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# Case Report

## Aggressive central giant cell granuloma of mandible in a 3-year-old child: a rare case report

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***ABSTRACT:***

**Introduction:** Central giant cell granuloma, formerly called giant cell reparative granuloma is a non-neoplastic proliferative lesion. CGCG is found predominantly in children and young adults, most commonly in the anterior portion of the mandible. Although many theories have been proposed to explain the aetiology and pathogenesis of CGCG, its true nature is still unknown. **Materials and Methods**: This paper presents the case of a 3-year-old boy with aggressive form of central giant cell granuloma in posterior region of mandible which was treated with intra lesional corticosteroid injections followed by surgical curettage. **Results:** The clinical and radiographic 1-year follow-up did not reveal any recurrence of the lesion. **Conclusion:** The early and precise diagnosis of CGCG allows conservative management without risks for the adjacent teeth or bone and can greatly improve long term outcomes.

**Keywords:** Central giant cell granuloma, curettage, corticosteroid therapy.

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### INTRODUCTION

Central giant cell tumours (CGCTs) are uncommon benign bone tumors in the jaw whose aetiology is usually unknown. 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. [1]Central giant cell granuloma (CGCG) is a locally aggressive form and can change into malignant CGCT with the risk of metastasis. It accounts for 7% of total benign tumours of jaws.[2]The incidence shows it is most seen in younger population with a predilection for females and is most often seen in posterior mandible.

CGCGs have varying microscopic patterns that range from vascular to fibrotic to myxoid stroma. The dominant stromal cells are fibroblastic cells. The giant cells in CGCGs are CD68-positive and vary in size, shape, and number.[3]

The curative treatment for these tumors is surgical curettage or resection, undesirable damage of the teeth or tooth germs is unavoidable, and surgical removal may lead to rupture of the cyst or iatrogenic fractures. The local recurrence rate of such lesions is as high as 20%. [4]

### CASE PRESENTATION

A 3-year-old male patient reported to the department of pediatric dentistry with the chief complaint of swelling in the right back region of lower jaw. The swelling had remained asymptomatic while gradually enlarging and was evident at the time of presentation. There was no history of trauma nor any systemic or local infections. The prenatal history was normal, and delivery was full term and normal. General examination revealed a moderately built boy with no known systemic disorder. A facial asymmetry due to a poorly defined swelling at the right side of the mandible was noted. Intra oral examination revealed bluish swelling on mandibular right posterior region with obliteration of vestibule in same region. Clinical examination revealed that 84 was missing and the swelling was immobile, oval, smooth surfaced, soft in

consistency & tender on palpation. A provisional diagnosis of eruption cyst was made. Differential diagnosis was eruption hematoma, pyogenic granuloma and peripheral giant cell granuloma. Investigations were intra oral periapical radiograph, OPG and cone beam computed tomography (CBCT). Radiographic examination showed impacted 84 and mixed radio opaque and radiolucent lesion surrounded by a well-defined radiolucency. CBCT findings revealed a malformed &malpositioned 84 with mixed radiolucent and radio opaque lesion. Right buccal view of CBCT showed super imposition of 84 with tooth bud of 1st premolar and perforation of buccal cortical plate. Right lingual view of CBCT showed lingually positioned crown of 84 & perforation of lingual cortical plate. The treatment plan that was made was surgical excision of the cyst and extraction of impacted primary first molar followed by space maintenance. A full thickness mucoperiosteal flap in the region from 83 to 85 was raised. Impacted tooth was visible after raising mucoperiosteal flap. Impacted primary first molar was extracted and as the tooth bud of first premolar which was fused with the roots of 84, the tooth bud came out along with it. Complete removal of the lesion was done, and a sample was sent for histopathological examination. Histopathological report confirmed it to be an aggressive variant of central giant cell granuloma. To rule out hyperparathyroidism endocrinology assessment of calcium, parathyroid hormone, and phosphorus weredone at a normal level. Considering the age of patient, the treatment plan was combined protocol entailed intralesional injections of corticosteroid followed by surgical curettage to eradicate the lesion.

