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Case Report

Aggressive Central Giant Cell Granuloma: A Rare Case Report

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ABSTRACT

Central giant cell granuloma is a nonneoplastic intraosseous lesion, and constitutes common nonodontogenic pathology in the jaws. Although "central" giant cell granulomas usually occur in the bone, some rare cases behave as extremely aggressive with perforation of the cortices and present themselves as exophytic growth leading to clinical misdiagnosis. Here, we are reporting a very rare case of "aggressive" central giant cell granuloma arising from the anterior mandible as an exophytic growth which resembled a wide variety of conditions leading to misdiagnosis. So, here is an attempt to enlighten the knowledge about such lesions and include them in the differential diagnosis list.

Key words *– Aggressive, giant cell, granuloma*

INTRODUCTION

Central giant cell ganuloma is still not able to be classified as a reactive, inflammatory, infective, hamartomatous, or neoplastic process. [1,2] It was first described by Jaffe in 1953 as an "indolent reparative giant cell granuloma" and branded it to be as 'reparative' in nature and self healing. But, the use of the term reparative has subsequently been discontinued since the lesion represents essentially a destructive processas almost every lesion does not regress without an intervention. [3,4]

Choung et al. (1986) had classified the lesion into two types and the summary of the features of these two types according to the literature review as. [1,2,4-6]

1. Non-aggressive, which is characterized by a slow, almost asymptomatic growth that does not perforate the cortical bone or induce root resorption.
2. Aggressive, which is characterized by pain, rapid growth, paraesthesia, a size of more than 5cm, expansion and/or perforation of the cortical bone and radicular resorption.

Most of the reported cases of CGCG in the literature till now are typically non-aggressive type. So hereby, we are reporting a very rare case of aggressive type of CGCG projecting as a proliferative exophytic growth with resultant perforation of the cortex in the anterior portion of the mandible crossing the midline.

CASE REPORT

A 35 year old female patient reported with the chief complaint of growth in the lower front teeth region since 3 months with a history of trauma 1 year back. It was associated with pain since 15-20 days, difficulty in eating, chewing, swallowing and shedding of two teeth 1½ months back. Her past medical and habit histories were non-contributory. On extra oral examination, a solitary diffuse swelling was noted in the chin area which was approximately 2.5 x 2 cm in diameter, slightly tender and firm with diffuse borders.

On intraoral examination, a solitary diffuse growth was noted in the anterior part of the mandible extending from tooth i.r.t 33 to tooth i.r.t 42, crossing the midline. It was reddish in color, spherical in shape, 5 x 4 cm in diameter, surface was smooth with diffuse borders. On palpation, the growth was tender and bleeding on provocation of the growth was noted. It was soft to firm in consistency and was slightly compressible (Fig. 1). Hard tissue examination revealed missing teeth i.r.t 31 and 41 with displacement of 32 and 42 along with Grade III mobility.

Abbreviations used: -
CGCG – Central Giant Cell Granuloma
IOPA – Intra Oral Peri Apical radiogrpah
i.r.t – in relation to
RANKL - Receptor Activator of Nuclear factor kB Ligand

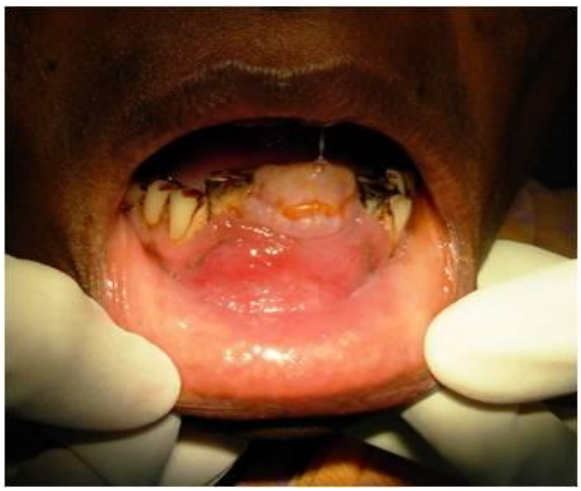

Fig. 1 – Intraoral growth in the anterior mandibular area

Hematological investigation revealed normal blood picture, where as IOPA i.r.t 32 and 42, occlusal and panoramic radiographs showed small thin wispy septa criss-crossing the radiolucent mass and resorption of the roots i.r.t 33,34,43 and 44 (Fig. 2,3 & 4). A clinical diagnosis of central giant cell granuloma was given. Differential diagnosis included peripheral ossifying fibroma, peripheral giant cell granuloma, peripheral ameloblastoma and traumatic or irritational fibroma.

