



CASE REPORT

CENTRAL GIANT CELL GRANULOMA : A CASE REPORT

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ABSTRACT:

Central giant cell granuloma (CGCG) was first described by Jaffe in 1953. It is widely considered to be a non-neoplastic lesion. It is an intra-osseous lesion consisting of cellular fibrosis tissue containing multiple foci of haemorrhage, multinucleated giant cells and trabecules of woven bone. This lesion accounts for <7% of all benign jaw tumors. It has been reported that this lesion is diagnosed during the first two decades of life in approximately 48% of cases, and 60% of cases are evident before the age of 30. It is considerably more common in the mandible than in the maxilla. Radiographically, majority of CGCG(87.5%) present as an expansile radiolucency, either unilocular or multilocular which is generally traversed by bony spicules. The common therapy is aggressive curettage, peripheral ostectomy or resection, which may be associated with loss of teeth and, in younger patients, loss of dental germs. A number of alternative nonsurgical approaches have been advocated in recent years for the management of CGCGs. These include intralesional corticosteroid injections, calcitonin injections and subcutaneous -interferon injections. This paper presents a case of CGCG in a 12 year old male involving the mandibular anterior region with clinical, radiological, histopathological and surgical aspect of the lesion.

KEYWORDS :- Osteoclastoma, myeloid sarcoma, chronic hemorrhagic osteomyelitis, giant cell reparative granuloma.

INTRODUCTION

Central giant cell granuloma (CGCG) was first described by Jaffe in 1953. It is an uncommon, benign and proliferative non-neoplastic process. Jaffe considered it as a locally reparative reaction of bone, which can be possibly due to either an inflammatory response, hemorrhage or local trauma.¹ It is a localized osteolytic lesion with varied biologic behaviour of aggression which affects the jaw bones. The etiology of CGCG is unknown, but some indications implicate genetic abnormalities. The mandible is twice as likely to be involved as the maxillary. Approximately 10% of all benign lesions in the mandible are due to CGCG. Over 60% of the cases described in the literature occur in patients under 30 years of age, although CGCG may also develop in children and the elderly. Females are more frequently affected than males.² Radiographically, majority of CGCG(87.5%) present as an expansile radiolucency, either unilocular or multilocular which is generally traversed by bony spicules.



Central Giant Cell Granuloma has been classified on the basis of radiographic and clinical features.

1. Aggressive lesion: They are found in young patients characterized by rapid growth, pain, expansion and/or perforation of cortical bone, induce root resorption and high recurrence rate.
2. Non-aggressive lesion: It is characterized by slow growth that does not perforate the cortical bone or induce root resorption and has low recurrence rate.³

Histologically, this lesion is characterized by the presence of numerous multinucleated giant cells in a fibro-vascular stroma, hemorrhagic foci with hemosiderin pigments, and occasionally areas of osteoid tissue.³

CGCG can be treated with several therapies, including surgical excision, simple curettage, and en bloc resection as well as intralesional corticosteroid injections, calcitonin intradermal injection or nasal spray, and alpha-interferon -2a (IFN- 2 a) injections. All these treatments have had varying success.²

The purpose of this case report is to understand the diagnostic challenge that CGCG presents in the dental clinic as well as the surgical challenge in the treatment of CGCG.

CASE REPORT :-

A 12-year-old male reported to our Department of Oral Medicine and Radiology, Maitri College of Dentistry and Research Centre, Durg with a chief complaint of pain and swelling in lower right back region of jaw since 2 months. It started as a small painless swelling in right mandibular canine region which is associated with intermittent pain and gradually progressed to present size causing facial disfigurement evident at the time of presentation.. There was no history of trauma, neurological deficit, fever, loss of appetite, loss of weight. There was no similar swelling present in any parts of the body. Patient was systemically healthy. The prenatal history was unremarkable and delivery was at full term and normal. There was no history of similar disease in any of the siblings or the parents of the affected child. A physical examination revealed a moderately built and nourished male.

On extraoral examination a single, diffuse swelling was seen on the right side of the face in the region of anterior mandible. The swelling measured about 3X4 cms in dimensions. The surface of the swelling was smooth and extended antero-posteriorly, from a point 3 cm from the symphysis region to the point 4 cm from the right angle of mandible region and superio-inferiorly, at the level of corner of mouth to the inferior border of mandible. The overlying skin appeared normal, while the swelling itself was bony hard and non tender to palpation (Fig-1).



Fig-1 : Extraoral swelling is seen on right side of face

Intra oral examination showed a diffuse enlargement in the alveolar portion of the right mandibular teeth # 43, 44, 45 region with obliteration of the buccal vestibule in the same region. Tooth displacement and mobility were also evident in the same quadrant. The adjacent dentition and the oral mucosa did not reveal any abnormality. A tooth vitality test revealed normal pulpal response of the teeth in the same quadrant. A provisional diagnosis of central giant cell granuloma was made with the differential diagnosis of ameloblastoma, odontogenic myxoma and fibrous dysplasia (Fig-2).



