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CASE REPORT

CENTRAL GIANT CELL GRANULOMA- DIAGNOSTIC STEPLADDER WITH A CASE REPORT

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Abstract
Central giant cell granuloma (CGCG) formerly called as giant cell reparative granuloma is a non neoplastic proliferative lesion of unknown etiology. It is a benign intraosseous lesion of the jaws that is found predominantly in children and young adults. 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. It exhibits a spectrum of clinical behavior ranging from nonagressive to aggressive variants. Although benign, it may be locally aggressive, causing extensive bone destruction, tooth displacement and root resorption. This is the report of a case of central giant cell granuloma arising from the anterior mandible.
Key Words: Central giant cell granuloma, Case report, intraosseous lesion

INTRODUCTION

Central giant cell granuloma (CGCG) is a relatively uncommon benign bony lesion of a variably aggressive nature accounting for less than 7% of all benign jaw lesions. First identified in 1953 by Jaffe who initially termed it as a central giant cell reparative granuloma, but nowadays the reparative word has been deleted.[1] WHO 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.[2]

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CASE HISTORY

A 13 year old male patient presented with a swelling in mandibular anterior region of 4 months duration. History revealed an asymptomatic swelling which grew progressively in size. Extraoral examination showed diffuse swelling on lower third of face extending superoinferiorly from lower lip till submental region and mediolaterally from left commisure till midline of lower lip (Fig 1, 2). The lesion was firm and non tender with no rise in local temperature.

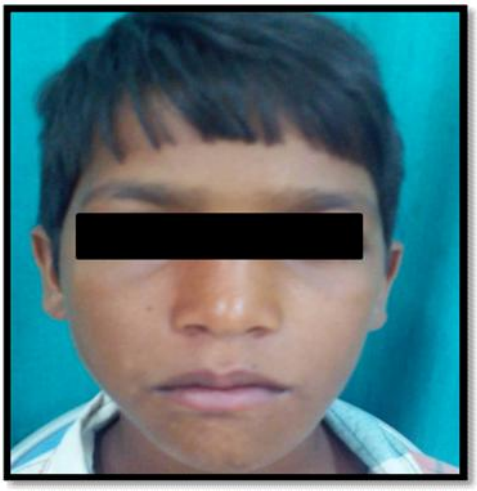


Fig 1- Diffuse swelling in lower third of face

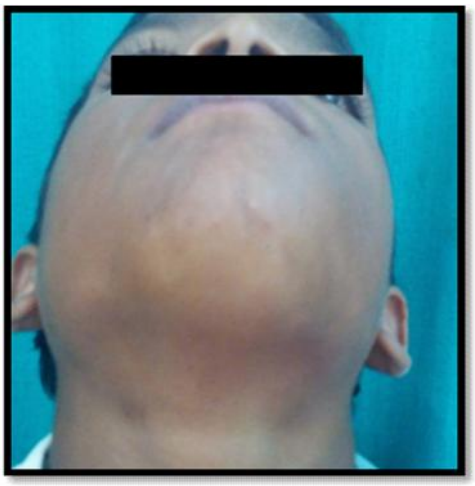
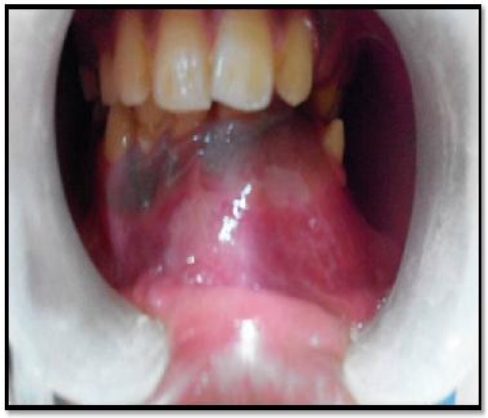


Fig 2- Swelling extending till submental region.

