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# **CASE STUDY**

## **CENTRAL GIANT CELL GRANULOMA IN A 10 YEAR OLD CHILD – A CASE REPORT**

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#### ABSTRACT

**Background:** Central giant cell granuloma is 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.

Aims: This is a case report of a 10 year old male who was diagnosed with central giant cell grauloma that had deviated the path of eruption of permanent canine.

**Materials and Methods:** Clinical, radiographic, aspiration and histological investigations were done to make the diagnosis for the lesion. Enucleation of the lesion was done under local anaesthesia and the biopsy report confirmed the lesion to be a central giant cell granuloma (CGCG). The lesion healed both clinically and radiologically with no report of reoccurrence.

**Results and Conclusion:** The clinician needs to be aware of possible oral pathology when unexpected tooth mobility, displacement, delayed eruption, early exfoliation or various other pathological signs and symptoms are present.

Key Words: Central giant cell Grauloma, Fine needle Aspiration cytology, Enucleation, Biopsy.

#### **INTRODUCTION**

Central giant cell granuloma (CGCG) is defined by the World Health Organization (WHO) as an intraosseous lesion consisting of cellular fibrous tissue that contains multiple foci of hemorrhage, aggregation of multinucleated giant cells and occasionally trabeculae of woven bone (Kaffe et al., 1996). It is a relatively uncommon benign bony lesion of a variably aggressive nature, accounting for less than 7% of all benign jaw lesions (Potter et al., 1993). The condition was first identified in 1953 by Jaffe who initially termed this lesion as a Central giant cell reparative granuloma. Currently the term 'reparative' is not used for description because of the destructive nature of the giant cell granuloma. The true nature of this lesion is controversial and remains unknown but the competing theories are that it could be a reactive lesion, a developmental anomaly, genetic mutation or a benign neoplasm. The mandibular / maxillary ratio has been variously reported in literature ranging from 2:1 to 3:1 mostly affecting the anterior portion of the mandible (Whitaler et al., 1993). It commonly occurs in children and young adults with a slight predilection for females. Based on the clinical behavior and radiographic features CGCG is classified as following:

- 1. *Aggressive lesion:* They are found in young patient characterized by rapid growth, pain, expansion and/or perforation of cortical bone, root resorption and high recurrence rate (Amar *et al.*, 2008).
- 2. *Nonaggressive lesion:* It is characterized by slow growth that does not perforate the cortical bone or induce root resorption and has a low recurrence rate.

\*Corresponding author: Dr. Meenakshi Bodh Department of Pedodontics and Preventive Dentistry, Post Graduate Institute of Dental Sciences, Rohtak-124001 (Haryana) This case report presents a 10 year old male with central giant cell granuloma, where the lesion had displaced the erupting canine but timely management healed the lesion completely and also redirected the path of eruption of canine.

#### **Case History**

A 10-year-old boy presented to the department of Pedodontics and preventive dentistry, PGIDS, Rohtak, with the chief complaint of painless swelling in the left side of the lower jaw since 4 months. Swelling was initially small but gradually increased to reach the present size. According to his mother, the mandibular right and left primary canines exfoliated 6 months back and the right permanent counterpart erupted uneventfully but on the left side the swelling continued to enlarge and permanent tooth had not yet erupted. There was no history of fever, pain, sensory or motor disturbance, bad taste or traumatic injury.



Figure. 1. A preoperative photograph showing swelling over the left mandibular area



Figure. 2. An OPG revealing irregular monolocular radiolucency in the left mandibular canine and premolar region with a maximum diameter of about 3 cm and the canine displaced inferiorly and anteriorly

His medical and family history was insignificant. Extra oral examination revealed a diffuse swelling on the left side of lower jaw extending almost up to the midline. The swelling had no localized elevation of temperature and no associated lymphadenopathy. The overlying skin appeared normal. Intra oral examination revealed a swelling of a size approximating 3X2 cm on both buccal and lingual aspect, extending from lower left lateral incisor to the left second premolar causing obliteration of the buccal sulcus (figure1). The swollen area was hard in consistency. Both mandibular lateral incisors and left mandibular canine were missing.