Fig-2 : Intraoral swelling is seen involving 43, 44 and 45 region

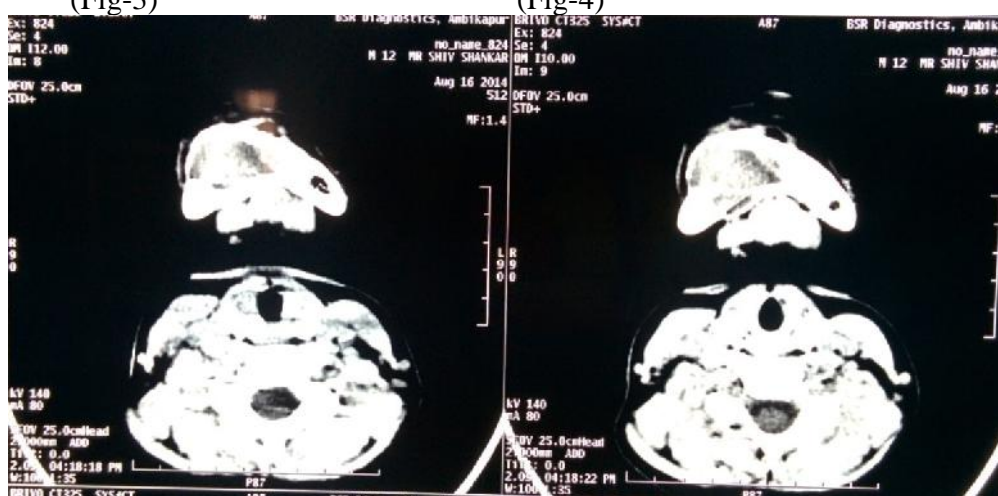
The radiographic examination with an Orthopantomogram revealed a well-defined, expansile, unilocular radiolucency with varying degrees of expansion of the cortical plates occupying the area of right ramus of mandible (Fig-3). C.T. scan revealed a large unilocular radiolucent lesion with well defined margins with the interspersed septae within the lesion on the right side. Radiographic appearance of the lesion is not pathognomic and may be confused with that of many other lesions of the jaws (Fig-4,5).



(Fig-3)



(Fig-4)



(Fig-5)

An incisional biopsy was planned and performed (Fig-6a,6b) which revealed a connective tissue made up of mature collagen fibres, fibroblasts and showing numerous multinucleated giant cells with foci of osseous structures (Fig-7a,7b). On the basis of histopathological and radiological findings, a diagnosis central giant cell granuloma was established.



Fig-6a : Site of Incisional biopsy



Fig-6b : Collected specimen

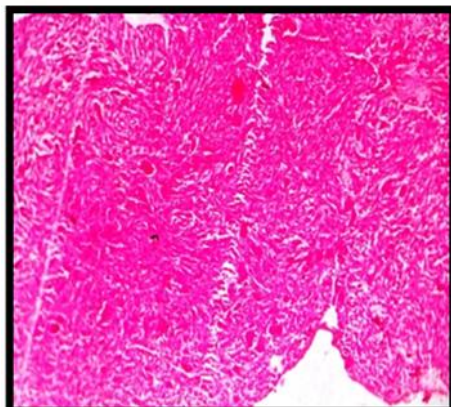


Fig-7a : Low magnification view

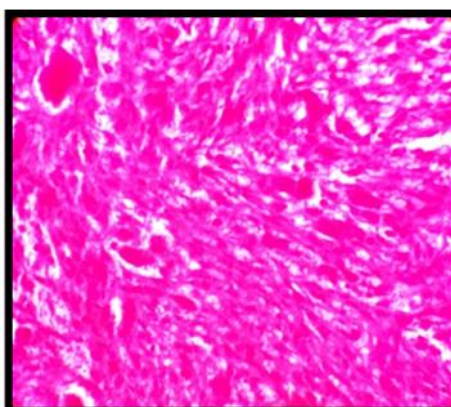


Fig-7b : High magnification view

For the present case, treatment protocol was surgical resection which is performed somewhere else under supervision of an oral surgeon (Fig-8). However, post surgical follow up is recorded by us which shows radiographically that teeth 43 and 44 were extracted with preservation of continuity of the mandible with at least a 3 mm margin (Fig-9).



