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Central Giant Cell Granuloma of Mandible (Cgcg): Case Report

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INTRODUCTION

Central Giant cell tumors of the jaws are non-odontogenic, benign but aggressively destructive osteolytic lesions. The tumor is not a true granuloma and is not having reparative process. This tumor is histopathologically and behaviourally identical to the benign giant cell tumor of long bones. In the jaws, the tumor presents as a painless, expansile having a short (2 week to 2-month) ascendancy. The expanded lesion may appear blue due to its cortical and mucosal thinning and internal vascularity. The peak range of occurrence is between 5 to 15 years of age, although some cases develop in 20s and 30s as well. Women are affected twice as frequently as men. The mandible is involved three times as frequently as the maxilla. In mandible, it occurs in anterior jaw regions, but the posterior regions are affected as well[1]. This lesion shows occasional extension across the midline.

Central giant cell tumors are not high-pressure vascular lesions as hemangioma. It will either fail to return blood or will return only a small amount. In most cases, an incisional biopsy can be performed. A thoroughly curet the entire lesion if it is small and the access is good. The red-brown friable tissue is curreted out from the region. It is necessary to obtain a serum calcium determination to rule out both primary and secondary hyperparathyroidism. In primary hyperparathyroidism, the brown tumor will evidence hypercalcemia and secondary hyperparathyroidism producing a brown tumor will evidence hypocalcemia.

The primary cells of central gland cell tumors are fibroblasts. Secondary cells are multinucleated giant cells. Accessory cells are also seen in considerable amount which includes macrophages, factor XIIIa, dendrocytes and endothelial cells. Tumor fibroblasts are believed to be responsible for recruitment and retention of monocytes which subsequently transformed into multinucleated giant cells[2]. Cyclin D1 protein overexpression may be involved in the formation of the giant cells and the pathogenesis of central giant cell granuloma[3]. CD68-positive mononuclear cells constituted a small population of cells in all tumors. Most mononuclear cells were positive for fibroblast-associated antigen. No phenotypic differences were detected between aggressive and non-aggressive tumors[4]. Giant cells of bone and soft tissue tumors are reactive cell forms and not of neoplastic origin[5].

Radiographically, the central giant cell tumor will classically present a multilocular radiolucent lesion with severely thinned cortices, including the inferior border. It causes scalloping of the inferior border, displace the teeth and resorb interradicular bone. It also resorbs the roots to some degree. The margins of the lesion are relatively well demarcated. In some cases, central giant cell tumor presents a more aggressive clinical and radiographic picture. These "aggressive" central giant cell tumors may cause pain or paresthesia. They exhibit rapid growth, root resorption, perforation of cortical plate and a higher recurrence rate.

CASE REPORT

A 35 years old female reported to the Department of Oral and Maxillofacial Surgery, of our institution, with the chief complaint of gradually increasing, painless overgrowth in her left posterior lower jaw without knowing for time of evolution (Fig. 1). On clinical examination a firm swelling extending from left mandibular first premolar region to retromolar region of the mandible with expansion of buccal cortical plate and lower border of mandible measuring in size 5x4 cm^2 (Fig. 2). Root stumps of second premolar on the left side and first, second premolars and molars on right side are present. The overlying mucosa was normal in colour and there was no tenderness on palpation.

The OPG revealed a multilocular radioluscent lesion with smooth margins and expansion of lower border of mandible. The radiopaque septa gave the appearance of soup bubbles of different sizes. The root stump of second premolar was present touching the radioluscent lesion. The lesion was extended from lower first premolar to retromolar regions of left side of mandible. Significant thinning of the lower border of mandible in the area of the tumor was evident (Fig.3). Incisional biopsy was taken from the posterior region confirming the diagnosis of giant cell tumor.