Figure. 3. Mandibular occlusal radiography revealing buccal expansion of cortical bone

Panaromic radiography revealed well defined monolocular radiolucency without any sclerotic margin situated in the left mandibular canine and premolar region with a maximum diameter of about 3 cm that had displaced the erupting left lower canine inferiorly and anteriorly (Figure2). Also, the mandibular incisors were found to be congenitally absent. Mandibular occlusal radiography revealed buccal expansion of cortical bone (figure 3). Laboratory values for serum calcium, phosphorous, alkaline phosphatase and parathyroid hormone were within normal limits, which ruled out the suspicion of any related systemic condition. Fine needle aspiration cytology of the lesion was done and microscopic examination revealed presence of cholestol crystals and diagnosed it as inflammatory lesion.



Figure. 4. Enucleation of the lesion

Enucleation under local anaesthesia was planned because of the size of the lesion. A sharp probe was used to mark out the extent of the bony defect; incisions were subsequently made at least 1 cm away from the margins of the bony defect. After reflection of the soft tissue a fissured bur was used to cut the cortical bone around the lesion approximately 0.5 cm from its margin. The lesion was reflected in toto with the associated tissues and was removed completely (figure4).



Figure. 5. Histopathologic image of biopsy specimen showing giant cells (H&E stain, 10x, 4x, 40x magnification)

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The specimen was then sent for biopsy which revealed cellular vascular connective tissue with a proliferation of osteoclast type giant cells with multiple nuclei granulation tissue rich in mononuclear inflammatory cells and hemosiderin pigments (figure 5). All the findings clinically, radiographically and histologically were consistent with those of CGCG. The patient was kept on regular follow up and showed complete healing both clinically and radiographically (figure 6,7,8) .The displaced lower left canine was also found to follow the normal path of eruption after removal of the lesion in a time period of 3 months (figure 9,10) .A post-operative X-ray was taken at an interval of 3 month and 6 months and it showed complete healing.



Figure 6: A postoperative intraoral photograph



Figure 7: A postoperative mandibular occlusal radiograph at 3 months.



Figure 8: A postoperative view of OPG at 3 months.



Figure. 9. A postoperative mandibular occlusal radiograph at 6 months.



Figure. 10. A postoperative view of OPG at 6 months.

### DISCUSSION

WHO classified CGCG as a bone-related lesion, not a tumour, although its clinical behaviour and radiographic features often are those associated with a benign tumour. It mostly exists as an asymptomatic lesion that is often discovered during routine radiographic examination<sup>5</sup>. CGCG is categorized into aggressive and non-aggressive types based on their clinical and radiographic characteristics. The more common, nonaggressive, lesions grow slowly and usually presented clinically as painless swellings, with only 20% of patients complaining of pain or parasthesia (Richard et al., 2008). The present case report falls under non aggressive form of CGCG as here the child presented a slow painless expansion of the affected bone. The anterior part of the mandible has been identified as the most common location for CGCG of the jaw and is usually unifocal. In children with mixed dentition, a pathologic lesion may be the underlying cause of regular tooth mobility and exfoliation of primary teeth and can easily be overlooked. The clinician needs to be aware of any possible oral pathology when signs such as tooth mobility, displacement, early exfoliation or delayed eruption, are evident. The CGCG may occur at any age but it is most commonly seen in the first 3 decades predominantly affecting females, however, the present case describes CGCG in a young boy.

There is no definite etiology to CGCG although proposed three probable theories are:

- 1. Response to previous traumatic or inflammatory episode.
- 2. A true neoplastic process.
- 3. A developmental anomaly closely related to aneurysmal bone cyst.