Intra lesional corticosteroid injections were given once a week for 6 weeks followed by curettage. Sample were again sent for re-biopsy. Follow up radiograph showed improvement of lesion with proper bone formation.

**Outcome and Follow-Up**

The prognosis of present case was fair. One year follow up showed there has not been any recurrence and the facial profile became almost normal.

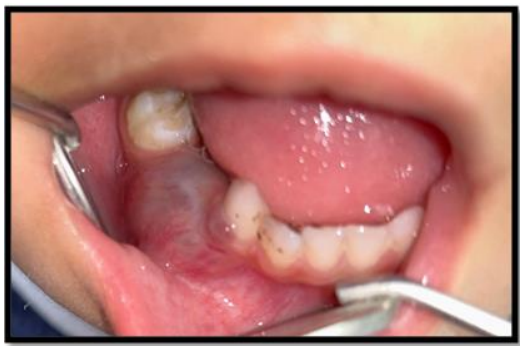

**Figure 1 - intra oral picture**

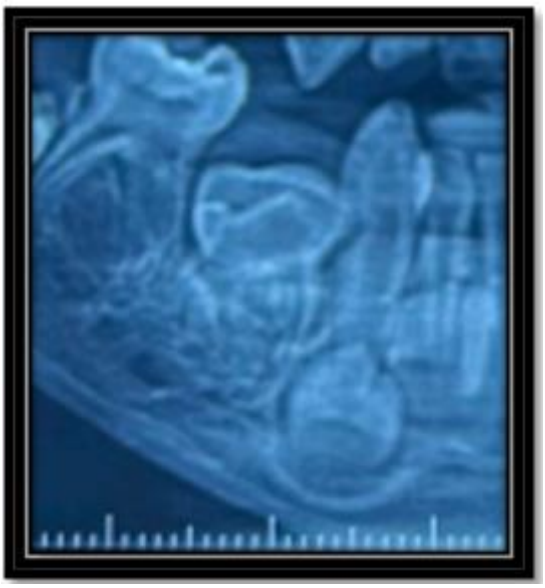

**Figure 2– IOPAR w.r.t 84, 85**

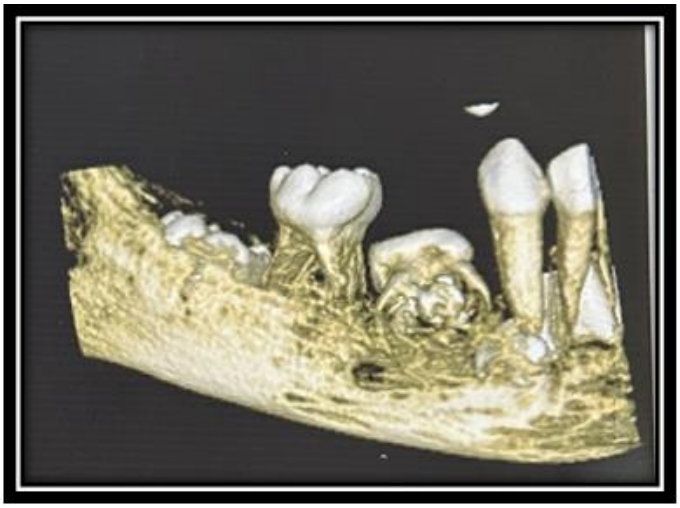

**Figure 3 – CBCT in 84, 85 region**

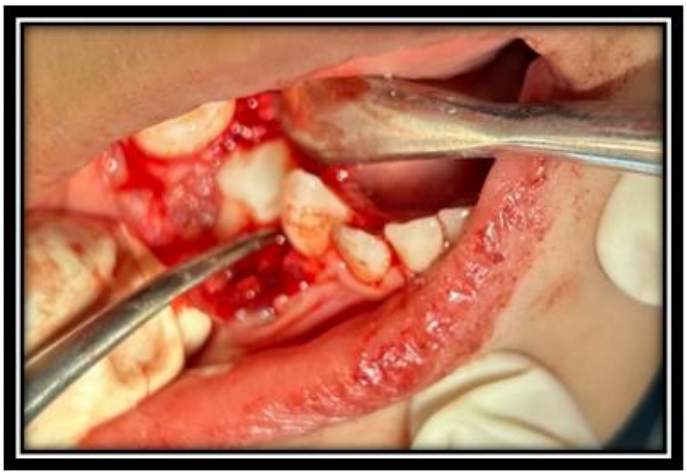

**Figure 4 – surgical extraction of 84**

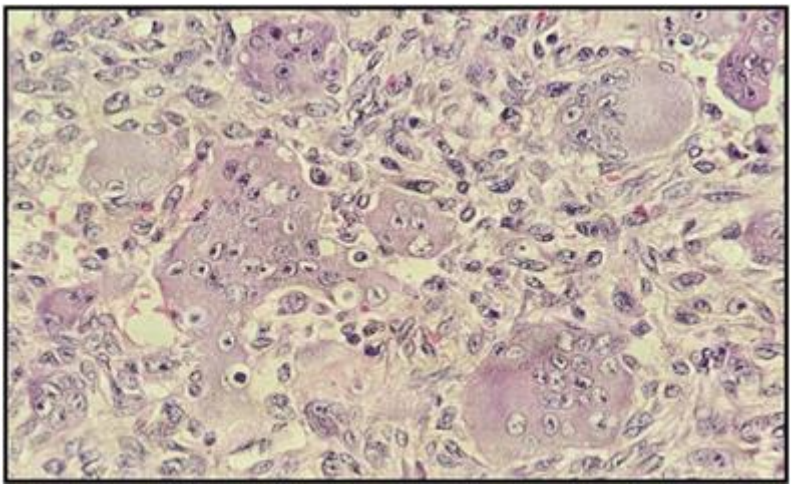

**Figure 5 – Histopathologic picture confirming CGCG**

**Figure 6 – Intra Lesional Corticosteroid Injection**

**Figure 7 – curettage of lesion done**

**Figure 8 – follow up radiograph**

## DISCUSSION

Central Giant Cell granuloma is a benign tumour of unknown aetiology, belonging to a group of giant cell tumors. It is a rare bony lesion in the head and neck region. That can be noted in every age group but is more common in patients below 30 years of age. It is predominantly seen in mandible than in the maxilla with a predilection for women than men (ratio 2:1).[2] World Health Organization in 1992 defined CGCG as an intraosseous lesion consisting of fibrous tissue containing multiple foci of haemorrhage, aggregates of multinucleated giant cells and trabeculae of woven bone.[5] Waldron in 1966 and Shafer in 1983 described the lesion as a reactive response of bone to repeated unidentified trauma or a reaction to some form of hemodynamic disturbance in bone marrow which is associated with trauma and haemorrhage.[6] The clinical behaviour of CGCG ranges from slow-growing, asymptomatic swelling to an aggressive lesion which manifests with pain and swelling. It can be an incidental finding during routine radiographic examination or associated with facial asymmetry, loosening or displacement of teeth and difficulty in mastication.[2] The radiological appearance of CGCG is