Initially we used triamcinolone (kenalog) injection given intralesionally weakly 8 weeks but no change in the size of tumor was observed, thereafter we planned for surgery. The patient was operated under general anaesthesia by Propofol. A standard mucoperiosteal flap extending from symphysis to the posterior border of mandible was raised. Extensive curettage was performed through the exposed bony growth by the roenguers and curettes. The root stump of second premolar was extracted because most of the time the tumor remains at the apex of the tooth involved in the mass. The curettage was done to remove expansile bone and friable bleeding tissue (Fig. 4). Excessive bleeding was encountered during the curettage. This was controlled by putting pressure gauze pack soaked with adrenaline. The bleeding was stopped after the complete removal of the tumor mass. Two units of blood were transfused to the patient to compensate the blood loss during the surgery. The defect was thoroughly irrigated with normal saline. Primary closure was achieved with 3-0 vicryl. The surgical specimen was sent for histopathological examination and the diagnosis was confirmed as central giant cell tumor. The patient has been followed up for one month with no evidence of recurrence (Fig. 6 & 7).

DISCUSSION

Central giant cell tumor of jaw is a non-odontogenic benign aggressively expansile lesion. These lesions are osteoclastic precursors because they develop the ruffled borders typical of osteoclasts. The tumor is benign although aggressive and sometimes quite large. The occurrence of central giant cell tumor is common in young patients. In our case the patient was of 35 years of age which is coinciding with the incidence of the lesion. The most frequent involvement is the mandible occurring in the posterior region. The occurrence in our case is the mandible

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involving the retromolar region. In the jaws the central giant cell tumor presents as a painless, non tender on palpation and patient notices only a swelling. The patient is clinically asymptomatic and complains only about the expansion of bone causing disfigurement of face. The radiographic examination reveals multilocular with soap bubble appearance. There is thinning of cortical plate with involvement of the lower border of the mandible. No midline cross-over was observed in our case. Incisional biopsy is mandatory to confirm the diagnosis whether the tumor is benign or malignant. Differential diagnosis is suggestive of several lesions having multilocular, expansile, radiolucent lesion in young patients such as an odontogenic keratocyst, an odontogenic myxoema, an ameloblastic fibroma, ossifying fibroma and adenomatoid odontogenic tumor. Because of the bleeding potential and generally young age of presentation, as well as a multilocular "soap bubble" radiolucency, a central arteriovenous hemangioma must be considered. Other lesions containing multinucleated giant cells include aneurismal bone cyst and cherubism. The aneurismal bone cyst is diagnosed by the identification of sinusoidal blood spaces within the tumor mass. Cherubism is diagnosed on historical, clinical and pathological grounds.

The treatment of central giant cell tumor is a thorough curettage of the bony cavity. The lesion itself is confined to and requires bone for its existence. The central giant cell lesion does have a recurrence potential. Recurrences are seen more frequently with larger lesions and those that involve significant numbers of teeth. These recurrences are related to incomplete removal of a friable, bleeding lesion, which is more difficult to remove from between the teeth and furcations. The weak points in curettage are the areas between the teeth, and the neurovascular bundle areas. Additionally, the vascular nature of this lesion, which produces an oozing type of blood loss, obscures the clinician's view. To reduce bleeding the following approaches were taken in our procedure, intraoperative reduction of local blood pressure by the anesthetic technique, use of local vasoconstrictor (adrenaline) pack and transfusion of blood.

Lesions with aggressive clinical features also exhibit a tendency to recur, necessitating more extensive surgical approaches. Intralesional injections of corticosteroids (triamcinolone) have been reported as a non-surgical method for management of these lesions. The protocol that has been suggested is a 50/50 mixture of 2% lidocaine with 1:100,000 epinephrine with triamcinolone (Kenalog) and to inject 2 ml/1 cm of lesion as seen on a Panorex X-ray and to repeat this six times at weekly intervals. Experience with this technique is limited, but it does appear to work more successfully in unilocular lesions than multilocular lesions, and this is probably because of the ease of access in a unilocular lesion, whereas in a multilocular lesion some areas may be missed. In the hands of those who use this technique on a regular basis, it appears that it is successful in around 50% of cases [9,10,11] but there is an appreciable failure rate The use of exogenous calcitonin may also have some merit in the treatment of aggressive lesions[2]. Recently use of interferon alfa has been reported as an additional treatment modality on the basis of an antiangiogenic mode of action [3].

