UNILATERAL MANDIBULAR CENTRAL GIANT CELL GRANULOMA IN A CHILD: A CASE REPORT

1ARVIND KUMAR, 2RAVISH AHUJA
3ANIL KOHLI, 4PARVATHI DEVI
5VISHAL MEHROTRA, 6BHUVAN JYOTI

ABSTRACT

A 10 year old female child was seen in the Department of Pedodontics of Rama Dental College and Hospital (India) with a diffuse swelling about 2 into 3 cm in size on the left side of her face. It was firm but not tender. Left sub-mandibular lymph nodes were palpable, mobile, soft and tender. Intra orally a growth about 3 into 4 cm was present in the lower left vestibule.

CT 3D reconstruction image showed destructive lesion in 33-37 region. Histopathology of incisional biopsy revealed predominantly sheets of spindle to ovoid fibroblastic cells with interspersed osteoclast like giant cells. Enucleation was done under GA and tissue subjected to histopathology which confirmed the diagnosis.

Key words: Giant cell granuloma, benign lesions

INTRODUCTION

It is an uncommon, benign and proliferative lesion whose etiology is not defined. It was Jaffe who first introduced the term central giant cell reparative granuloma to distinguish this lesion from the giant cell tumor of long bones. However, since a reparative response was quiet rare and most of these lesions were found to be destructive rather than reparative, the word ‘reparative’ was omitted from that term.1

For many years this lesion has been discussed (with great controversy) with giant cell tumor of bone and other related lesions such as hyperparathyroidism, reparative response to injury, aneurysmal bone cyst, fibrous dysplasia, nonossifying fibroma and cherubism. All these manifest as intrabony lesion showing giant cells microscopically.2

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.3

The diagnosis of a central giant cell granuloma requires an evaluation for hyperparathyroidism. Serum calcium and alkaline phosphatase levels should be obtained prior to surgical removal of a giant cell granuloma and if abnormal, parathyroid hormone (PTH) levels should be assessed. Lesions identical to the giant cell granuloma occur in primary and secondary hyperparathyroidism and the treatment involves management of the hyperparathyroidism rather than treatment of the giant cell granuloma.4 This case was reported in Rama Dental College Hospital and enucleation of the lesion was performed under general anaesthesia.

1 Reader
2 PG Student
3 Professor, Dept of Pedodontics
4 Professor
5 Senior Lecturer
6 PG student, Dept of Oral Medicine & Radiology, Rama Dental College Hospital & Research Center, Kanpur, Uttar Pradesh, India

Corresponding Author: Dr Arvind Kumar, Flat No 406, Staff Accommodation, Rama Dental College, Hospital, Kanpur-208024, Email: drarvindverma29@rediffmail.com
CASE REPORT

A 10 year old female child reported to the Rama Dental College, Kanpur with the chief complaint of swelling in relation to the left side of the face since 2 years. Initially the swelling was small in size and has gradually attained the present size.

On extraoral examination, a diffuse swelling was seen in relation to left side of face (Fig 1), measuring approximately 2x3 cms, extending antero-posteriorly from the corner of the mouth up to angle of the mandible and superior-inferiorly from 2 cm below the ala-tragal line up to the inferior border of the mandible. On palpation the swelling was firm and non tender. Left submandibular lymph nodes were palpable, mobile, soft in consistency and tender.

Intraorally a solitary growth, measuring about 3x4 cms was present involving the lower buccal vestibule and the attached gingiva in the region of 34 to 37 (Fig 2), extending up to the occlusal level of 24-27. On palpation, the growth was soft to firm in consistency and non-tender. Buccal cortical plate expansion and vestibular obliteration and tenderness were present in

![Fig 1: Shows extraoral swelling on the left side of the face](image1)

![Fig 2: Shows intraoral lesion in 34-37](image2)

![Fig 3: OPG showing destructive lesion in 33, 34, 35](image3)

![Fig 4: Occlusal view shows multilocular radiolucencies with cortical plate expansion](image4)

![Fig 5: CT 3D-reconstruction image showing destructive lesion in 33-37](image5)
relation to 34-37. On palpation the cortical bone yielded in region of 35-36. There was no lingual cortical plate expansion.