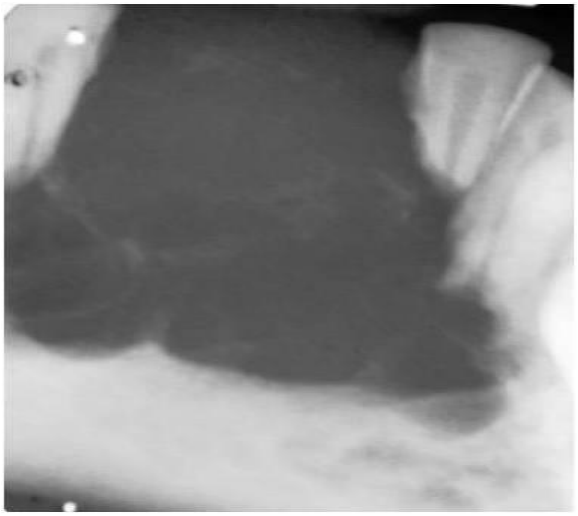

Fig. 2 – IOPA showing small thin wispy septa criss-crossing the radiolucent mass

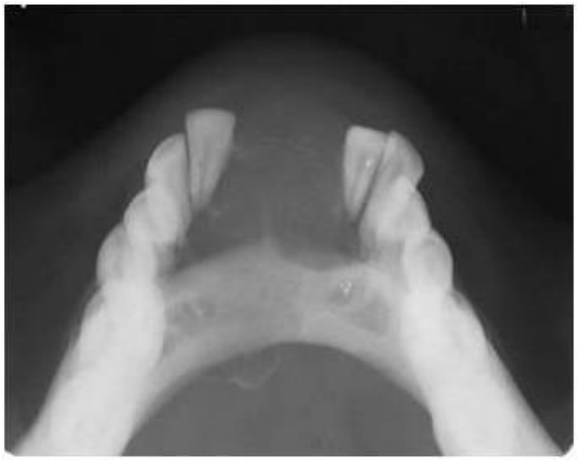

Fig. 3 – Occlusal radiograph showing a multi locular radiolucent lesion with small thin wispy septae within the mass with a corticated border at the periphery

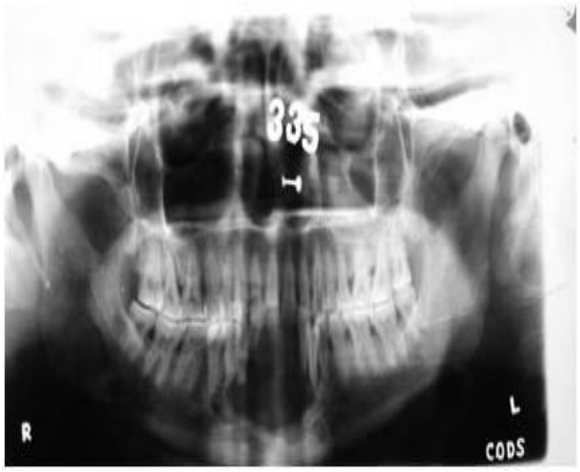

Fig. 4 – Panoramic radiograph showing a well defined radiolucency

Incisional biopsy of the lesion was carried out and the specimen was sent for the histopathological examination, which showed a significantly high number of giant cells and histopathological diagnosis was made as central giant cell granuloma (Fig. 5). So a final diagnosis of aggressive type of central giant cell granuloma was made. Most of the cases of central giant cell granulomas are associated with hyperparathyroidism, especially in older adults. However, in this case although the patient was in middle 40's, it was decided to investigate the patient's serum calcium levels and were found to be within normal limits.