variable. The lesion may appear as a unilocular or multilocular radiolucency. It may be well defined or ill-defined with variable expansion and destruction of the cortical plate. In our case we noted a well-defined radiolucency with perforation of cortical bone plate. Depending on clinical and radiographic features, central giant cell granuloma can be classified into two types: The first type is non-aggressive, slow growing, does not show root resorption or cortical perforation, and often shows new bone formation. The second type is aggressive, rapid growing, painful, shows cortical bone perforation and root resorption, displaces anatomical structures such as teeth, the mandibular canal and the floor of maxillary antrum with a high recurrence rate as reported by OÕ Regan et al.; Marx & Stern; Sholapurkar et al., 2008; Rajendran et al., 2009. The features of present case are in favor of aggressive type. The distinctive feature of this lesion is the difficulty of diagnosis. Clinical and radiological features are usually not specific and pathognomonic and may be confused with that of many other lesions of jaws.[7] The final diagnosis rests on histopathology. Histologically, CGCG contain focal arrangements of giant cells within a vascular stroma.[2] Giant cells are larger and rounded, uniformly dispersed and have a greater number of nuclei. Giant cell granuloma shows a mass of proliferative vascular connective tissue packed with giant cells lying in vascular stroma.[8] In our case histological examination showed the presence of predominantly cellular connective tissue containing numerous multi nucleated giant cells with moderate amount of blood vessels. Giant cells were foreign body type containing 6-40 round to oval vesicular nuclei and were evenly distributed throughout the stroma. The stromal cells appeared mononuclear, spindle to round, highly proliferative with vesicular nuclei. There was evidence of nuclear and cellular pleomorphism at places, but no evidence of abnormal mitosis was evident. Few bony calcifications containing osteocytes, mild amount of chronic inflammatory cells predominantly macrophages were also evident. Presence of foreign body type giant cell (as seen in our case and absence of stromal tumour cells differentiate CGCG from a giant cell tumour. The differential diagnosis includes giant cell tumour, cherubism, aneurysmal bone cyst and jaw tumour of hyperparathyroidism. However, aneurysmal bone cyst shows blood filled cystic cavities. Cherubism is an autosomal dominant genetic disorder with bilateral involvement which is different from our case. Hyperparathyroidism brown tumour is characterized by involvement of multiple locations and shows high serum levels of parathyroid hormone, serum calcium, phosphorous and alkaline phosphate.[9] In our case only one lesion was present and the phosphocalcic assessment was shown to be normal which rules out the brown tumour of hyperthyroidism. The features of aggressive form of CGCG are similar to GCT and shows risk of malignant transformation.[10] However, GCT is 10 times less frequent than CGCG, and is mostly seen in older patients, which does not correspond to the age of our patient. Several authors by comparing some histopathological pictures of aggressive CGCGs which were totally indistinguishable from GCT concluded that GCT and CGCG are on a spectrum of a single disease process. This led these scientists to believe that CGCGs and GCTs of the extragnathic are not distinct entities but rather represent a continuum of the same disease process modified by the age of the patient, site of occurrence, and possibly other factors that are yet not clearly understood.[11] The management of CGCG depends on clinical and radiographic findings. Surgery is the most accepted and traditional form of treatment in which tissue removal ranges from simple & more conservative approach (curettage) to radicle en-bloc resection.[10] Several other treatment alternatives have been suggested in the literature to avoid the need for mutilating surgery in children, but the choice of treatment depends on the lesion's behaviour (aggressive versus nonaggressive), the location, size, and radiographic appearance of the lesions. The alternative treatment consists of administration of systemic calcitonin which inhibits osteoelastic activity. Intralesional injections of corticosteroids appear useful in the management of aggressive CGCG and subcutaneous a- interferon injections with antiangiogenic effects. Bisphosphonates have been also administered intravenously in CGCG with promising results.[12] Recurrence rate ranges from 4 - 20%, whereas higher incidence of recurrence (72 %) is found in aggressive lesions and in younger patients, especially in young male patients.[13] These recurrences are related to incomplete removal of a friable, bleeding lesion, which is more difficult to remove between teeth, or to a greater possibility of incomplete excision in a larger-sized lesion. There is a small difference in recurrence rate between the maxilla and the mandible (28.6 % and 23.2 %, respectively). A possible explanation for this difference may be that surgical curettage in the maxilla is more difficult as compared to mandible.[14] Two other treatment concepts have been advocated to reduce the recurrence of CGCG. One is the use of Carnoy's solution as a cellular fixative; another is to perform endodontic therapy of erupted teeth within the lesion. However, these solutions do not show a reduction in recurrences.[11] The present case was treated with corticosteroid therapy followed by surgical curettage; the patient showed no signs of recurrence after 1 year follow up.

## CONCLUSION

Central Giant Cell Granuloma is a less frequent pathology in daily dental practice. The clinical behaviour of this lesion is quite variable and difficult to predict. Hence the need of rigorous diagnosis approach based on meticulous history taking, careful clinical and radiological examinations, targeted biological assessment and anatomopathological

analysis. This case demonstrates the importance of considering CGCG in the differential diagnosis of rapidly progressive mandibular lesions in pediatric population. Prompt diagnosis and management can greatly improve long term outcomes.

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