

Massive central giant cell granuloma of the mandible causing difficulty in speech and mastication

Abstract

Central giant cell granuloma (CGCG), a non odontogenic lesion of the oral cavity the involving jaws, is relatively uncommonly seen. It accounts about 1–7% of all benign lesions of the jaw. It is bony lesion of the head and neck region which is never seen in any other bone of the skeleton. It is usually seen in young adults between second to third decades of their life. CGCG has been classified, as central (bone) and peripheral (gingival tissues) according to type of tissue involved.

Keywords: central giant cell granuloma, non-odontogenic lesion, bony lesion of jaw

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Introduction

Central giant cell granuloma (CGCG) is a benign lesion of the bone which was first described by Jaffe in 1953.¹ It affects children and young adults between 2nd and 3rd decades of life.² Females are affected more than males in ratio of approximately 2:1.^{3,4} CGCG may involve mandible twice compared to maxilla, lesion is frequently seen anterior to first molar in early age and posterior to mandible after second decade, in case of maxilla lesion frequently involve anterior to canine.^{5,6} True nature and origin of CGCG is unknown and controversial, whether it is a reactive lesion, a developmental anomaly or a benign neoplasm.⁵⁻⁷ Neville et al.⁴ has classified CGCG as a non-neoplastic lesion whereas World Health Organization (WHO)⁸ considers it as a bony lesion not a tumour. Aetiology of CGCG is unknown however its progression has been associated with trauma, inflammation^{9,10} onset of pregnancy and menarche.¹¹ Treatment of CGCG is either medical or surgical depends upon size and site of lesion.

Case presentation

A 25 years old female presented to Department of Oral and Maxillofacial Surgery King George's Medical University Lucknow with complaint of progressively increasing painless swelling in oral cavity since 8 month. Some mobility also appeared in teeth over right side of lower jaw. Patient reported to a local clinic where some antibiotics were prescribed for 10 days but there was no improvement and swelling was gradually increasing in size causing difficulty in mastication, speech, swallowing and closing the mouth. Spontaneous exfoliation of right second premolar, first and second molar occurred within 6 months. Medical history of the patient was insignificant. No history of any ill habit like pan masala, tobacco chewing was reported. On extra-oral examination a swelling measuring 5x5cms was seen at the mandible obliterating the oral cavity. It was reddish in colour and there was no ulceration or pus discharge. Bleeding was seen on probing. On palpation it was soft to firm in consistency and non tender. No submandibular or submental lymph nodes were palpable. Intra-oral examination revealed a large mass extending from left canine

to retromolar triangle of right side measuring 10x8cm in dimension obliterating the buccal sulcus. Right central, lateral incisors, canine and left central, lateral incisors and canine were mobile and got proclined because of swelling. Tongue was not visible due huge size of lesion (Figure 1). All the haematological and biochemical investigation were within normal limits. Orthopantomogram (OPG) showed large lytic lesion involving right side of body of mandible and bone loss at the alveolar process of central, lateral incisor and canine teeth (Figure 2).



Figure 1 Extra-oral view showing extent of the lesion.

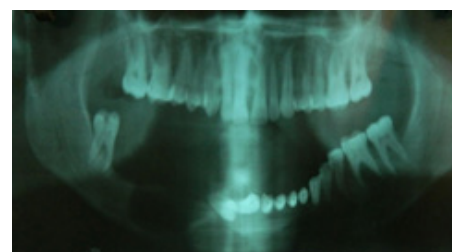


Figure 2 Pre-operative OPG showing large expansile lytic lesion involving body of mandible.

CT scan revealed well defined heterogeneous enhancing osseous lesion measuring 6.6x7.9x3.5cm in size involving g body of mandible

and lower alveolar arch causing their destruction with adjacent soft tissue lesion with bilateral level Ib lymphadenopathy (Figure 3&4). Histopathological finding revealed stratified squamous epithelium showing epithelial hyperplasia with ulceration. Sub-epithelial zone showed fair number of giant cells in edematous stroma along with mixed inflammatory cell infiltrates comprising lymphocytes, neutrophils, plasma cells, macrophages along with proliferating vascular channels (Figure 5). Differential diagnosis includes Ameloblastoma, aneurysmal bone cyst, odontogenic fibroma, odontogenic myxoma, brown tumor of hyperparathyroidism

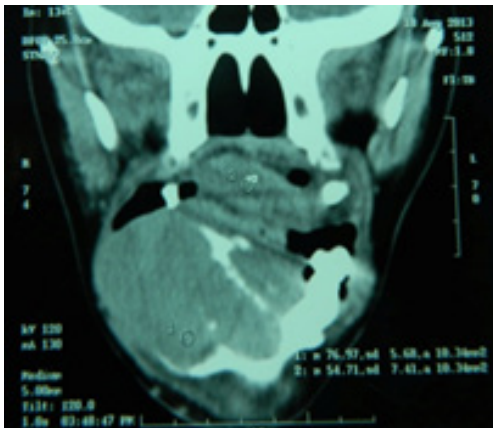


Figure 3 CT scan axial showing large expansile heterogeneously enhancing osseous destructive lesion involving body of the mandible.

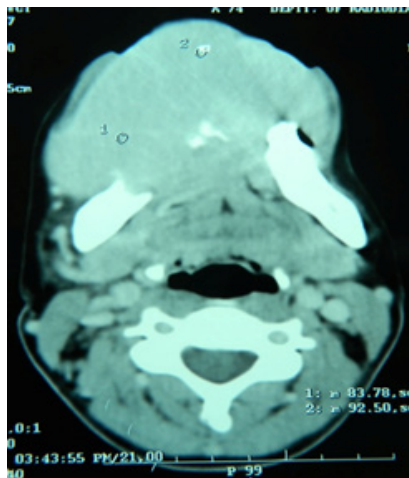


Figure 4 CT coronal view showing large expansile heterogeneously enhancing osseous destructive lesion involving body of the mandible with adjacent infiltration.