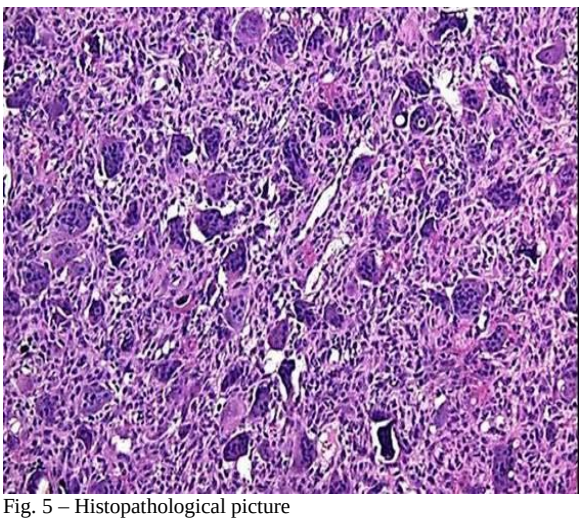
Fig. 5 – Histopathological picture

DISCUSSION

The etiopathogenesis of central giant cell granuloma is still elusive. Many theories have been put forward but none is universally accepted. The most common theory from different authors can be summarized as, [1,3-9,10]

Epigenetic event (which is poorly understood / even probably hemorrhage from a recent trauma)

↓

Results in the formation of mononuclear tumor spindle cells from the mesenchyme of marrow which escape form cell cycle controls

↓

Recruits monocytes from vascular system through cytokines like Monocyte Chemoattranct Protein (MCP)

↓

Capillary monocytes undergoes differentiation through cytokines like RANKL

↓

Through differentiation forms osteoclastic tumor giant cells

↓

Results in bone resorption by secreting lysosomal proteases

↓

Formation of central giant cell granuloma

CGCG typically produces an asymptomatic painless expansion or swelling of the affected jaw. Although in some cases cortical plates are thinned, with sometimes perforation but gross soft tissue involvement is rare as often remains limited to its effects to periosteum. [1] But in this case there was a large exophytic growth with almost involving whole of the anterior part of the mandible which prompted us to think about some of "peripheral" growths initially. So in this aspect this case can be considered as a unique and thus we can

increase our sphere of thinking in such cases.

Radiographic features in this case were typically as that of CGCG. In this case, both in IOPA and occlusal radiographs multilocular radiolucent lesion approximately of the size of 50 mm was noted with small thin wispy septa criss-crossing with noncorticated border along with resorption of teeth roots. These findings were also noted by many of the authors and in this case our radiographic diagnosis was primarily CGCG. [1,8] After all these considerations, a clinical diagnosis of central giant cell granuloma was made.

Aggressive lesions usually demonstrate a higher relative size index (RSI) of giant cells with increased rate of mitosis, larger fractional surface area (FSA), as well as the number of giant cells was also significantly higher as that was in our case [1,4,6] (Fig. 5). Whereas, non-aggressive cases of CGCG shows a minimal to moderate cellularity and a non-vesiculated fibroblast population. [9] So depending on the clinical, radiographic and histopathologic features, a final diagnosis of aggressive type of central giant cell granuloma was made in this case.

Aggressive lesions of CGCG are usually managed by surgery coupled with curettage, peripheral osteotomy, cryotherapy with liquid nitrogen, use of Carnoy's solution, radiotherapy, or postoperative use of interferon- ά. In this case, we had decided to start with intralesional corticosteroids with 2 ml/ 2 cm of radiolucency in multiple locations in a weekly regimen for six weeks. However, we did not notice any change in the size of the lesion after six weeks. But, these injections may stop the further progress without actually decreasing the size of the lesion, as these may cause cessation of bone resorption via the following two mechanisms. [4,6]

1. Inhibition of the extracellular production of lysosomal proteases.

2. Steroidal apoptotic action on osteoclast-like cells.

Once the corticosteroid regimen was over surgery with peripheral osteotomy was carried out. Synthetic human calcitonin, Osteoprotegerin (OPG), Imatinib are some of the other proposed therapies for the medical management of CGCG along with or without surgical options. [1,4,6] Still many research works have to be carried out in the use of above mentioned therapies for their efficiency in treating CGCG.

CONCLUSION

The relatively rare incidence of aggressive type of CGCG's may pose a problem in diagnosis and thus in the proper treatment as well. As these lesions have a high recurrence rate, we need to follow up very frequently to reduce the post treatment morbidity.

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