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AGGRESSIVE VARIANT OF CENTRAL GAINT CELL GRANULOMA

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ABSTRACT
Central giant cell granuloma (CGCG) is an uncommon, benign, proliferative, and nonodontogenic lesion whose etiology is not defined. Central giant cell granuloma (CGCG) consisting of cellular fibrous tissue containing multiple foci of hemorrhage, multinucleated giant cells and trabecules of woven bone. Here we present a case of central gaint cell granuloma in a 35 year old female patient with review of literature.

INTRODUCTION

The central giant cell granuloma, also known as giant cell lesion, was first described by Jaffe in 1953. The WHO has defined central gaint cell granuloma(CGCG) as an intraosseous lesion consisting of cellular fibrous tissue that contains multiple foci of hemorrhage, aggregations of multinucleated giant cells and occasionally trabeculae of woven bone [1]. Some authors separate CGCG into two types, referring to its clinical and radiographic features as (a) Non aggressive lesion which is usually slow growing and asymptomatic;[2] and (b) Aggressive lesion which is usually found in younger patients and is painful, grows rapidly, is larger overall, and often causes cortical perforation and root resorption and has a tendency to recur[3]. Central giant cell granuloma 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 but the radiographic appearance of CGCG is not pathognomonic and specific [4]. Here we present a case of CGCG in a 35 year old female with review of literature.

Case report

A 35 year old female reported to our department of oral medicine and radiology with the chief complaint of swelling and in her lower left back teeth region for past 3 months. History reveals that she had sensitivity in her lower left back teeth region 3 months ago following which she noticed a swelling that increase in size gradually to attain the present size also associated with paraesthesia in that region. Her medical history was non contributory and her dental history revealed that she had visited a private dentist for the same compliant and underwent extraction of her lower tooth 3 weeks back following which the swelling has not subsided (fig-1). Clinical examination reveals the number of teeth present is 30, missing in relation to 28 and 35. Early dental caries in relation to 38, 46, 47 and 48. Intraorally the swelling was diffuse extending from 31 region till 36 region with obliteration of the left lower buccal vestibule (fig-2). On palpation the swelling was firm in consistency with mild tenderness and no compressibility. Chair side investigations such as pulp vitality test was performed which reveals no response in 31 32 33 34 and FNAC was also performed which is negative. Clinically paraesthesia was also evident in lower lip on left side and in the swelling region. With this a provisional diagnosis of benign odontogenic tumor was considered.

Following which a routine radiological investigation was performed with OPG which reveals a well-defined radiolucency measuring approximately 4 X 3 cm extending from midline till mesial aspect of 36 region, with well corticated periphery, resorption of the root in relation to 33 and displacement of inferior alveolar canal inferiorly (fig-3). Following this an advanced investigation with CBCT was taken which reveals through and fro perforation of the mandible measuring 3.6 X 2.4 cm with root resorption in relation to 33(fig-4). The routine hemogram were within the normal limits and the serum chemistry of calcium, phosphorous, parathyroid harmone was also normal, there by excluding the possibility of hyperthyroidism. Surgical procedure was performed with wide margin was performed under GA. The obtained specimen is subjected for histopathological examination which revealed dense fibrocollagenous connective tissue stroma with proliferation of spindle shaped fibroblasts, numerous multinucleated gaint cells are scattered in the connective tissue and is composed of 5-10 nuclei. Proliferating endothelial cells associated with hemorrhagic areas and bony trabeculae rimmed by osteoblasts are evident which suggest Central gaint cell granuloma(fig-5).

