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NON-AGGRESSIVE CENTRAL GIANT CELL GRANULOMA IN MANDIBLE – A CASE REPORT

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ABSTRACT
Central giant cell granuloma is an uncommon benign proliferative lesion accounting for less than 7% of all benign jaw lesions of unknown etiology, and known to occur in anterior mandible in the first three decades of life with female predilection. Clinical, radiological and histopathological parameters describes Aggressive and Non aggressive lesion with features of high recurrence in the aggressive forms which are found in younger age group. This case report presents a 16 year old female with classical features of non aggressive central giant cell granuloma crossing mid line in the mandible which is extending posteriorly.

KEY WORDS: Giant cell, C-T Scan, Central lesion, Contrast, Non Contrast, Non Aggressive.

INTRODUCTION

Central giant cell granuloma is an uncommon, benign, and proliferative lesion whose etiology is not defined. The W.H.O defines C.G.C.G as an intra osseous lesion consisting of cellular fibrous tissue that contain multiple foci of hemorrhage, aggregations of multinucleated giant cells, and occasionally trabeculae of immature woven bone.[13]

Jaffe first used the term "giant cell reparative granuloma"[13-18] in 1953 to distinguish this lesion of the jaw from the giant cell tumor, histologically similar lesion of the long bones, once thought to be separate entities. It is now believed that these represent the same disease process and shows diversity by the site of occurrence and the age of the patient with a predilection for women.

However giant cell lesions of jaws demonstrate variable clinical behavior and radiological changes. Although most jaw lesions are slow growing, circumscribed that responds to simple curettage. A few cases demonstrate aggressive behavior that is characterized by pain, root resorption, cortical bone destruction and tendency to recur after treatment.[10] Aggressive lesions tend to occur in younger age group with features of rapid growth, pain, perforation of bone, root resorption, and recurrence.[19-7]

The non aggressive lesions are characterized by a slow almost asymptomatic growth that does not perforate the cortical bone or induce root resorption, have low tendency to recur.[22] Both the forms of C.G.C.G exhibit similar histopathological features although aggressive form correlate with large cells and frequency of osteoid bone in the lesion.[10]

With the afore mentioned findings in mind we report a case of non-aggressive C.G.C.G in the mandible crossing the mid line and extending posteriorly with classic clinical, radiographic, and histo patho- logical features.

Case report:

A 16 year old female visited to the dental O.P.D with a progressive painless solitary swelling in the left mandibular region since one month. Extra orally a solitary diffuse swelling in the left mandible measuring approximately 2x2.5 cms extending anteriorly 1.5 cms away from the left commisure, inferiorly to the lower border of the mandible, posteriorly about 2cms in front of the angle of the mandible and superiorly till ala-tragus line. The surface of the swelling was smooth, hard, non-tender with diffuse margins. (Fig.1) Intraorally a diffuse swelling extending from 33 to 36 on buccal aspect measuring about 3x2.5cms with vestibular obliteration i.r.t 33 and 36 (Fig.2). Lingually it was extending prominently from 34, 35, and 36 regions with no associated secondary changes (Fig.3). On palpation it was non tender firm to hard in Consistency with lobulated surface. Associated teeth appeared to be normal but on vitality test (thermal) 34, 35 were non-vital.

The patient was investigated with noninvasive and invasive diagnostic techniques.Radiological examination IOPA revealed the presence of well defined radiolucency (Fig.4).OPG revealed mulitlocular, well defined, corticated radiolucency extending from 33 to mesial aspect of 36. The inferior cortical border was resorbed but continuity was maintained. (Fig.5). Occlusal view of mandible revealed well defined multilocular radiolucency with buccal and lingual cortical plate expansion i.r.t. 33-36 (Fig.6).On the basis of clinical, radiological, examination provisional diagnosis of Myxoma, and differential diagnosis of C.G.C.G, Aneurysmal bone cyst, Ameloblastoma, was made. Non-contrast and Contrast enhanced CT-scan revealed lytic, expansile lesion with multiple hyper dense internal separations of variable thickness involving the anterior mandible on the left side with extension across the symphysis on the right side as well (Fig.7).