Intraoral periapical radiographs of 33-37 and OPG (Fig 3) revealed only root displacement in relation to 34 and horizontal bone loss in relation to 35-36. Mandibular left lateral topographic occlusal view (Fig 4) revealed buccal cortical plate expansion with multilocular radiolucent lesion. CT 3D-reconstruction image showed destructive lesion irt 33-37 (Fig 5).

Aspiration was positive and blood tinged. Provisional diagnosis of Ameloblastoma with differential diagnosis of central giant cell granuloma, odontogenic keratocyst and ameloblastic fibroma was made and patient was advised for incisional biopsy.

The histopathology revealed predominantly sheets of spindle to ovoid fibroblastic cells with interspersed numerous osteoclast like giant cells. Foci of haemorrhage and hemosiderin deposits along with a trabeculae of reactive bone were also seen.

A diagnosis of central giant cell lesion was made and the patient was advised to undergo blood investigations to rule out hyperparathyroidism. The results were as follows: Serum calcium 8.8 mg % (normal 8.1-10.4 mg %), Alkaline phosphatase 93 iu/l (normal 37 to 147 iu/1), Enucleation of the lesion (Fig 6) was performed and tissue (Fig 7) was subjected to histopathology (Fig 8) and the report re-confirmed the diagnosis.

DISCUSSION

Central giant cell granuloma occurs more frequently in the mandible than in the maxilla, generally anterior to the first molar and often crosses the midline. The central giant cell granuloma is a lesion of young people with most cases diagnosed before the age of 30 years. The mandibular/ maxillary ratio has been reported as being from 2:1 (Kaffe et al) to 3:1 (Whitaker & Waldron). Waldron & Shafer found a 0.17% incidence of this lesion in their review of 20,000 oral biopsies. In gnathic lesions, females are affected more frequently (2:1 ratio) than males.

The lesion may present no signs or symptoms and may be discovered accidentally, but sometimes, central giant cell granuloma may lead to an expansion of the cortex and perforation, mobility, displacement and root resorption of associated tooth. The borders of the lesions may be regular or diffuse.

It is essentially a destructive lesion, producing a radiolucent area with either a relatively smooth or a ragged border and sometimes showing faint trabeculae. The majority of CGCG's (87.5%) present as expansile radiolucency, either unilocular or multilocular, which is generally traversed by bony spicules. Definite locu-
Central Giant Cell Granuloma

lations are often present particularly in larger lesions. The cortical plates of the bones are often thin and expanded and may become perforated by the mass. Displacement of the teeth by the lesion is seen with some frequency. The appearance of the giant cell granuloma is not pathognomonic and may be confused with that of many other lesions of the jaw, both neoplastic and non neoplastic.\(^1\)

Depending on clinical and radiographic features, central giant cell granuloma can be classified into two types: The first type of lesion is non aggressive, slow growing, does not show root resorption or cortical perforation, and often shows new bone formation. The second type is an aggressive type which grows quickly, shows pain, cortical perforation and root resorption.\(^1\)

In the histologic features loose fibrillar connective tissue stroma with many interspersed proliferating fibroblasts and small capillaries are seen. The collagen fibres are not usually collected into bundles however, groups of fibres will often present a whorled appearance. Multinucleated giant cells are prominent throughout the connective tissue, but not necessarily abundant. The giant cells vary in size from case to case and may contain only a few or several dozen nuclei. In addition, there are usually numerous foci of old extravasated blood and associated hemosiderin pigment, some of it phagocytized by macrophages. Foci of new trabeculae of osteoid or bone are also often seen, particularly around the periphery of the lesion.\(^1\)

Treatment usually involves enucleation and curettage. Other treatment modalities include systemic cancitonin, intralesional injections of corticosteroids and subcutaneous \(\alpha\)-interferon injections. Adornato and Kenneth\(^3\) successfully treated a patient with intralesional corticosteroids. Recently a case that did not respond to corticosteroids was treated successfully with calcitonin subcutaneous injection every day for 11 months.\(^2\) Recurrence rates ranging from 11\% to 49\% have been reported.\(^4\)

**CONCLUSION**

Diagnosis of CGCG even with advanced diagnostic aids is difficult. Histopathological report alone cannot confirm its diagnosis hence other haematological investigations must be considered imperatively to rule out other similar appearing lesions. Etiopathogenesis of CGCG is obscure and has varied clinical presentations and treatment modalities.

**REFERENCES**