



TRAUMA INDUCED CENTRAL & PERIPHERAL GIANT CELL GRANULOMA IN A SINGLE PATIENT: A RARE CASE REPORT

Dental Science

Dr. Upendra Talathi

Reader, Department of General surgery, Yogita Dental College & Hospital, Khed, Ratnagiri, Maharashtra, India.

Dr. Shitalkumar G Sagari

Reader, Reader, Department of Oral Pathology & Microbiology, Yogita Dental College & Hospital, Khed, Ratnagiri, Maharashtra, India.

ABSTRACT

We describe multiple Giant cell granulomas in a 17-year-old female who has presented in three months back with a large central giant cell granuloma of the right mandible with a history of trauma and she was treated with a surgical enucleation. A small recurrence was then identified on left maxilla (24, 25 region) after total extraction of teeth and the lesion was surgically excised and histologically confirmed as Peripheral giant cell granuloma with successful management and further referred for prosthesis. It is possible that the present case may indeed represent a new syndrome or subtype of multiple giant cell granulomas. The problem of treating such giant cell granulomas within a short period is also addressed in the context of recent advances of surgical and medical management.

KEYWORDS:

Peripheral giant cell granuloma, maxilla, reactive, Giant cells.

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. Females are affected more frequently than males commonly in the molar and premolar area, some may extend up to the ascending ramus of mandible¹.

Peripheral giant cell granulomas (PGCGs) are reactive, extraosseous and exophytic, located in the alveolar ridge in edentulous area, alveolar ridge and gingiva. It usually occurs as a result of local irritants such as bacterial plaque, calculus, food retention, chronic infections, chronic irritation, trauma related to exodontia, poorly finished fillings, poorly fitted dental prostheses, occlusal forces and supernumerary teeth^{2,3}. As to histology, both lesions are similar. However, their behavior differs in terms of aggressiveness. CGCL are generally more invasive and tend to recur, whereas PGCL may have at most superficial bone resorption. As PGCL bear close microscopic resemblance to CGCL, some pathologists acknowledge that it may represent a counterpart of the soft tissue of the central bone lesion^{4, 5}

Case report

A 16-year-old female patient referred to the Department of Oral Medicine department, with a swelling on the right side of lower jaw existing for two months with a history of trauma. The swelling was reported to be insidious in onset and had progressed slowly. The patient also reported that all the teeth in oral cavity had become mobile. The swelling was not associated with any systemic symptoms. There was no paresthesia or discharge. Medical and familial histories were noncontributory. The patient did not present any deleterious oral habits. Extraoral examination revealed a diffuse swelling on the right side of the jaw resulting in facial asymmetry (Figure 1). The overlying skin was normal. The swelling had slightly localized elevation of temperature. There was no associated lymphadenopathy.

Intraoral examination revealed a poor oral hygiene with loss of teeth. There was a swelling in the buccal aspect extending from the mesial of lower right second premolar to the distal of lower right second molar, obliterating the gingiva (Figure 2). It had an irregular surface with no evidence of fluctuation on palpation. There was no discoloration of the teeth. The teeth were tender on percussion.

Further On radiological examination, orthopantomogram (OPG) exhibits a well-defined radiolucency with radiopaque border in right posterior body of mandible extending towards ramus posteriorly and

inferiorly. Right second & third molar space seen as edentulous areas, indicated missing tooth with. Based on the history, clinical & radiological examination the differential diagnoses of Aneurysmal bone cyst, Central giant cell granuloma & Odontogenic keratocyst was given. Routine hemogram were done, which turned out to be normal. The serum calcium, phosphorous, parathyroid hormone were also normal, thus excluding the possibility of hyperthyroidism.

Hence the lesion underwent conservative surgical treatment involving curettage under local anesthesia for histopathological examination. Biopsied specimen revealed connective tissue made up of mature collagen fibres, fibroblasts and showing numerous multinucleate giant cells with foci of osseous structures (Figure 4). On the basis of histopathological and radiological findings, a diagnosis of CGCG was established. Further the patient underwent for total extraction of remaining teeth due to loss of alveolar bone height and all the tooth were mobile with loss of periodontal support.

