



## Central giant cell granuloma of mandible: A case report

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### Abstract

Central Giant Cell Granuloma (CGCG) and giant cell tumour of jawbone are grouped underneath same umbrella but with distinct clinical behavior. CGCG is a rare benign lesion of the jaws distinguish by the presence of multinucleated giant cells within a fibrous stroma. The pathogenicity of CGCG still remains an enigma, which demands the differentiation from other look-alike lesions in order to have proper diagnosis and treatment planning. In this article, a case of CGCG in a 30-year-old female patient who presented with complaints of gradually increasing swelling and discomfort in the mandible is described.

**Keywords:** Central giant cell granuloma (CGCG), giant cell lesion, giant cell tumour, multilocular lesions

### Introduction

Jaffe's in 1953 first introduced the term giant cell granuloma, having two different pathological entities, the central giant cell granuloma (CGCG) arising within the bone and the peripheral giant cell granuloma (PGCG) arising in the soft tissues. CGCG, also termed as reparative giant cell granuloma, which is a rare benign lesion of the jaws characterized by the presence of multinucleated giant cells within a fibrous stroma<sup>[1]</sup>.

CGCG occurs most commonly in jaws-more so in mandible comprising approximately 70% of cases. Etiopathogenesis of lesion is unclear, although various theories have been proposed. Some studies suggest a reactive response to local trauma or inflammation, while others implicate hormonal factors, genetic predisposition, or dysregulation of osteoclast function. Recent advancements in molecular biology and immunohistochemistry have provided insights into the pathogenesis of CGCG, highlighting the role of RANK/RANKL signaling pathway, cytokines, and growth factors in the recruitment and activation of multinucleated giant cells<sup>[2]</sup>.

The CGCG of the jaw accounts for approximately 7% of all tumors of the jaw. This enigmatic entity predominantly affects young adults, with a peak incidence in the second and third decades of life, although it can occur at any age. The usual clinical presenting feature is of a painful or painless swelling or localized bony expansion, often discovered incidentally on routine radiographic examination or following complaints of facial asymmetry or discomfort<sup>[3]</sup>.

Most of the CGCG are located in the anterior to first molar in mandible in the incisor, canine and premolar regions. Radiographically, CGCG may manifest as a unilocular or multilocular radiolucency with well-defined or ill-defined margins, occasionally exhibiting cortical expansion or perforation<sup>[4]</sup>.

Histopathologically, CGCG is characterized by the presence

of multinucleated giant cells dispersed within a background of spindle-shaped or ovoid mononuclear cells arranged in a collagenous stroma<sup>[5]</sup>.

In this article, a case of CGCG in a 30-year-old female patient who presented with complaints of gradually increasing swelling and discomfort in the mandible. Furthermore, it underscores the variable clinical presentation and unpredictable behavior of this intriguing lesion, emphasizing the need for individualized treatment approaches tailored to the patient's specific clinical and radiographic characteristics

### Observation

A 32-year-old female patient reported to the department of oral medicine and radiology with a chief complaint of swelling over lower left jaw since 6 months. Swelling was initially small and gradually enlarged to cause facial disfigurement. The swelling was associated with pain in since last 1 month. Pain was insidious in onset, mild, throbbing, intermittent and non-radiating in nature. The past medical and family history was non-contributory. No history of trauma was elicited.

A physical examination revealed moderately built and well-nourished woman with no history of any known systemic disease.

On extraoral examination a facial asymmetry noted. A diffused swelling noted on lower left side of jaw roughly extending supero-inferiorly from line joining the corner of mouth to ear lobe up to lower border of mandible, antero-posteriorly from corner of mouth to angle of mandible. (Figure 1)) The overlying skin were normal however, on palpation, the swelling was non-tender, non-compressible, non-pulsatile. No paresthesia noted.



