

CENTRAL GIANT CELL GRANULOMA OF MANDIBLE: A CASE REPORT

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Abstract

Central Giant Cell Granuloma (CGCG) of the jaws is an uncommon benign intraosseous lesion commonly seen in the mandible, anterior to first molar with a gender predilection for females. The true nature of this lesion is controversial and it presents with varied biologic behaviour. Clinically they present as asymptomatic expansile swellings. Radiographically they manifest as multilocular radiolucency causing profound expansion of the cortical bone. The clinical and radiographic features of CGCG overlap with many other lesions and so a definitive diagnosis can be established only through histopathology. This article discusses a case of CGCG involving the posterior mandible and the considerations related to diagnosis.

Key-words: Giant Cell Tumour, Intraosseous Lesion, Multilocular Radiolucency.

Introduction

Central Giant Cell Granuloma (CGCG) was defined by World Health Organization in 1992, 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.”¹ CGCG is an uncommon, benign, nonodontogenic lesion with unknown etiology. Jaffe in 1953 first introduced the term central giant cell reparative granuloma (GCRG) to distinguish this lesion from the giant cell tumor of long bones. However, since a reparative response was quite rare and most of these lesions were found to be destructive rather than reparative, the word ‘reparative’ was omitted and the term GCRG was changed to CGCG.² CGCG was once regarded as a reactive lesion as it was thought to represent a reparative response to intrabony haemorrhage and inflammation. However, because of its unpredictable and occasionally aggressive behaviour and its possible relationship to the giant cell tumor of long bones, CGCG is best classified as a benign neoplasm.³

Case Report

A 38 year old female patient presented with a slowly progressive painless swelling in relation to the left side mandible since 7 months. Her medical and family histories were not significant. She gave a history of extraction of grossly decayed left lower posterior teeth 6 yrs back.

Extra oral examination revealed a swelling of size approximately 4cm × 4cm over the left mandibular body and ramus. The surface of the swelling was smooth with no evidence of sinus opening or pus discharge. On palpation the swelling was bony hard in consistency with no tenderness. There was no significant regional lymphadenopathy. Intraorally a swelling of size approximately 3cm×4cm was evident extending from the distal aspect of 35 till the retromolar pad posteriorly. There was obliteration of buccal vestibule and lingually swelling extended up to the floor of the mouth. Except for the indentations of opposing tooth, there was no other surface

changes on the overlying mucosa and the teeth 36, 37, 38 were clinically missing. [Figure 1]



Figure 1: Intraoral swelling extending from 35 to retromolar region and surface shows indentations of opposing tooth

On palpation the swelling was hard in consistency; non-tender with expansion of bucco- lingual cortices. Based on the history and clinical findings we arrived at a provisional diagnosis of a benign tumour probably Ameloblastoma. Differentials considered were odontogenic myxoma, CGCG, brown tumor of hyperparathyroidism and central ossifying fibroma. Thermal vitality test revealed immediate response in relation to teeth 33, 34 and 35. On lesional aspiration few drops of blood was obtained. Routine hemogram, Serum calcium, phosphorous, and parathyroid hormone assay were in normal limits. Intraoral periapical radiograph revealed a mixed radiolucent – radiopaque lesion extending from the distal aspect of 35 with widening of periodontal ligament space, loss of lamina dura and mild root resorption in relation to 35 and the internal architecture showed multiple curved septations. [Figure 2] The mandibular lateral occlusal radiograph showed bucco-lingual cortical expansion.

Orthopantomogram revealed a well defined mixed radiolucent – radiopaque lesion surrounded by a thin corticated scalloped margin extending from the distal aspect of 35 till the sigmoid notch posteriorly with thinning of inferior cortical border of mandible. The internal aspect consisted of multiple thin ill defined curved septations with

uneven large locules and the inferior alveolar nerve canal was not traceable on the involved side. [Figure 3]



Figure 2: Intraoral periapical radiograph revealing a mixed radiolucent – radiopaque appearance with multiple septations



Figure 3: Orthopantomogram showing a well defined mixed radiolucent – radiopaque lesion with a soap bubble appearance and thinning of inferior cortical border of mandible.

CT coronal bone window revealed a heterodense expansile mass in relation to left side posterior mandible with marked thinning and expansion of bucco- lingual cortices and a defect in the lingual cortex. [Figure 4A]



Figure 4A: CT coronal bone window depicting a heterodense expansile mass in relation to left side posterior mandible with expansion of bucco- lingual cortices.