Surgically excised soft tissue mass routinely fixed, processed and stained with H&E revealed fibrocellular connective tissue with numerous multinucleated giant cells through out the stroma (Fig-8). Fibrous tissue comprised of collagen fibers with fibroblasts that are spindle shaped having plump nuclei with few areas of thin corticated resorbed bone at periphery of the sections. Giant cells are surrounded by chronic inflammatory cells and extravasated RBCs and areas of granulation tissue. (Fig: 9-10). Quantitative giant cell measurement was performed to assess the aggressiveness of the lesion by FSA (fractional surface area occupied by giant cells) and RSI (relative size index of the giant cells) are calculated by morphometry using IMAGE PRO PLUS soft ware. Mean areas, Mean volume, and average number of nuclei per giant cell were assessed and the results are correlated with the data provided by Auclair et.al.1988[9] and were correlating with the features of non aggressive lesion. With the above findings diagnosis of **NON AGGRESSIVE CENTRAL GIANT CELL GRANULOMA** was made.

Discussion:

The term CGCG was first reported by Jaffe in 1953 and it was the first approach for indicating the clinical and histopathological differences between Giant cell tumor and CGCG of bone.[1] CGCG affects predominantly children and young adults in the first three decades of life with female preponderance[25-26]. According to Bender's, Bar-Ziv J actively developing craniofacial skeleton to accommodate osteogenesis, exfoliation and eruption of teeth these process cease in adulthood and may therefore predispose to CGCG in younger individuals[14]. Csilag.et.al. (1997), Littler BO (1980) explained that occurrence of CGCG is predominant in females due to increased levels of hormonal secretion (estrogen)[3-16].

The site of occurrence for CGCG most commonly in the anterior region with predilection to mandible (66%) compared to maxilla (34%) How ever Kaffe. et.al in their study stated that no site predilection for the anterior region (deciduous tooth bearing area)[13].The present case exhibited above features but extending posteriroly till 36 region.

CGCG manifests clinically as an asymptomatic osteolytic lesion and is usually diagnosed during routine radiographic examination or when facial asymmetry, impaired nasal breathing, the loosening or displacement of teeth[25-1]. Localized swelling is an important clinical feature with smooth surface, palpation reveals a rubbery, elastic sensation where the bone is thin[23]. It grows slowly without expansion to an aggressive painful process accompanied by root resorption, cortical bone destruction and extension into soft tissue.

Variability in the radiographic description of CGCG changes with the size and nature of the lesion [25]. Wood and Goaz claim that CGCG appears as a unilocular radiolucent cyst-like lesion, which changes to a multilocular cyst like lesion with a bubble appearance[18]. Several authors have reported the frequency of mulitlocular cysts appearing in 50%[8], 51%[13], and 61%[10] of patient population. Small lesions usually appear to be unilocular radiolucent and deprived of internal septa. However larger lesions usually appear to be multilocular radiolucent and like wispy bony septa in this area[25]. The present case reveals the features of multilocular radiolucency due to its larger size. But non-contrast and contrast enhanced C-T scan revealed extension of the lesion across symphysis on the right side of the mandible as well. The CGCG is reported to have a low growth index, therefore their borders appear to be distinct and non diffusable[10]. The present case corroborates with the above findings. Based on clinical, radiological, and histopatholigical findings Aggressive and Non Aggressive lesions are classified[15]. Aggressive lesions are characterized by their ability to resorb bone, teeth, and displace anatomical structures such as teeth, the mandibular canal and floor of the maxillary antrum.

Cytometric imaging study proved that clinical criteria used by Choung.R. et. al in diagnosing the

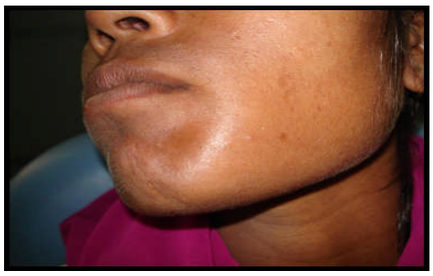

Fig.1.Extra oral view of the swelling on left side of the face.

