

Central Giant Cell Granuloma of Mandible: A Case Report

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Abstract

Central Giant Cell Granuloma (CGCG) is a benign tumour of the jaws. It is relatively uncommon. The most common site involved is anterior part of mandible, especially in females under 30 years of age. The aim of this paper is to present such an uncommon entity in 25 year old Indian female and the diagnostic challenges it posed as it mimics various giant cell lesions of genetic disorders with its treatment options.

Keywords: benign tumor, central giant cell granuloma, neurofibromatosis.

Introduction

Central giant cell granuloma is a relatively uncommon benign bony lesion of a variable aggressive nature, accounting for less than 7% of all benign lesions of jaw.¹ It was first described by Jaffe in 1953. He initially coined the term central giant cell reparative granuloma for this lesion. Nowadays, as reparative response is quiet rare and most lesions of CGCG are destructive, the word reparative has been deleted from that term.²

The true nature of this lesion is controversial and remains unknown. The three competing theories are that it could be a reactive lesion, a developmental anomaly or a benign neoplasm. Neville et al consider this entity to be a non-neoplastic lesion and the World Health Organization (WHO) classifies it as a bone-related lesion, not a tumour, although its clinical behaviour and radiographic features often are those associated with a benign tumour.² Recently the World Health Organization has defined it as localized benign but sometimes aggressive osteolytic proliferation consisting of fibrous tissue with haemorrhage and haemosiderin deposits, presence of osteoclast-like giant cells and reactive bone formation.³

On the basis of clinical and radiographic features, central giant cell granuloma has been classified into two types:

1. Aggressive lesion: It is found in young patient characterized by rapid growth, pain, expansion and/or perforation of the cortical bone, induce root resorption and high recurrence rate.⁴
2. Non-aggressive lesion: It is characterized by slow growth that does not perforate the cortical bone or induce root resorption and has a low recurrence rate.⁵

The incidence of CGCG in the general population is estimated to be 0.0001%.⁶ 60 to 70% of cases are diagnosed in patients younger than 30 year old.⁷ Gender predilection reports are variable, but the majority occur in females with a female–male ratio of approximately 2:1. Lesions develop twice as often in the mandible with an epicentre anterior to the first molar in young patients and there is a tendency for the epicentre to occur in the posterior aspect of the jaws after the first two decades of life.

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Histologically, the features of CGCG are indistinguishable from the brown tumour of hyperparathyroidism and from giant cell lesions of genetic disorders such as cherubism, Noonan syndrome and neurofibromatosis Type 1.2

The aim of this paper is to present a rare case of central giant cell granuloma in 25 year old Indian female. This case report also describes the classification, clinical features, radiographical features of the lesion along with its treatment options.

Case history

An Indian female who was 25 years. of age, reported to the Department of Oral Medicine and Radiology at Maharishi Markandeshwar University, Mullana with a chief complaint of swelling in the right lower back tooth region since 5 months. Initially the swelling was small in size but gradually it attained the present size. There was history of fever when the swelling first appeared. It was not associated with any pain or discharge. While taking patient’s past dental history, it revealed that the patient underwent uneventful extraction w.r.t 46 as it was badly decayed.

On examination, asymmetry was seen on the right side of the face near the angle of the mandible (Fig.1). The swollen area was hard in consistency and without overlying hyperaemia. It was non-tender and was not fixed to underlying structures. A single right submandibular lymph node of approx 1 cm in size, oval shaped was palpable and was non tender.



Fig.1 Asymmetry on the right side of the face near the angle of the mandible



Fig.2 Ex-pansive bony mass of about 2 x 2 cm in size extending from the first to second molar region



Fig.3 Obliteration of buccal vestibule

Intraoral examination revealed an ex-pansive bony mass of about 2 x 2 cm in size extending from the first to second molar region (Fig.2). There was presence of obliteration of buccal vestibule in the same region. Surface of the swelling was smooth and the colour was same as that of surrounding mucosa (Fig.3). On palpation, it was firm, non tender. It was not associated with any motor or sensory disturbance.

Panoramic radiography showed a 2x2 cm well defined monocular radiolucency in the right body of the mandible, in the first molar region. Displacement of right mandibular 2nd molar was noted, but no root resorption of the surrounding teeth was observed (Fig.4).