**Fig 1:** Extraoral Examination: Gross facial asymmetry due to diffuse swelling on left side of mandible

An intraoral examination revealed a solitary swelling in lower left buccal vestibule extending from distal aspect of 37 to retromolar region. The overlying mucosa appeared normal in color whereas, on palpation the swelling was firm in consistency with buccal cortical expansion. (Figure 2)

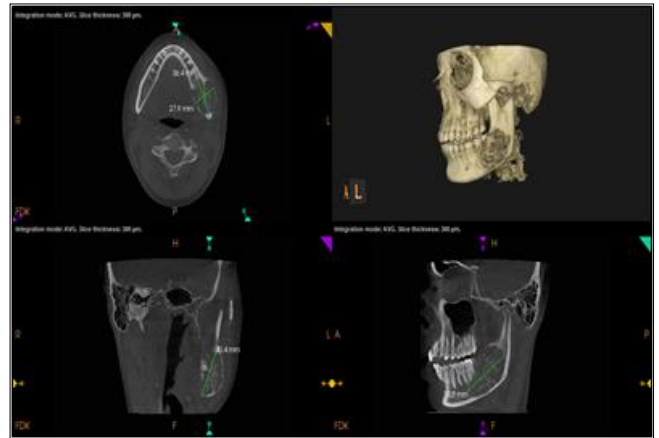


**Fig 2:** Intraoral examination: Mild obliteration of buccal vestibule in 37 and 38 regions

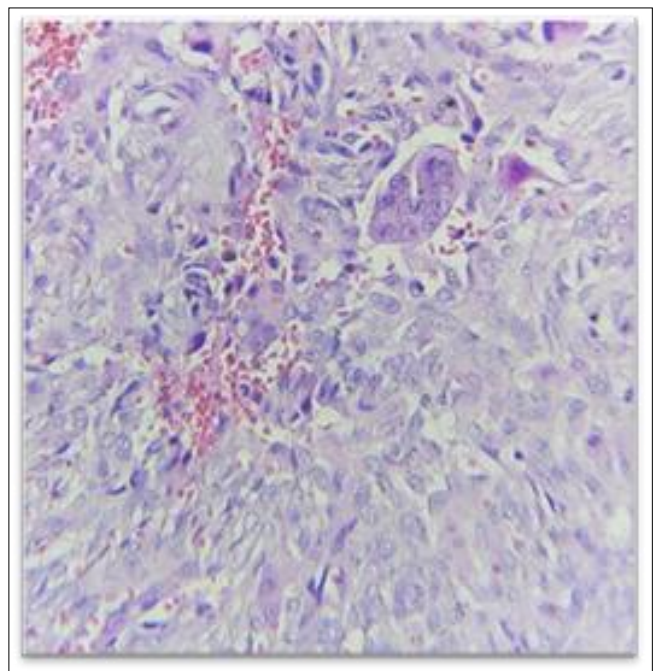
Reconstructed panoramic radiograph shows (Figure 3), well-defined multilocular lesion with whispy, granular septa at right angle to periphery with thinning of inferior border of mandible.



**Fig 3:** Reconstructed Panoramic: Well-defined multilocular lesion with whispy, granular septa at right angle to periphery with thinning of inferior border of mandible.



**Fig 4:** CBCT Imaging: Showing extension of lesion in sagittal, coronal and axial section.



**Fig 5:** Photomicrograph hematoxylin and eosin, ×40: Multinucleate cells which are distributed in highly vascular stroma comprising both spindle-shape and round cells and found mostly in area of hemorrhage



**Fig 6:** Reconstructed panoramic Image: Follow up after 4 months

CBCT scan revealed well-circumscribed expansile mass with the presence of subtle granular bone pattern at the periphery of the expanded bone with internal septa. The lesion extending from distal aspect of 36 posteriorly involving about one third of ramus of mandible in anteroposterior direction, alveolar crest to inferior border of

mandible in superior-inferior direction. In axial section, the lesion involved the ramus of mandible, and expansion and thinning of inner cortical plate of mandible. Coronal slices demonstrated the resorption of the outer cortical boundary, a multilocular appearance, an expansible nature, and thinning and resorption of the inner cortical boundary. (Figure 4)

Based on above findings, provisional diagnosis of aggressive benign tumour was given. Central giant cell granuloma, hyperparathyroidism, ameloblastoma, keratocystic odontogenic tumour (KOT) were considered in differential diagnosis. Laboratory investigation of parathyroid hormone was within normal limits excluding the hyperparathyroidism in differential diagnosis.

Incisional biopsy was done, which showed, fibro-cellular connective tissue stroma with numerous giant cells. The giant cells are multinucleated resembling osteoclast. The connective tissue showed loose bundle of polygonal to spindle cell resembling fibroblast arranged in sheets. Connective tissue is also infiltrated with mild chronic inflammatory cells infiltrate mainly lymphocyte and plasma cells. Extravasated RBCs and bony spicule were noted in focal areas. (Figure 5) Based on the above histopathological features diagnosis were modified to central giant cell granuloma.