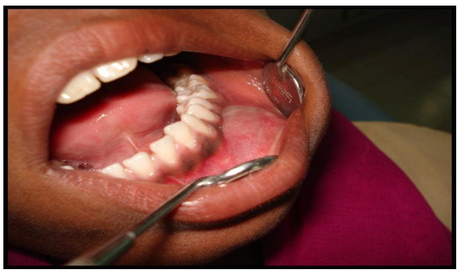

Fig.2.Intra oral view of the swelling extending from 33-36 region on the buccal aspect.

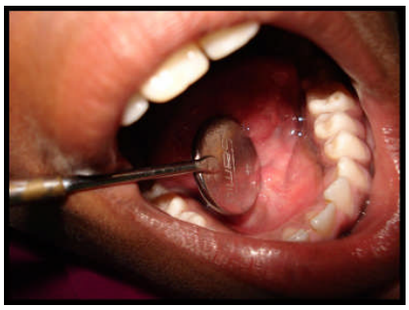

Fig.3.Intra oral view of the swelling extending from 33-36 region on the lingual aspect.

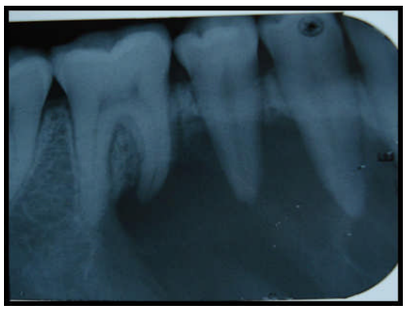

Fig.4. **IOPA radiograph with well defined radiolucency i.r.t 33-36.**

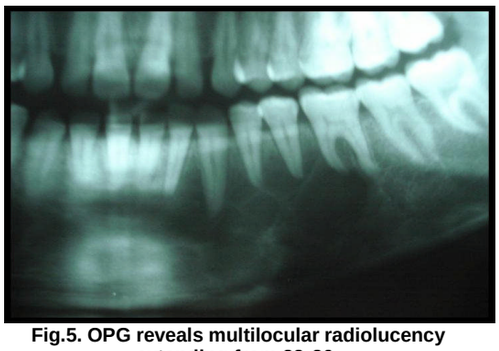

Fig.5. OPG reveals multilocular radiolucency extending from 33-36.

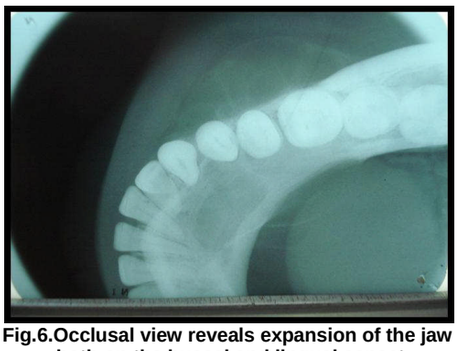

Fig.6.Occlusal view reveals expansion of the jaw both on the buccal and lingual aspect with multilocular appearance.

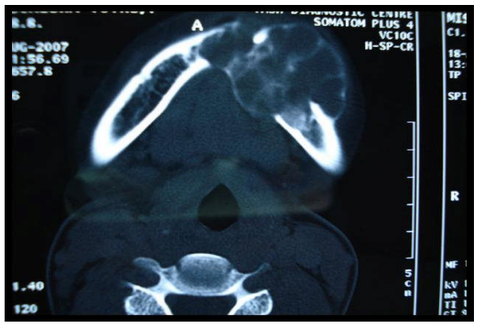

Fig.7. C.T Scan reveals lytic, expansile lesion with multiple hyper dense internal separations of variable thickness involving the anterior mandible on the left side with extension across the symphysis on the right side as well

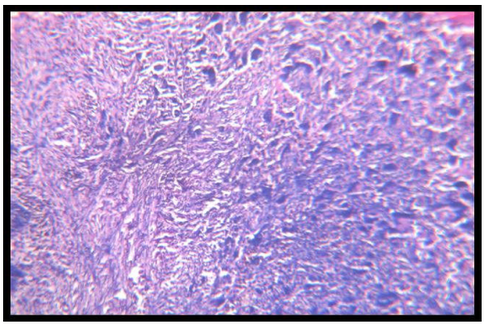

Fig.8.Fibro cellular connective tissue with numerous multinucleated giant cells through out the stroma