Intraoral examination revealed erupting 33, 34, displaced 31, 41 with an expansive bony mass in the left mandibular vestibule from the 42 to 33 which was firm and non tender in nature (Fig 3, 4). A provisional diagnosis of ameloblastoma was made. Intraoral periapical radiographs with 34, 35 and 41 revealed ill defined multilocular radiolucency (Fig 5, 6).



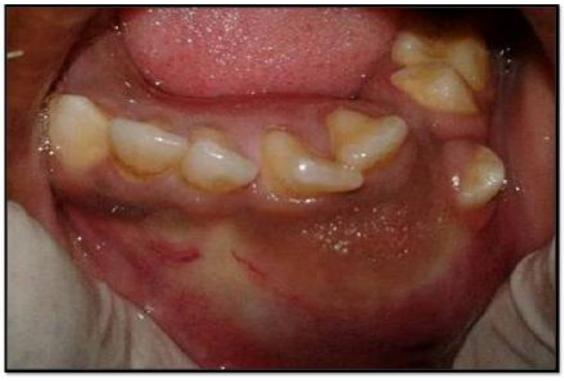


Fig 3, 4 - Expansive bony mass in the left mandibular vestibule from the 33 to 42

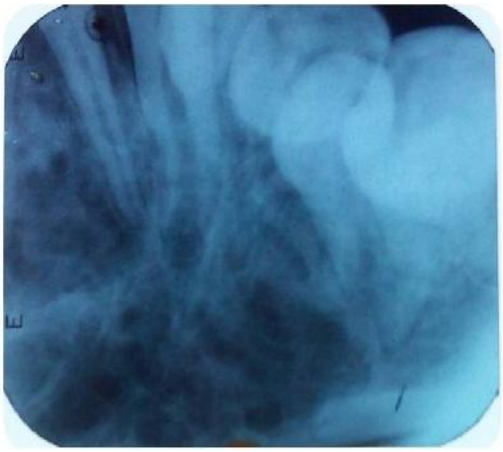


Fig 5- IOPA with 34, 35 revealing ill defined multilocular radiolucency

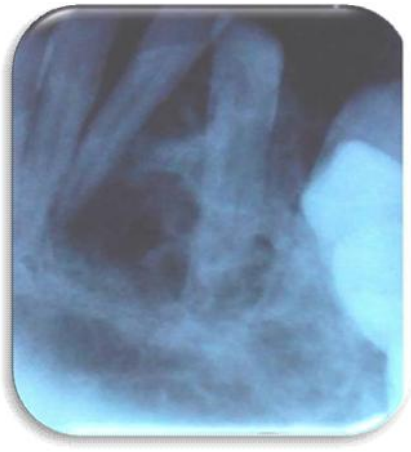


Fig 6- IOPA with 31, 41 revealing ill defined multilocular radiolucency

Mandibular occlusal radiograph showed multilocular radiolucency with displaced 31, 41 along with expansion of buccal and lingual cortical plates (Fig 7). Panoramic radiograph showed a 3x2 cm radiolucent lesion with poorly defined borders in the mandibular anterior region extending from 34 - 44 extending till the lower border of the mandible causing thinning of the same (Fig 8).

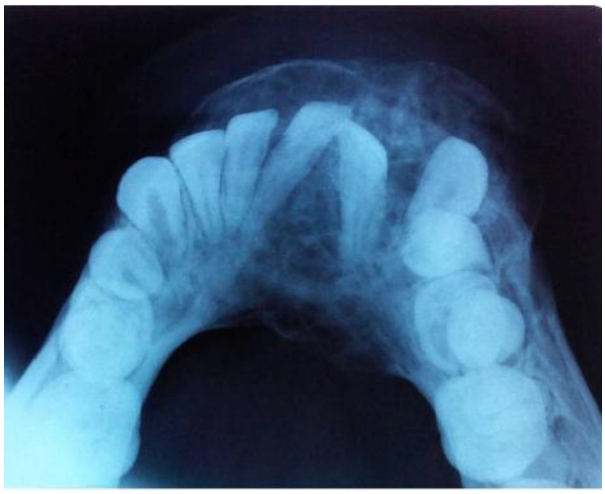


Fig 7- Mandibular occlusal radiograph showing multilocular radiolucency with displaced 31, 41 and buccolingual expansion.