Fig 1. Facial profile

Fig 2. Intra oral obliteration of buccal vestibule

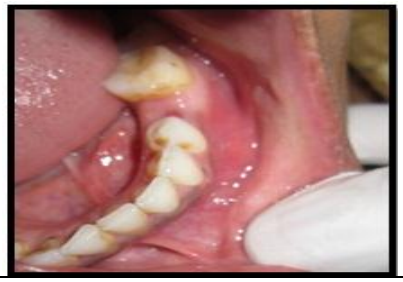


Fig 3. OPG

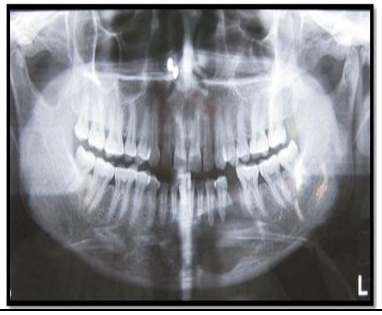


Fig 4. CBCT reveals the thro and thro perforation

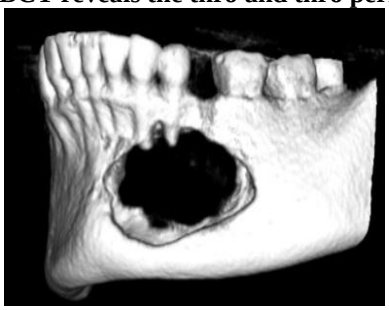
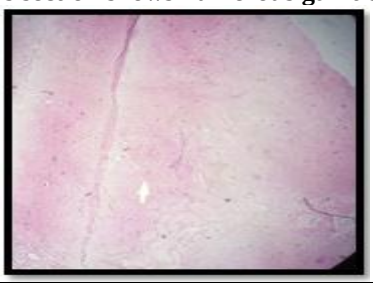


Fig 5. This section shows numerous gaint ells (40X)



DISCUSSION

The central giant cell granuloma, also known as giant cell lesion, was first described by Jaffe in 1953. The WHO has defined central gaint cell granuloma as an intraosseous lesion consisting of cellular fibrous tissue that contains multiple foci of hemorrhage, aggregations of multinucleated giant cells and occasionally trabeculae of woven bone [1]. The lesion is usually seen in children and young adults, with more than 60 percent of all cases occurring before the age of 30 years. There is a distinct female sex predilection, with a female-to-male ratio of 2:1, [5] which may be explained by recent suggestions of the association between hormonal secretion and the appearance of CGCG in Females [6]. Our patient was a female in her 4th decade of life.

The exact process behind pathogenesis of CGCG remains unknown. While the giant cells remains to be the most prominent feature of these lesions, it is actually the mononuclear spindle cell which is the proliferating cell (in cell cycle). This is indicated by the expression of the cell cycle protein Ki-67 in CGCGs. It is believed by some that this spindle cell (fibroblast or fibroblast-like) recruits monocytes from the vascular system and induces them to differentiate into osteoclastic giant cells through release of

cytokines. It has been proposed that this spindle cell takes its origin from the mesenchyme of marrow and an epigenetic event (poorly understood) signals them to release cytokines and finally the osteoclastic giant cell causes bone resorption making the hallmark feature of CGCG.

Another hypothesis is that CGCG is a vascular proliferative lesion, which means that angiogenesis under the influence of the tumour cells is required for tumour growth, invasion, and destruction of local tissue. The possible spontaneous involution theory favours this hypothesis.

The location of CGCG it is commonly noticed in jaw bones and facial bones. In the jaws it is frequently noticed in mandible anterior region which may also crosses the midline.(5) In our patient the lesion was present in mandible premolar region which does not crosses the midline.

CGCG is commonly noticed during routine radiological investigations since in most of the cases it is asymptomatic sometimes associated with swelling. Lesion which causes perforation is considered to be rare and may associate with the pain [7,8] In our case the lesion is aggressive with complete perforation of both buccal and lingual cortical plate and also associated with pain and swelling.

The CGCG usually causes expansion in the cortex when it grows up and in case of enlarged lesion it cause perforation of the bone, tooth mobility, tooth displacement, and also root resorption [9] In our case perforation and root resorption also paraesthesia is seen which is further more rare.

Based on the clinical and radiographic features Choung et al in 1986 and Ficarra et al in 1987 have defined the lesion into two types [10].

1. Non aggressive lesions make up most cases, exhibit few or no symptoms, demonstrate slow growth and do not show cortical perforation or root resorption of teeth involved in the lesion.
2. Aggressive lesions are characterized by pain, rapid growth, cortical perforation, and root resorption. They show a marked tendency to recur after treatment, compared with the nonaggressive types.

The radiographic appearance of this lesion is not always pathognomic and specific, it usually appear as a small lesion with unilocular radiolucent and in rare cases with multilocular with large lesions [11] In our case eventhrough aggressive in nature it appears as unilocular radiolucency.

The management of CGCG for a small lesions include curettage or complete resection with the extraction of the teeth that is involved. Non-surgical treatment modalities include systemic calcitonin therapy and intralesional injection with corticosteroids [12]. The incidence of recurrence after surgery is 4–20 %. In our case a complete excision with curettage was done and patient is currently under follow up for past 8 months.

CONCLUSION

To conclude CGCG with aggressive nature causing bone perforation should be diagnosed in the early stage to reduce the morbidity. Aggressive CGCG lesion should be periodically monitored for the recurrence, since this aggressive type has high recurrence rate.

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CONFLICT OF INTEREST:
The authors declare that they have no conflict of interest.

STATEMENT OF HUMAN AND ANIMAL RIGHTS

All procedures performed in human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

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