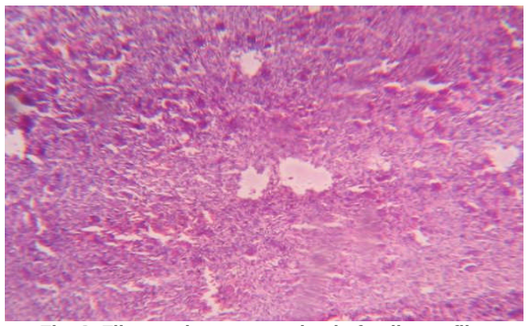

Fig. 9. Fibrous tissue comprised of collagen fibers with fibroblasts that are spindle shaped having plump nuclei

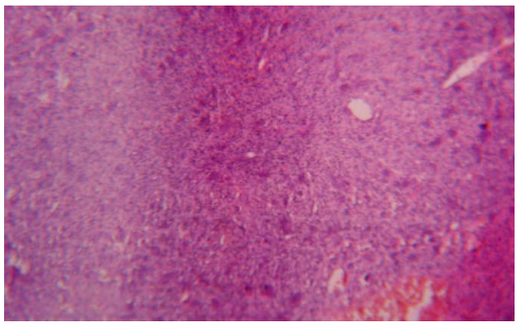

Fig.10. Giant cells are surrounded by chronic inflammatory cells and extravassated RBCs and areas of granulation tissue

aggressive and non aggressive type were reliable not only in determining the extent of tumor but also in showing its rapid growth and destructive behavior. It was concluded that the aggressive type of CGCG was more common in younger age group when diagnosed and had more frequent tendency of recurrence than the Non aggressive type[4]. Franklin et.al has shown that in giant cell granuloma the giant cells are smaller and contain fewer nuclei than those of giant cell tumor of skeleton[2]. Choung.R.et.al (1996) compared aggressive and non aggressive variants of CGCG and found higher FSA (fractional surface area occupied by giant cells) and RSI (relative size index of giant cells) in aggressive lesions. They also observed a prominent web of actin filaments in the aggressive tumors and their absence in the non aggressive lesions under electron microscope suggesting a possible difference between two groups[4]. The present case is in favor of above findings. Itanoga et.al indicated that the giant cells in

CGCG of the jaw are osteoclast like and formed from Monocyte/Macrophage precursors which differentiate into osteoclast[24].Ficcarra et.al used a computer guided image analysis in defining the four histopathological parameters (number of giant cells, mean number of giant cells, fractional surface area and relative size index). These parameters were used for investigating the presumptive initial histological signs and clinical behavior of CGCG[5].

Majority of CGCG of the jaws are generally benign in character, however some showed tendency of recurrence and an aggressive biologic behavior[5-4-8]. The true nature of CGCG remains unknown; despite considerable discussion controversy in the literature reveals various hypotheses for explaining the true nature of the lesion.

It has been proposed that the process represents a reparative response to intrabony hemorrhage and inflammation. Although the clinical progression of these lesions is inconsistent with repair, many investigators regard these lesions as reactive. Other authors view CGCG as a lesion related to the giant cell granuloma of long bones, which is considered to be a true neoplasm. A third theory is that this lesion may represent a developmental anomaly closely related to the aneurysmal bone cyst[12]. Flarggort et.al reported a case of recurrent mandibular CGCG in a patient with SOTO's syndrome (cerebral gigantism) during high dose estrogen therapy. They proposed that while the hormonal therapy of the case has no positive correlation with CGCG but extensively increased levels of estrogen lead to the development of CGCG in the jaws[17].

Traditionally the management of these lesions has been curettage or resection of the central lesion. Recurrences have been associated with this therapy and in case of large lesion, it results in loss of teeth, tooth germs as well as mutilation of the face[17]. Cohen.M.A.Hertzanuy suggested conservative surgical curettage, enucleation plus resection may be necessary in aggressive lesions of extensive destructive type[8].But Eisenbud et.al noted that curettage with peripheral osteotomy was a convenient method for the treatment of giant cell granuloma of the mandible.[7] The present case is treated with simple enucleation with intra lesional hydrocortisone injection, the patient is alright with out any signs of recurrence the patient is under regular follow up.

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