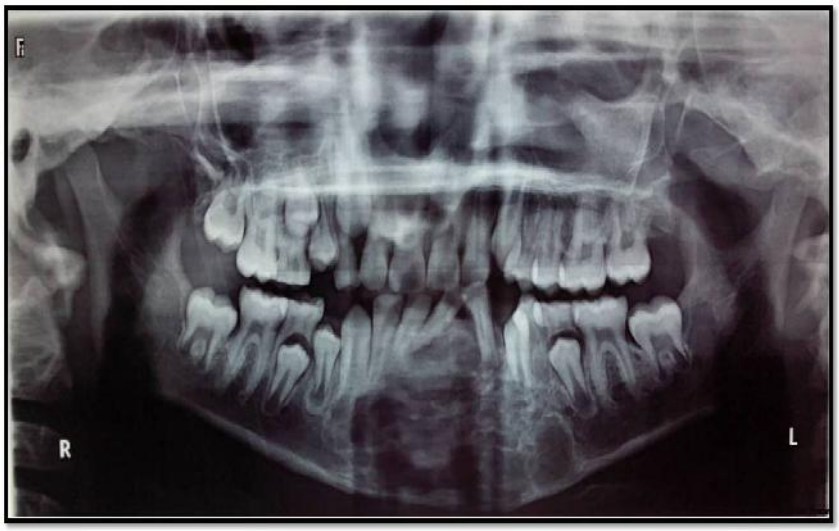


Fig 8- Panoramic radiograph showing radiolucent lesion with poorly defined borders in the mandibular anterior region.

Based on clinical and radiographic findings a diagnosis of Central Giant Cell Granuloma was given and a differential diagnosis of ameloblastoma and odontogenic myxoma was made. Incisional Biopsy was done. Histopathology report showed numerous multinucleated giant cells in a fibrillar stroma. Giant cells consist of nuclei varying from 6-8. Stroma showed numerous proliferating fibroblasts and capillaries (Fig 9).

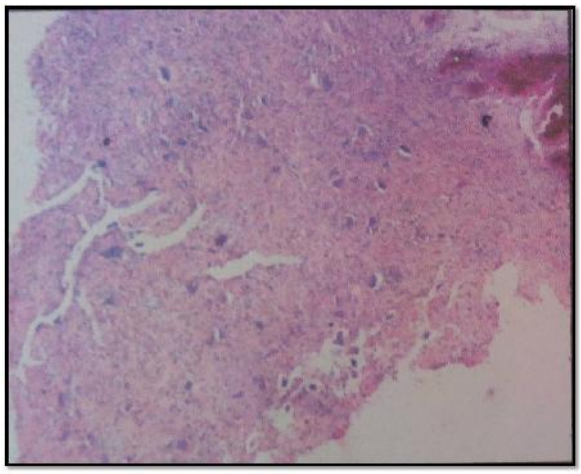


Fig 9- Histopathological view of Central Giant Cell Granuloma

The diagnosis was compatible with CGCG. Patient was sent to the Department of Oral and Maxillofacial Surgery for surgical management of the same. Postoperative evaluation after 3 weeks revealed uneventful healing.