Fig.4 Well defined monocular radiolucency in the right body of the mandible, in the first molar region with displacement of right 2nd molar

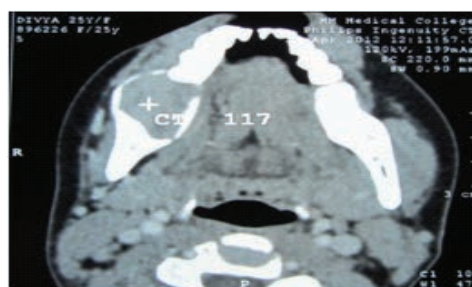


Fig.5 Lytic expansile mass with heterogenous enhancement of size 2.2 cm x 2.1 cm involving mandibular alveolus on the right side in relation to the lower premolar & molar teeth extending to the mandibular ramus causing severe erosion and expansile destruction of the mandible

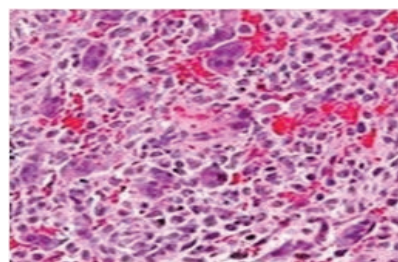


Fig.6 Interspersed multinucleated giant cells

CT scan revealed evidence of lobulated soft tissue predominantly lytic expansile mass with heterogenous enhancement of size 2.2 cm x 2.1 cm involving mandibular alveolus on the right side in relation to the lower premolar & molar teeth which was extending to the mandibular ramus causing severe erosion and expansile destruction of the mandible (Fig.5). Laboratory values for serum calcium, phosphorous, alkaline phosphate and PTH were also within normal limits.

Intraoral biopsy was performed. It revealed high cellular connective tissue stroma with areas showing bone formation. The cellular stroma predominantly consists of spindle shaped plump mesenchymal cells and between these cells multinucleated giant cells with 8-12 nuclei and deeply eosinophilic cytoplasm were interspersed. These giant cells were present throughout the lesion. On the basis of laboratory investigations and histopathological investigation diagnosis of central giant cell granuloma was given.

The patient underwent curettage of the lesion followed by removal of the peripheral bony margins through a retromolar approach. The postoperative course of the patient was uneventful.

Discussion

CGCG primarily occurs in the jaw and facial bones, but it is also found in other areas of the body.² CGCG predominantly affect females, with occurrence between 56% and 64%.² The present case describes CGCG in young female. It is usually asymptomatic painless expansion of the affected bone as seen in our case. However, our case is also consistent with previous reports of CGCG presenting in the mandible more often than the maxilla. Lesions of CGCG do not induce paraesthesia.

The radiographic appearance of CGCG is not pathognomonic. Both multilocular and unilocular lesions are possible, but multilocular lesions show slight predominance over unilocular lesions. In the present case the lesion was unilocular in appearance.

To differentiate from other giant cell lesions laboratory investigations were done. Laboratory values for serum calcium, phosphorous, alkaline phosphate and PTH were within normal limits as were the blood cell counts.

In cases of brown tumor of hyperparathyroidism there is hypercalcemia, hypophosphatemia and elevated parathyroid hormone secretion. Radiographically, there are multiple lytic radiolucencies. In cherubism, neurofibromatosis type 1 and Noonan syndrome, there are multiple lytic lesions. In the present case, all the laboratory investigations were in normal range. Radiographically, the lesion was unilocular.

Histologically, CGCG mimics brown tumor of hyperparathyroidism, Cherubism, Neurofibromatosis type-1, Noonan syndrome. Because of this histological similarity, it has been hypothesized that CGCG may have a genetic aetiology, although there is no convincing evidence to support this hypothesis. Therefore, the final diagnosis of CGCG was made on the basis of clinical-radiological-biochemical-histological investigations.

Surgery is the most accepted treatment in CGCG. In our case, the patient subsequently underwent curettage of the mass. Patient was given interrupted sutures at the site of the wound to prevent hematoma formation. The post operative course was uncomplicated.

Conclusion

CGCG though a rare disease of head and neck sometimes shows aggressive behaviour. Therefore, it is necessary to establish correct diagnosis on the basis of clinical-radiographical-biochemical and histological investigations. Surgery is the most accepted treatment for CGCG. It may be combined with local injections of steroids and calcitonin to reduce recurrence rate.

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