Treatment

Provisional diagnosis of CGCG was made and planned for surgery under general anaesthesia. Even though the huge size of the lesion extra warranted extra-oral approach, taking the age and sex of patient consideration intra-oral approach was decided upon. A small incision was given and after securing haemostasis incision was extended forward. In this way lesion was excised in toto through intra-oral approach with adequate margins (Figure 6&7). Post-operative course was uneventful and patient recovered well. Patient is under regular follow-up and no recurrence has been seen after one year of surgery (Figure 8&9).

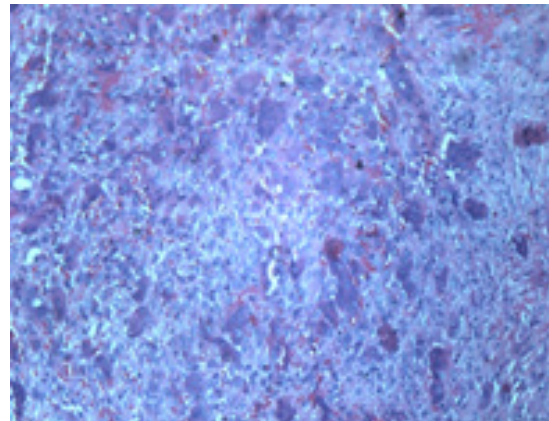


Figure 5 Histopathology of the lesion showing fair no of giant cells, chronic inflammatory along with minimal fibrosis in background. (haematoxylin 400X).



Figure 6 Intra-operative photograph after excision of lesion.



Figure 7 Excised specimens.

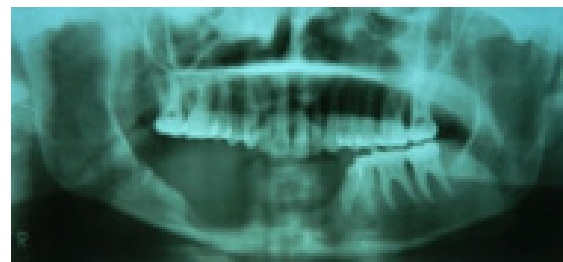


Figure 8 Post operative OPG showing well defined sharp post-operative margins.



Figure 9 Post operative photograph of patient.

Discussion

Giant cell tumors are frequently seen in oral cavity. They are classified according to their origin as central and peripheral. Central giant cell tumor originate from bone whereas peripheral tumor originate from gingival soft tissue, periosteum or periodontal ligament.¹¹ Reported lesion was central type as it originated from bone. On the basis of clinical and radiographic features CGCG can be classified in two categories aggressive and nonaggressive types.^{12,13} The non-aggressive type is more common and present as slow growing painless swelling. Few cases (20%) present with pain or paresthesia.^{3,5} Radiographically they are seen as well-defined unilocular or multilocular radiolucencies with undulating borders.⁵ Aggressive type of CGCG is fast growing swelling with high recurrence rate.³ Fast growth of the lesion is evident as ill-defined borders and destruction of cortical bone.⁵ Our patient had aggressive type of CGCG because swelling was fast growing and became huge within short period of time. Histological differential diagnosis of CGCG includes, brown tumour of hyperparathyroidism, cherubism, Noonan syndrome, neurofibromatosis Type 1^{14,15} fibrous dysplasia and aneurysmal bone cyst.¹⁰ In hyperparathyroidism there is increase in serum levels of calcium, phosphorus, alkaline phosphatase and renal function is deranged. Cherubism usually present as intra-osseous fibrous bilateral swellings of the jaw which is hereditary and microscopically indistinguishable from GCRG. In fibrous dysplasia there is Chinese letter like trabeculae of immature or woven bone. Aneurysmal bone cysts have multiple large spaces which are filled with blood and fluid level can be seen on the radiograph. The radiographic differential diagnosis includes aneurysmal bone cyst, odontogenic cyst, ameloblastoma, odontogenic fibroma and odontogenic myxoma.¹⁶ There are two treatment modalities for treatment of CGG. i.e. medical and surgical. Surgical part includes curettage and resection. Medical treatment includes intra-lesional injection of corticosteroids and calcitonin by subcutaneous route or as nasal spray.^{14,17,18} Reported case was treated surgically because medical treatment is effective in small size lesions. Recurrence depends upon various factors. Main cause of recurrence is incomplete excision or inadequate margins taken during surgery. Other factor is nature of the lesion, aggressive lesions show greater tendency of recurrence. Recurrence rates for CGCG ranges between 11%–49%.⁶ No recurrence was seen in our case after one year of surgery, in spite of its aggressiveness as we had taken adequate margins.

Conclusion

CGCG is relatively uncommon benign lesion frequently seen in young adults between second to third decades of life. On the ground of origin it has been classified as central and peripheral types. On the ground of nature it may be aggressive and locally destructive or non aggressive and slow growing type. Giant cell granuloma may present with differing clinical presentation. Early diagnosis makes the treatment easy and more conservative avoiding radical surgeries and facial disfigurement. Medical treatment is recommended for small lesions but even huge lesions can be surgically managed by intra-oral approach, if proper surgical technique is applied.

Acknowledgments

None.

Conflicts of interest

The authors declare that there is no conflict of interest.

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