DISCUSSION

CGCG is a non neoplastic proliferation of unknown etiology.[3] Incidence of CGCG is 0.0001%.[4] Etiopathogenesis of the CGCG of jawbones is the result of an exacerbated reparative process related to previous trauma and intraosseous haemorrhage.[2] Cytogenetic abnormalities involving translocations between sex chromosomes and autosomes were identified in a case of CGCG of the phalanx.[5] Association of t(X;4)(q22;q31.3) has also been reported.[6] Regezi and Sciubba proposed three probable theories

1. Response to previous traumatic or inflammatory episode.
2. A true neoplastic process.
3. A developmental anomaly closely related to aneurysmal bone cyst.[7]

The biologic behavior of CGCG of the jaws ranges from absence of symptoms, root resorption or cortical perforation, slow growth, and low recurrence rate, to an aggressive pathological process, characterized by pain (5-11%[1]), rapid growth, root resorption, cortical perforation.[8] Mandible-maxilla ratio is 2:1, with propensity for right side. It can occur from infants to 7th decade of life with general involvement of 2nd – 3rd decade with 74% patients below 30 yrs.[3] Female predominance seen in 68% of cases. Some authors have proposed that while the hormonal therapy has no positive correlation with CGCG, excessively increased levels of estrogen lead to the development of CGCG in the jaws.[9] CGCG also involves small bones, skull, spine, clavicle, tibia, humerus, and ribs. Peripheral type was four times more common than central type and is seen in middle aged and elderly patients.[3] Radiographically it is seen as a unilocular or multilocular radiolucency; well-defined or ill-defined and shows variable expansion and destruction of the cortical plate.[10] CT scan is excellent for demonstration of bone thinning or destruction. In MRI it shows low to intermediate intensity signals on both T1 W and T2 W images.

Histologically multinucleated giant cells in a cellular vascular stoma with new bone formation are detected. The osteoclast like giant cells has irregular distribution and is associated with areas of hemorrhage. Ultra structurally the proliferating cells include spindle shaped fibroblasts, myofibroblasts and inflammatory mono nuclear cells.[3] Its differential diagnoses includes Radicular cyst, AOT, CEOC, Desmoplastic Ameloblastoma, Fibrous Dysplasia,[1] aneurysmal bone cyst, giant cell tumor and brown tumor of hyper parathyroidism.[3]

Chuong et al in 1986 and Ficarra et al in 1987[4] categorized it into : (a) Nonaggressive lesion - slow growing , asymptomatic, without cortical resorption or root perforation and less recurrence and (b) Aggressive lesions – seen in younger patients, painful, grows rapidly, large, often causes cortical perforation and root resorption and has a tendency to recur.[2]

CGCG has also been associated with lesions like fibrous dysplasia, ossifying fibroma, Pagets disease, fibro osseous lesions. Immuohistochemical analysis reveals overexpression of MDM2[7] and cyclin D1.[5] Higher percentage of Ki-67 and PCNA positive cells in CGCG correlates with higher proliferative activity. Immunocytochemistry of fine needle aspirates from central giant cell granuloma have revealed mononucleated and multinucleated cells.[7]

Traditionally management has been surgical by means of excision by curettage in well- located lesions. Curettage with peripheral ostectomy and bone resection is reserved for recurrences. The most aggressive form require en bloc bone resection and reconstruction using autogenous iliac crest graft, dental implants and overdenture prosthesis.[2] Recently, weekly intralesional

corticosteroid injections daily subcutaneous injection of Calcitonin and the use of Interferon Alpha have also been suggested as possible treatments for multiple or large lesions to avoid the need for mutilating surgery in growing children. Radiotherapy is contraindicated because of the potential for malignant transformation.[1] The incidence of recurrence after surgery is 4–20 %. The eradication of lesion does not require more than two excisions.[3] Malignant transformation occurs in 15-20% of cases.[9]

CONCLUSION

Diagnosis of many lesions of the oral cavity is challenging to most clinicians because of their uncommon revalence. A number of cystic, metabolic, osteodystrophic, microbial, tumour and tumour like lesions of the oral cavity present with characteristic giant cell lesions; which makes their diagnosis difficult.[1] CGCG is one of those lesions which have obscure etiopathogenesis with differing clinical presentations and treatment modalities.[7]

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