparathyroidism, cherubism, fibrous dysplasia, non-osteogenic fibroma, osteosarcoma and true GCT.² Clinical and radiographic features are not definitive diagnosis in CGCG. Two major histological features are diagnostic

in CGCG. There is highly cellular, fibroblastic stroma with plump, spindle-shaped cells with high-mitotic rate. The multinucleated giant cells are irregularly distributed and are prominent throughout the fibroblastic stroma. Histologically, the features of CGCG are indistinguishable from brown tumor of hyperparathyroidism and giant cell lesions, but biochemical tests such as serum calcium, phosphorus, and alkaline phosphatase can be taken into consideration to rule out these lesions.⁹

At present, surgical curettage is still the most frequently applied therapy in CGCG. However, several alternative treatments have been suggested in the literature, including corticosteroid injections, calcitonin, and IFN. In several studies the results of surgical therapy have been evaluated and recurrence rates ranging from 11% to 49% have been reported. In 1988, Jacoway et al. first reported on the treatment of CGCG with corticosteroids. A weekly injection of steroids into the lesion during a period of 6 weeks resulted in a complete resolution in 3 patients. Calcitonin therapy is based on an immunohistochemical study, using osteoclast-specific monoclonal antibodies to demonstrate that giant cells in CGCGs are osteoclasts. Interferon (IFN) is an antiviral and anti-angiogenic agent that is used in a variety of conditions, including life-threatening hemangiomas and several types of malignant tumors. IFN is produced by recombinant DNA technology or is purified from cultured human cells. Among other effects, IFN suppresses the production of fibroblast growth factors (FGF), which are involved in neo-angiogenesis in tumors.⁹

Recurrence rates range from 3 to 72% in young patients, however, lesions with cortical perforation have a greater tendency toward recurrence. Besides the biological behavior of the tumor, the treatment modality is also related to the recurrence rate.^{10,11}

4. Conclusion

CGCG are non-neoplastic and non-proliferative intraosseous lesions with unclear pathogenesis. It can occur in any part of body and shows symptoms as per the location of the lesion. The diagnosis of GCGG is clinical, radiological, and, above all, biological and anatomopathological in nature. The use of a multidisciplinary approach in the diagnosis and treatment of giant cell granulomas involving the maxillofacial skeleton is critical for optimal functional and cosmetic outcomes and rehabilitation.

5. Source of Funding

None.


6. Conflict of Interest

The authors declare no conflict of interest.

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Author biography

Varsha Patel G B, Post Graduate Student  <https://orcid.org/0000-0001-7636-3682>

Sangeeth Siddabasappa, Professor

Shivaprasad S, Professor

Ashok L, Professor and Head

Cite this article: Patel G B V, Siddabasappa S, Shivaprasad S, Ashok L. A central giant cell granuloma in anterior mandible- A case report. *J Oral Med, Oral Surg, Oral Pathol, Oral Radiol* 2022;8(2):97-100.