The patient underwent surgical resection of the lesion. The surgery and recovery were uneventful.

The patient underwent surgical resection of the lesion. The surgery and recovery were uneventful. The patient followed up 3 months after the surgery. There was no residual symptomatology. (Figure 6)

## Discussion

Central giant cell granuloma is locally aggressive lesion of unknown etiology. There is debate as to whether CGC lesions are reactive or neoplastic lesions in origin. Initially it was thought to be reparative reaction of bone to trauma, inflammation or intramedullary hemorrhage. However, due to its aggressive behavior it is considered to be benign neoplasm<sup>[6]</sup>.

CGCG of jaw is benign osteolytic lesion, which accounts for nearly 7% of benign neoplasm of jaw. It shows predilection for young female below 30 years of age. CGCG can be seen in both maxilla and mandible with most cases reported in mandible anterior to molars. In maxilla the common site is anterior to canine<sup>[6]</sup>.

The initial presenting complaint is painless slow growing lesion. In aggressive lesion the palpation of area may elicit tenderness, and in some cases, the patient may complain of pain. Because of highly vascular nature these lesions, the overlying mucosa may have purple color. CGCG, has wide range of radiological features ranging from unilocular to multilocular lesion with well-defined to ill-defined margins<sup>[4]</sup>. 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<sup>[7]</sup>. CBCT visualize structure in multiplanar views and reveal detailed radiographic features of CGCG. Initial lesions are mostly unilocular radiolucent with internal septa. In large aggressive lesion, multilocular lesion with poorly defined border, subtle granular pattern of calcification the septa are right angle to periphery and divide the internal aspect into compartments. CGCG has strong propensity to expand and in some cases bony cortex is destroyed. Inferior alveolar canal may displaced inferiorly and resorption of roots can be seen<sup>[8]</sup>.

CGCG must be differentiated from other similar looking multilocular lesion such as Ameloblastoma, aneurysmal bone cyst (ABC), brown tumor of hyperparathyroidism, odontogenic myxoma and cherubism. Ameloblastoma mostly seen in older age group in molar-ramus region of mandible with internal structure showing thick and coarse septa and has highest propensity to expand than CGCG. ABC is rare lesion seen in posterior mandible with profound expansion than CGCG. Odontogenic myxoma is also rare lesion seen in posterior mandible, has one or more straight septa with less expansion as compare to CGCG. If CGCG occurs after second decade, hyperparathyroidism should be considered and necessary laboratory investigation should be advised to rule out the same<sup>[9]</sup>.

Differentiating giant cell tumor (GCT) from CGCG is difficult and confusing. GCT are benign and locally aggressive true neoplasm. These occurs in third and fourth decades of life can go into malignant transformation. Both these lesions can differentiate based on histopathological characteristics. CGCG have relatively fewer multinucleated giant cells than GCT with increased incidence of osteoid, fresh haemorrhages and hemosiderin deposits. In contrast, the giant cells are more evenly distributed in GCT<sup>[10]</sup>.

Surgical management is the most common modality considered. Which is done using two different modality such as curettage and adjunctive treatment such as cryotherapy, osteotomy and resection. Literatures have reported showing higher recurrence rate of 33-75% after curettage and 10-20 % recurrence rate after resection so, total surgical resection is considered to be best for CGCG. Post surgical morbidity may be high due to severe aesthetic and functional problems.

Different non-surgical approaches including  $\alpha$  interferon, calcitonin and intralesional corticosteroid injections have been evolved over last years to avoid mutilating surgery as well as to reduce recurrence after surgical management<sup>[11]</sup>.

## Conclusions

In conclusion, the early diagnosis of central giant cell granuloma (CGCG) is crucial for successful management and treatment outcomes. This case report underscores the significance of prompt recognition and intervention in cases presenting with characteristic clinical and radiographic features. Further research is warranted to elucidate the underlying mechanisms driving CGCG pathogenesis and to explore novel therapeutic strategies aimed at improving treatment outcomes and minimizing recurrence rates.

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The authors declare that they have no conflicts of interest in relation to this article.

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