However in this case no such significant association could be identified. Radiographically they appear as well-defined unilocular or multilocular radiolucencies with undulating borders. Aggressive lesions can be differentiated from the non aggressive lesions as the former lesions grow quickly and are associated with pain, cortical perforation and root resorption with ill-defined borders whereas the latter exhibit a slow growth rate, do not exhibit root perforation or cortical perforation, and often show new bone formation. Giant cell granuloma usually destroys the lamina dura and causes roots resorption and displacement of teeth. In an attempt to distinguish aggressive and nonaggressive subtypes of CGCG and to predict the prognosis of newly diagnosed CGCGs, numerous studies have been conducted using cytometric and immunocytochemical methods. It has been shown that aggressive types have a higher number and relative size index of giant cells and a greater fractional surface area occupied by giant cells.

In paediatric patients, especially in the case of mixed dentition, the superimposition of anatomical structures, frequently occurs complicating the radiographic analysis, and it can delay diagnosis and aggravate the case (Bonder et al., 1996). Cone Beam Computed Tomography (CBCT) is the modality of choice to evaluate the location and the extension of the lesion to its surrounding structures especially in cases of large lesions as they tend to destroy the outer cortex. . CT imaging aids surgical treatment planning by demonstrating the extent of the lesion, cortical expansion and bony destruction. Histologically, the features of CGCG are indistinguishable from the brown tumor of hyperparathyroidism and from giant cell lesions of genetic disorders such as cherubism, Noonan syndrome and neurofibromatosis. Microscopic examination of giant cell granulomas shows numerous multinucleated giant cells and mononuclear cells (fibroblast and histiocyte-like cells and monocyte-macrophages) within a prominent fibrous stroma. There is evidence that these giant cells represent osteoclasts (bone-eating cells); others suggest they are more like macrophages. The giant cells may be diffusely located throughout the lesion or focally aggregate in the lesion. The giant cells are typically either large and round, or small and irregular, and can vary greatly in size and shape. Close examination may reveal some hemosiderin deposits as well. It is not uncommon for malignant or benign lesions of jaw to contain multinucleated giant cells as osteogenic carcinoma, fibrosarcoma, malignant tumour of giant cells and benign lesions as fibrous dysplasia, ossifying fibroma, cementifying fibroma, aneursymal bone cyst, brown tumour of hyperthyroidism and the gaint cell tumour, so they must be considered for 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 GCRG. Fibrous dysplasia is characterized by the presence of Chinese figure-like trabeculae of woven or immature bone within a proliferating fibroblastic

stroma. Aneurysmal bone cysts show large spaces filled by blood. These lesions were thus excluded by histology. This case however revealed no association with systemic and genetic diseases. Curettage or enucleation can be chosen as treatment for the CGCG, it is usually employed for smaller lesions of CGCG, as performed in our case. This therapy, however, is associated with recurrence, which in case of extensive lesions, results in a serious mutilation of the jaw and face (Lange et al., 1999). Terry et al. (1994) suggested that aggressive tumors that present with pain, rapid growth and facial swelling or cortical perforation can be treated with enbloc resection. For aggressive lesions, en bloc surgical resection is occasionally used. Surgical treatment of CGCGs can be associated with recurrence and serious facial mutilation and loss of teeth and tooth germs are also unavoidable. Recently weekly intraregional corticosteroid injections, daily subcutaneous injection of Calcitonin and the use of Interferon alpha have also been suggested as possible treatments for multiple or large lesions to avoid the need for mutilating surgery in growing children (Kurtz et al., 2001). The main drawback to these nonsurgical approaches is the need for continual treatment over a prolonged time period. Radiation treatment is contraindicated because of the potential for malignant transformation .However, in this case considering the behaviour, location and size of lesion enucleation was done and it showed satisfactory healing and no reoccurrence has been noted till date. In children with mixed dentition, a pathologic lesion could be the underlying cause of abnormal tooth mobility, early exfoliation of primary teeth or delayed eruption and can easily be overlooked; especially in cases that are not accompanied by an obvious bony expansion. The clinician should suspect any possible oral pathology when such abnormal disturbances are present so that future complications can be avoided.

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