(Fig-8)



(Fig-9)

DISCUSSION :-

CGCG is an intra osseous lesion which occurs predominantly in teens and adults. 60 to 70% of cases are diagnosed in patients younger than 30 year old. In the jaws, lesion develops in the mandible more frequently than maxilla. Sometimes these lesion tends to cross the midline. Females are affected more frequently than males.⁴

The **World Health Organization** has defined it 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”.⁵

The etiopathogenesis of the CGCG of jawbones has not been clearly established but it has been suggested that it is the result of an exacerbated reparative process related to previous trauma and intraosseous haemorrhage that triggers the reactive granulomatous process.⁶ However, because of its unpredictable and occasionally aggressive behaviour, and because of its possible relationship to the giant cell tumor of long bones, CGCG is best classified as a benign neoplasm.⁵



The clinical behavior of the lesion varies from an asymptomatic osteolytic lesion that grows slowly without expansion, to an aggressive, painful process accompanied by root resorption, cortical bone destruction, and extension into the soft tissues. In the past, lesions were classified as aggressive or non-aggressive, based on their clinical and radiological behavior. Aggressive lesions are characterized by their ability to destroy bone, resorb teeth, and displace anatomical structures, such as teeth, the mandibular canal, and the floor of maxillary antrum.⁷

Radiographically, CGCG presents as radiolucent defect, which may be unilocular or multilocular. The defect usually is well-circumscribed and, in some cases, displacement of teeth can be found. Central giant cell granuloma is expansive in its growth; it does not grow around or invade nerve trunks. It also does not invade perineural sheaths or spread via perineural spaces. Histologically, CGCG contain focal arrangements of giant cells within a vascular stroma with thin-walled capillaries adjacent to the giant cells. There is a spindle cell stroma which may well be the cell of origin.⁸ The radiographic appearance is indistinguishable from that of odontogenic cyst, aneurysmal bone cyst(ABC), ameloblastoma, odontogenic myxoma, odontogenic fibroma.⁹

Histologically, CGCG shows cellular fibrous tissue containing multiple foci of hemorrhage, aggregations of multinucleated giant cells and occasional trabeculae of bone. Numerous lesions as cherubism, fibrous dysplasia, primary and secondary hyperparathyroidism (brown tumor), ABC and Giant cell tumour should be considered in differential diagnosis. GCT is distinctly unusual in the jaw; moreover, giant cells are regularly and uniformly distributed in GCT, while they are clumped in areas separated by virtually devoid areas in CGCG. Fibrous dysplasia can be excluded by the presence of Chinese figure-like trabeculae of woven or immature bone within a proliferating fibroplastic

stroma. Aneurysmal bone cysts show large sinusoidal spaces filled by blood. Both histologic and radiographic similarity has been reported in brown tumors and CGCG, but normal serum levels of calcium, phosphorus, alkaline phosphatase and good renal function help in diagnosis of CGCG and excluding the condition of hyperparathyroidism. Cherubism (hereditary and intraosseous bilateral swellings of the jaw) is also microscopically indistinguishable from CGCG, but its usually bilateral presentation, in a young individual with a hereditary autosomal dominant mode, allows its recognition.⁹

Provisional diagnosis of CGCG can be made with the history of patient, clinical and radiographic investigation. However, definitive diagnosis can only be established with combination of all the above as well as histopathological and biochemical investigations.¹⁰

The conventional therapy of CGCG has been local curettage, and this has been associated with a high success rate and a low recurrence rate. Consequently, a number of different treatment options have been recommended, including aggressive curettage possibly coupled with adjunctive treatment such as liquid nitrogen cryotherapy, Carnoy's solution and gold standard for therapy en bloc resection with negative histologic margins, which result in a major facial deformity. This is of great concern, especially in children and young adults with developing dentition and jaws. A number of alternative nonsurgical therapies have been described for the management of CGCG. Radiotherapy has not proven to be a satisfactory alternative, because irradiation of giant cells lesions may provoke malignant degradation. Other alternative treatments include pharmacologic therapy with α -interferon or calcitonin, which are administrated via subcutaneous injection over several months.¹¹



The use of calcitonin was proposed in 1993 by Harris, based on the similarity that exists between CGCG and the tumours of the hyperparathyroidism at histological level (Harris 1993). Although the calcitonin's mechanism of action remains unclear, it is suggested that it has a direct inhibitory effect of the osseous reabsorption through the osteoclasts, increasing the absorption of calcium.¹² Calcitonin and interferon therapy are complicated owing to the great amount of discomfort, possible side effects and the relatively long duration of treatment, which is not well tolerated by some patients, especially children.¹¹

Intralesional corticosteroid injection is another alternative treatment method which is simple and inexpensive and, most important, saves the vital structures, thus avoiding a large facial deformity.¹¹ The accepted clinical protocol for corticosteroid administration is weekly intralesional injections for 6 weeks, although a successful outcome was recently reported for a case in which combined systemic and local, intralesional glucocorticoids were used.¹³

CONCLUSION :-

The current case thus concludes that the diagnosis of these lesions at an early stage will make the treatment simple and more conservative; it can also avoid spread of lesions into inaccessible regions, radical surgeries, and facial disfigurement. The present case report emphasizes the need for a thorough assessment of any case in clinical practice to prevent morbidity due to late and/or false diagnosis.

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