Later once the healing was complete patient was recalled after one month for prosthesis, but we noticed a single, diffuse swelling on intraoral examination, which was seen on the left side of maxilla on edentulous alveolar ridge. The swelling measured about 2 × 2 cm. The surface of the swelling was lobulated and present in relation first molar region. The swelling was pedunculated, firm in consistency and pinkish in color, and the overlying mucus membrane was intact (Figure 5). Orthopantomogram, intraoral periapical radiographs showed no bone resorption.

Excisional biopsy was planned under local anesthesia, the overlying mucosa was incised and undermined. Lesion was separated from the adjacent tissue and removed in one piece and was sent for histopathologic examination. Biopsied specimen revealed connective tissue stroma which was highly cellular, consisting of proliferating plump fibroblasts. Numerous giant cells of numerous shapes and sizes, containing 10 - 15 nuclei, were seen with proliferating and dilated endothelial lined blood capillaries with extravasated red blood cells (RBCs). The lesional tissue with surface covered with stratified squamous epithelium which was diagnosed as PGCG.

Discussion

The CGCG appears as a painless expansile mass. The clinical behavior of the CGCG ranges from a slowly growing asymptomatic swelling to an aggressive lesion causing pain, local bone destruction, root resorption or displacement of the tooth. Giant cells are the most prominent histopathological feature of CGCG and many studies have

been concentrating to the role of the original mononuclear cells in its pathogenesis⁶.

Regarding etiology, which is a much debated topic, there are local and systemic factors as well as possible mutations described in exons 3, 4, 9 and 11 of SH3BP2 gene. Local factors include traumas and vascular damage, which cause intramedullary hemorrhage and intraosseous replacement fibrosis. The etiology and nature of PGCG still remains undecided. Several propositions had been suggested to explain the nature of multinucleated giant cells, including the explanation that they were osteoclasts left from physiological resorption of teeth or reaction to injury to periosteum^{5,1}.

There is also a growing body of opinion that giant cells in PGCGs may simply represent a reactionary component of the lesion and are derived via blood stream from bone marrow mononuclear cells and may be present only in response to an as yet unknown stimulus from the stroma⁷.

Among the systemic causes, CGCGs are chiefly associated with Neurofibromatosis type I, Noonan syndrome, Ramon syndrome, Jaffe-Campanacci syndrome, association with cherubism, pregnancy and hormonal disorders such as hyperparathyroidism⁸. Radiographically, the CGCGs appear as uni or multilocular well-defined radiolucent bone defects of variable size, depending on the aggressiveness of the lesion. In the present case the lesion was seen in right posterior body of mandible as a well-defined, expansile, unilocular radiolucency with well-defined sclerotic border which was pathognomonic feature of CGCG⁵.

Even though the PGCG develops within soft tissue, "cupping" superficial resorption of the underlying alveolar bony crest is evident sometimes. In some cases it may be difficult to decide whether the mass is a peripheral lesion or a central giant cell granuloma eroding through the cortical plate into the gingival soft tissues⁸. But in the present case there was no cupping of underlying bone and lesion was seen in very short period on edentulous left maxillary ridge after total extraction.

Histologically, CGCGs are characterized by dense proliferation of oval or spindle-shaped mesenchymal cells as well as variable number of multinucleated giant cells containing 4 to 20 nuclei spread in the fibrous stroma adjacent to perivascular hemorrhagic area. Foci of bony trabeculae, dystrophic calcification and predominantly of mononuclear inflammatory infiltrate, particularly surrounding the periphery of the lesion, are also found⁹, similar histological findings are evident in the present case (Figure 5).

Whereas in PGCGs there is an unencapsulated tissue mass with acute and/or chronic inflammatory infiltrate permeating highly cellularized fibrous tissue with foci of hemorrhage and deposition of hemosiderin. There is vascular proliferation and multinucleated giant cells permeated by massive oval to spindle shaped mesenchymal cells. Giant cells have various shapes and sizes, typically containing 8 to 15 nuclei. The surface epithelial lining covering lesional area may be hyperplastic or ulcerated¹⁰.

Despite the benign nature of CGCG, there are few cases which showed metastasis and documented in the literature. Malignant transformations of CGCG to fibrosarcoma and osteosarcoma have also been reported in the literature. Most accepted form of treatment for CGCG is a conservative surgical treatment involving curettage alone or with peripheral osteotomy^{11,12}, whereas for PGCGs excisional biopsy under Local anesthesia is the standard surgical protocol which was performed in the present case.

