CASE REPORT

Peripheral Giant Cell Granuloma: Benign Tumor of Oral Cavity - A Case Report and Review of Literature

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Abstract

Peripheral giant cell granuloma (PGCG) is one of the most frequent giant cell lesions of the jaws and originates from the connective tissue of the periosteum or the periodontal membrane. It is also known as giant cell epulis, osteoclastoma, giant cell reparative granuloma, or giant-cell hyperplasia. The exact etiology of PGCG is unknown but the usual contributing factors include local irritating factors such as plaque, calculus, food impaction, trauma, badly finished fillings and tooth extraction. This article reports a case of huge peripheral giant cell granuloma arising at the left maxillary alveolus in a 60-year-old female and discusses the clinical, histopathological features and treatment along with the review of literature.


Key words: Giant cells, Peripheral Giant Cell Granuloma, Giant cell epulis, saucerisation

Introduction

Multinucleated giant cells are important mediators of tissue remodeling and repair and depending on the tissue where fusion occurs and the inflammatory insult, they assume distinctly different phenotypes. They were first described by Langhans who reported the presence of poly nuclear cells in tuberculoid granulomas. These cells are recognized as a common feature of granulomas induced both by immunological and nonimmunological stimuli.

A number of jaws lesions contain giant cells within them such as cherubism, giant cell granuloma of the jaws, giant cell tumor, aneurysmal bone cyst, traumatic bone cyst and jaw tumor of hyperparathyroidism. Their relationship to each other, however, is ill defined. The histological similarities cease with the finding of multinucleated giant cells of osteoclastic origin and the lesions themselves greatly differ in their

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genetic origin, etiopathogenesis and clinical behaviour.\textsuperscript{3}

Peripheral giant cell granuloma (PGCG) is one of the most frequent giant cell lesions of the jaws and originates from the connective tissue of the periosteum or the periodontal membrane.\textsuperscript{5} It is not a true neoplasm but rather a benign hyperplastic reactive lesion occurred in response to local irritation such as tooth extraction, poor dental restorations, ill-fitting dentures, plaque, calculus, food impaction and chronic trauma. Other names of this lesion are peripheral giant cell tumor, osteoclastoma, reparative giant cell granuloma, giant cell epulis and giant cell hyperplasia of the oral mucosa.\textsuperscript{6}

We present a case of a peripheral giant cell granuloma present in maxilla in a sixty year old female patient who was managed by a conservative surgical approach along with review of literature.

Case report

A 60 year old female was initially referred to the out patient department at Seema Dental College & Hospital Rishikesh for an exophytic lesion in the left maxillary alveolus. Her history suggested that the lesion had been present for 2 years and had gradually increased in size. It bled from time to time but the patient did not seek any treatment.

The patient had no disease antecedents of interest. Extraoral examination revealed no pathological findings; however, intraoral examination revealed an exophytic sessile lesion originating at the distal surface of the left maxillary second premolar and extending till the mesial surface of second molar. The lesion measured 3 x 3 cm, had a soft to firm consistency was reddish pink in color and appeared nodular. The patient had poor oral hygiene, with extensive plaque on the surface of all teeth. Calcium, phosphorus, alkaline phosphatase and parathyroid hormone levels were all within normal ranges.
stroma. Under higher magnification, epithelium was of parakeratinized stratified squamous type. Connective tissue stroma showed numerous proliferating blood vessels with extracellular hemorrhage. Numerous spindle shaped fibroblasts were also noted. Several multi-nucleated giant cells were seen interspersed in the connective tissue stroma. Hence histopathological findings confirmed the diagnosis of peripheral giant cell granuloma.

![Figure 4- H&E(10X) stained image of the lesion, showing epithelium and connective tissue with multinucleate giant cells.](image)

**Discussion**

Peripheral giant cell granulomas (PGCG) are relatively uncommon reactive exophytic lesions of the oral cavity. It is also known as peripheral giant cell epulis, peripheral giant cell reparative granuloma. The term reparative granuloma has been omitted from the literature as the lesion is not truly reparative. The aetiology and nature of PGCG still remains undecided. Local irritation factors such as poor dental restorations, dental extraction, plaque, and calculus accumulation play significant role in the development of a PGCG. Dental implants have also been reported as a predisposing factor. A possible hormonal (Estrogen & progesteron) influence for some Peripheral Giant Cell Granuloma has been postulated by Whitaker & Giansanti. Chambers discussing Caillouette & Mattar’s paper suggested that these hormones have immunosuppressive actions which contribute to growth of lesions.

PGCGs account for less than 10% of all hyperplastic gingival lesions. They originate from the periosteum or periodontal membrane following local irritation or chronic trauma. Generally, PGCG size varies from 0.5 to 1.5 cm in diameter. In our case the lesion measured 3X2.5 cm. There are no pathognomonic clinical features whereby these lesions can be differentiated from other forms of gingival enlargement. Their incidence rate has been reported as ranging from 5.1% to 43.6% among all reactive growth. Lesions are seen at a rate of 40% between 40-60 years of age, and at a rate of 20-30% between 10-20 years of age. Cases of PGCG have been documented in children, where the lesion appears to be more aggressive, with absorption of the interproximal crest area, displacement of the adjacent teeth and multiple recurrences. In our case the patient was 60 years of age.