CT 3D reconstruction revealed through and through cortical violation with destruction of inferior cortical border, angle and ramus of mandible. [Figure 4B]

Incisional biopsy was done and histopathology revealed a fibrovascular connective tissue stroma with irregularly distributed foreign body type of giant cells located mainly at the periphery of extravasated blood with no evidence of odontogenic epithelium suggestive of CGCG. [Figure 5]

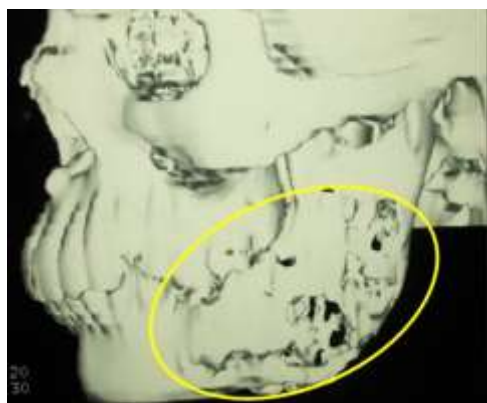


Figure 4B: 3D CT revealing cortical violation with destruction of inferior cortical border, angle and ramus of mandible.

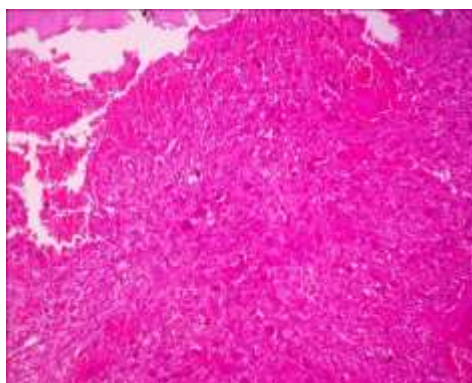


Figure 5: Histopathology shows fibrovascular connective tissue stroma with irregularly distributed foreign body type of giant cells at the periphery of extravasated blood (10X magnification)

Segmental mandibulectomy was done and the patient is being followed up periodically.

Discussion

CGCG was classified as a true neoplasm and a reactive proliferative process because of its histologic features, dynamic biologic characteristics, and variable clinical patterns.⁴ The etiopathogenesis of CGCG of jawbones has not been clearly established but it has been suggested that it is the result of an exacerbated reparative process related to previous trauma and intraosseous haemorrhage that triggers the reactive granulomatous process.⁵ CGCG occurs mainly in adolescents and young adults affecting the females more often than males in a 2:1 ratio and is seen most frequently under the age of 30 years⁶ which concurs with our case where the patient was a female. It occurs more often in mandible and the vast majority of the lesions appear anterior to the first permanent molar region, often crossing the midline and practically all occur in the tooth bearing area.⁶ In contrast the location of the lesion in our case was distal to 35 in the posterior mandible and it occurred in an edentulous site where teeth 36, 37 and 38 were missing. CGCG usually is an asymptomatic lesion, which may become evident during routine radiographic examination or

as a result of painless but visible expansion of the affected jaw.⁷ Despite CGCG is a benign reactive osseous lesion, it has been classified into two types based on its clinic-radiologic features into a slow growing asymptomatic, nonaggressive lesion, and an aggressive type encountered in younger patients which is painful grows rapidly into a large size, perforating the cortex causing root resorption with a tendency to recur.⁸ The present case belonged to the asymptomatic, nonaggressive type in which the patient presented with facial asymmetry due to a progressively enlarging painless swelling in relation to left side mandible.

The radiographic features of CGCG consist of a multilocular or, less commonly, unilocular radiolucency of bone. The margins of the lesion are relatively well demarcated, often presenting scalloped borders.³ The periphery shows no evidence of cortication and the internal structure occasionally contains granular bone organized into ill defined wispy septa dividing the internal aspect into compartments creating a multilocular appearance.⁹ Radiographically, CGCG appears as a lytic expansile lesion with a soap bubble multilocular radiolucency.¹⁰ This case manifested as a well demarcated mixed radiolucent-radiopacity with a thin corticated scalloped margin and the internal aspect consisted of granular bone pattern with multiple ill defined septa and internal compartments resembling a soap bubble. CGCG often displace and resorb the teeth and lamina dura within the lesion is usually missing.⁹ In our case the periapical radiograph depicted root resorption and loss of lamina dura in 35. The lesion has a strong propensity to expand the cortical boundaries and in some instances the outer cortical plate is destroyed and this feature was apparent in the present case. The clinical differential diagnosis for CGCG includes ameloblastoma, odontogenic myxoma, and odontogenic keratocyst. For patients in younger age group, ameloblastic fibroma, ossifying fibroma, and adenomatoid odontogenic tumor might be added to this list.¹¹

The radiographic appearance of CGCG is not pathognomonic and specific. Lesions such as ameloblastoma, brown tumor of hyperparathyroidism, aneurysmal bone cyst, odontogenic myxoma, Ossifying fibroma and cherubism, needs to be differentiated.^{12,13} The most widely accepted method of surgical treatment of CGCG is aggressive curettage. Curettage of the tumor, followed by the removal of the peripheral bony margins results in a low recurrence rate and good prognosis.

Conclusion

The clinical and radiographic presentation of CGCG is non specific and conflicting as it overlaps considerably with many other entities. The clinical behaviour of CGCG is difficult to predict; may be indolent in its course or may present as an aggressive lesion. Hence, the diagnosis of CGCG is perplexing and so the patient's history, clinical findings together with the meticulous observation of internal architecture of the radiographs and histopathological findings may help in arriving at a final diagnosis.

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