Conclusion

This is a rare case where CGCG and PGCG diagnosed in a single patient who presented with history of trauma and patient had to undergo extraction of all the teeth which were mobile due to trauma. Hence PGCG and CGCG are entities that arise in the peripheral or center regions, respectively. The true nature of CGCG remains indefinite and it has not been elucidated whether the lesion has a reactive, infectious, neoplastic or inflammatory origin and in our case it was of traumatic origin. An agreement has not been made in the literature whether the two lesions have different behaviors or whether they are really different entities.

Figure legends

Figure 1: Clinical picture showing facial asymmetry with diffused swelling on right mandibular posterior region



Figure 2: Photograph revealing missing tooth in 47 & 48 region and slight ulceration, along with intraoral swelling in the same region.



Figure 3: Panoramic radiograph showing well-defined radiolucency surrounded by sclerotic border in right mandibular posterior region with generalized alveolar bone loss & displaced teeth.



Figure 4: Pictomicrograph (20x) showing multinucleated giant cells (Black arrows) adjacent to numerous bony trabeculae nearby perivascular hemorrhagic area.

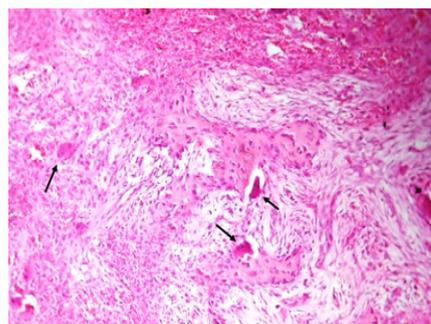


Figure 5: Intraoral picture showing well defined swelling on right maxillary alveolar ridge



References

1. Jaffe HL. Giant-cell reparative granuloma, traumatic bone cyst, and fibrous (fibro- osseous) dysplasia of the jaw bones. *Oral Surg Oral Med Oral Pathol* 1953;6:159-75.
2. Amaral F. R. et al. Quantitative expression analysis of apoptotic/antiapoptotic genes and association with immunolocalization of BAX and BCL-2 in peripheral and central giant cell lesions of the jaws. *Tumour Biol*, v. 32, n. 5, p.997-1003, 2011.
3. Bansal P. et al. Non-syndromic multiple impacted supernumerary teeth with peripheral giant cell granuloma. *Contemp Clin Dent*, v. 2, n. 1, p.41-4, 2011.
4. Kruse-Losler B. et al. Central giant cell granuloma of the jaws: a clinical, radiologic, and histopathologic study of 26 cases. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*, v. 101, n. 3, p. 346-54, 2006.
5. Tandon, PN, et al. Peripheral giant cell granuloma. *Contemp Clin Dent* 2012;3(1):118-21, 2012.
6. Whitaker SB, Waldron CA. Central giant cell lesions of the jaws. A clinical, radiologic, and histopathologic study. *Oral Surg Oral Med Oral Pathol* 1993;75:199-208.
7. El-Mofty SK, Osdoby P. Growth behaviour and lineage of isolated and cultured cells derived from giant cell granuloma of the mandible. *J Oral Pathol* 1985;14:539-52.
8. Vasconcelos R G, Vasconcelos M G, Guedes Queiroz L M. Peripheral and central giant cell lesions: etiology, origin of giant cells, diagnosis and treatment *J Bras Patol Med Lab* 2013;49(6):446-452
9. Valentine JC, Nelson BL. Central giant cell lesion. *Head Neck Pathol*, v. 5, n. 4, p. 385-8, 2011.
10. Etoz OA et al. The peripheral giant cell granuloma in edentulous patients: report of three unique cases. *Eur J Dent*, v. 4, n. 3, p. 329-33, 2010.
11. Thompson SH, Bischoff P, Bender S. Central giant cell granuloma of the mandible. *J Oral Maxillofac Surg* 1983;41(11):743-6.
12. Small GS, Rowe NH. A "true giant cell tumor" in the mandible? *J Oral Surg* 1975;33(4):296-301.