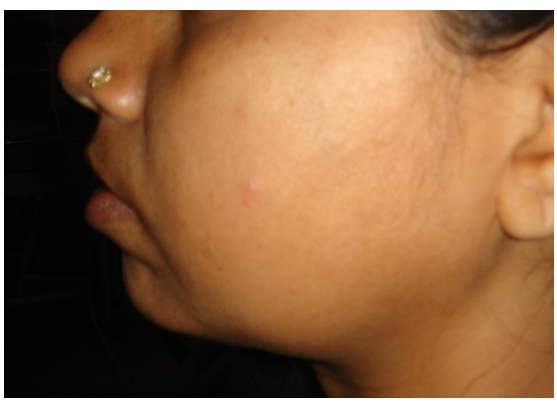

Fig 1 : Showing preoperative side profile of the patient

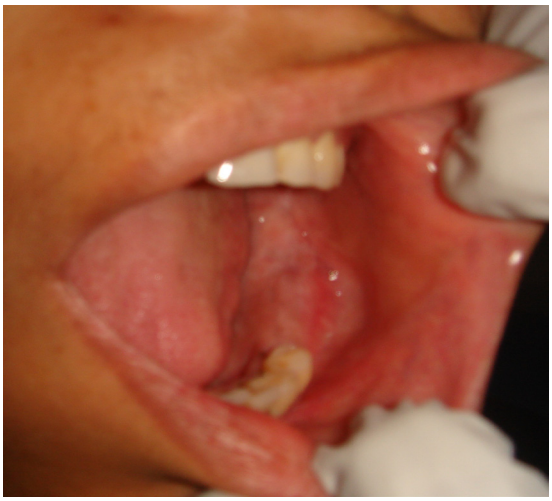

Fig 2 : Showing intra-oral expansion of cortical plate

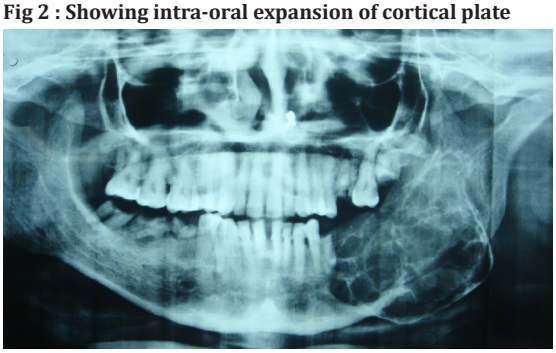

Fig 3 : OPG showing multilocular appearance of lesion on left posterior region of mandible

Fig 4 : Showing bony trabecular mass after curettage

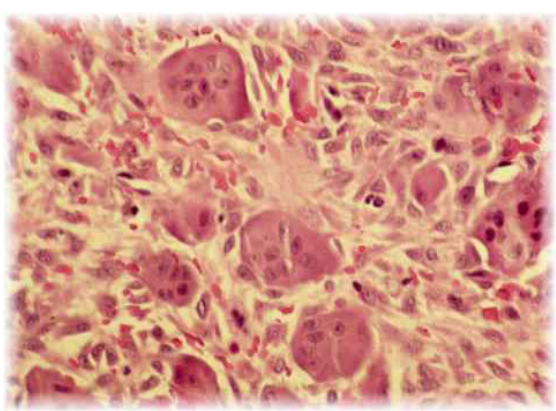

Fig 5 : Histopathological findings of the tissue showing multinucleated Giant cells

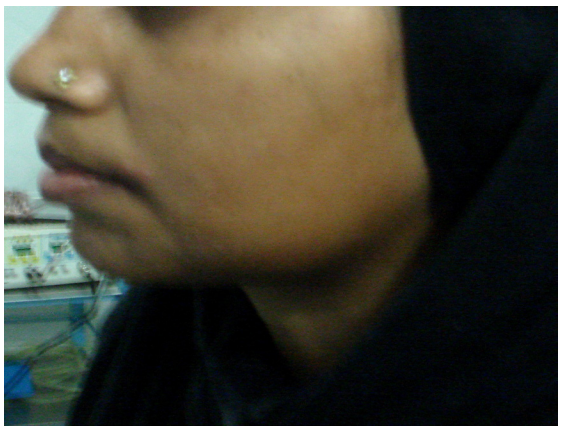

Fig 6``: Showing post-operative side profile after 1 year

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