PGCG is more common in the lower jaw rather than the upper jaw. The reported proportion is 2.4:1 and in most cases, it occurs anterior to molar region. According to Motamedi et al, PGCG more frequently involves the mandible, commonly in the areas posterior to canines region. In the present case the lesion was present in the maxilla in the first molar area.
Bhasker\textsuperscript{17} & Daley et al\textsuperscript{18} have shown male predilection whereas several authors have noted a female predilection. Our case which is mentioned there was of a female patient.

These lesions have a reported average diameter of less than 20 mm, but the extent of their growth capacity is not well known. Given that their highly vascularised nature makes large PGCGs susceptible to ulceration as a result of continuous occlusal trauma, it is likely that these lesions are excised before they achieve their full growth capacity. On the other hand, considering that the gum and alveolar crest are the only places where PGCGs have been reported, it is also possible that the growth capacity of these lesions is limited.\textsuperscript{19}

Small lesions rarely show any radiographic evidence but larger lesions exhibit a superficial erosion of the cortical bone surface along with widening of the adjacent periodontal space. In edentulous areas, the cortical bone exhibits a concave area of a resorption beneath the lesion, often referred to as "saucerisation". Radiographs are important to determine if the lesion is of gingival origin or of central origin with extension to the surface.\textsuperscript{20}

Differential diagnosis of peripheral giant cell granuloma involves giant cell tumour, nonossifying fibroma which differs from PGCG lesions in consistency and colour; pyogenic granuloma which is difficult to distinguish from PGCG lesions; CGCG which is an expansive and destructive intraosseous lesion that can perforate the cortex, mimicking PGCG; chondroblastoma which, localized in the gum, may provoke irregular bone destruction below the exophytic lesion; odontogenic cyst; parulis, which is frequently associated with a necrotic tooth or with periodontal disorder; haemangioma cavernosum, which is distinguished from PGCG lesions by their pulsatile nature; fissured epulis.\textsuperscript{8} Definite diagnosis can be established through histopathologic examination.

Histologically, PGCG is composed of nodules of multinucleated giant cells in a background of plump ovoid and spindle-shaped mesenchymal cells and extravasated red blood cells. The giant cells may contain only a few nuclei or up to several dozen. Abundant hemorrhage is characteristically found throughout the mass which often results in deposits of hemosidrin. The overlying epithelium may be ulcerated in 50\% of the cases. A zone of dense fibrous connective tissue usually separates the giant cell proliferation from the mucosal surface. Areas of reactive bone formation or dystrophic calcifications may be seen.\textsuperscript{21} The histopathological findings in our case were corresponding to the above description.

The pathogenesis of giant cells has not been thoroughly investigated. Several immunohistochemical studies have focused on identifying the nature and the interrelations between cellular components in the formation of GCG: the results have suggested that the mononuclear stromal cells may originate from fibroblasts and cells of histiocytic origin whereas the origin of giant cells has still been a source of controversy; in fact some authors suggest that they arise secondary to an alteration of the endothelial cells of the capillaries others as a consequence of a traumatic mechanism (some similarities to the osteoclasts).\textsuperscript{13} Palacios et al suggested that giant cell formation to be a fusion of histiocytes, endothelial cells and fibroblasts.\textsuperscript{22} Falaschini et al in their study found that the capillaries on the periphery of the lesions were strongly positive for CD34, whereas the reaction product was not evident in the lesion within the aggregations of multinucleate giant cells. This data may suggest that multinucleate giant cell do not arise from endothelial cells of the capillaries. Thus they concluded that multinucleated giant cell show an osteoclast phenotype and that probably derive from monocyte/macrophage lineage and that giant cells do not derive from the endothelial cells of the capillary.\textsuperscript{13}

There is also a growing body of opinion that giant cells may simply represent a reactionary component of the lesion and are derived via blood stream from bonemarrow mononuclear cells and may be present only in response to an as yet unknown stimulus from the stroma. This concept is based on the results of some more recent studies using cell culture and transplantation in which the giant cells have been found to be short-lived and to disappear early in culture in contrast to the
active proliferation of the stromal cells. A study by Willing et al. suggested that the stromal cell stimulates blood monocyte immigration into tumour tissue and enhances their fusion into osteoclast-like, multinucleated giant cells. Dayan D et al. however proposed that the stromal cells comprised of proliferating osteo-progenitor cells, pericytes, fibroblasts and myofibroblasts. The presence of myofibroblasts was made evident by histochemical procedures and electromicroscopy, which displayed intracellular collagen fibrils, supporting the reactive nature of these lesions.

Traditional treatment of a PGCG consists of surgical resectioning of the lesion and elimination of the etiological factors. When the periodontal membrane is affected, full resectioning may require extraction of adjacent teeth. As an alternative to surgery, carbon-dioxide laser resectioning involves less intra-operative bleeding, provides wound sterilization and requires no sutures. However, laser treatment is contraindicated in cases where the lesion is oriented close to the bone and where careful curettage is required. No malignant variations of PGCGs have been reported, and recurrence rates have been reported to range from 4.41%–50% with the differences in rates possibly related to the type of surgical resectioning procedure used.

Conclusion

Giant Cell Lesions (GCLs) form an important group of oral lesions. Late diagnosis and treatment of GCL will result in extension of the lesion and difficulties in treatment. PGCG is the most common giant cell lesion which can attain a large size and may follow an aggressive course so early and definitive diagnosis of the lesion on the basis of history clinical radiographic and histopathological examination allows conservative management with minimal risk to adjacent hard tissue. Proper therapy and regular follow-up will help in ensuring that there is adequate healing and minimal chance of recurrence, as demonstrated in this case.

References


