Case Report

Peripheral Giant Cell Granuloma – A Review and Case Report

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ABSTRACT

Peripheral giant cell granuloma is among the reactive lesions of oral cavity present either on the gingiva or alveolar ridge. It can occur due to local irritational factors, trauma, etc. This case report presents a peripheral giant cell granuloma on the lingual aspect of mandibular premolars with hard bony consistency and a firm, consistent growth seen in interdental region between left mandibular premolars in a 48 year-old female patient. Traditional surgical excision was performed under local anesthesia.

Keywords: Peripheral Giant Cell Granuloma (PGCG), Excisional Biopsy, Gingival Overgrowth, Mandible

INTRODUCTION

Oral cavity manifests a spectrum of lesions which could be either reactive, developmental and inflammatory to neoplastic. Constant external or internal stimuli causes the lesion to occur.1 Reactive hyperplastic lesions represent the most frequently encountered oral mucosal lesions in humans. Peripheral giant cell lesions (PGCL) are reactive, extraosseous and exophytic, located in the alveolar ridge in edentulous area or in the gingiva. It usually occurs as a result of local irritants such as bacterial plaque, calculus, food retention, chronic infections, chronic irritation, trauma related to poorly fit dental prostheses, supernumerary teeth, poorly finished fillings, occlusal forces and exodontia. If the lesion is excised along with the elimination of local factors, the recurrence rate is low.

Central giant cell lesions (CGCL) are intraosseous nonproliferative lesions whose etiology is unknown. It is less common than PGCL and occurs exclusively in maxillary bones. It has variable clinical manifestations with either rapid painful growth with recurrence or slow asymptomatic growth with no recurrence.

PGCL are derived from periosteum and periodontal ligament and occurs frequently in young adults. It occurs in variable sizes, sessile or pedunculated.2 PGCL appear as a reddish purple or purplish blue lump with smooth shiny or papillomatous surface. It is a well-defined lesion with exophytic growth and rarely exceeds 3 cm in their greater dimension. Although they are encountered at any age, the fourth to sixth decades are more frequent with a slight female predilection.3 A few cases have been reported occurring in children, and in these cases the lesion appeared to be more aggressive with resorption of the interproximal crest area, displacement of the adjacent teeth and multiple recurrences. Previously, the lesion was called peripheral giant cell reparative granuloma. However, its reparative effect has not been proved yet, hence osteoclast activity seems doubtful.4,5,6 Although PGCL arise in soft tissues, the "cup-shaped" resorption of the subjacent alveolar bone may be occasionally observed.7,8
Peripheral giant cell lesion can be differentiated from other inflammatory hyperplastic lesions by presence of multinucleated giant cells whose origin is undetermined.

The differential diagnosis of peripheral giant cell granuloma involves lesion with very similar clinical and histopathological features such as CGCL, fibrous hyperplasia, peripheral ossifying fibroma, pyogenic granuloma, inflamed irritation fibroma, hemangioma, lymphangioma, amelanotic melanoma and metastatic tumors. Rarely a giant cell epulis may be due to hyperparathyroidism, representing the so-called osteoclastic "brown tumours" associated with this endocrine disorder (Smith et al, 1988; Burkes & White, 1989) and is also associated with other lesions in bones and changes in the blood chemistry. The gingiva in extra-osseous lesions of cherubism appears very similar to giant cell epulide. However, the other distinctive clinical and radiological features of cherubism indicate the correct diagnosis (Odell & Morgan, 1998).

Early diagnosis based on clinical and radiological findings and confirmed by pathological analysis allows for conservative management with less risk of destruction for the adjacent teeth and tissues.

**CASE-REPORT**

A female patient of 48 years presented with chief complaint of soft to firm swelling in lower front region of the jaw (inner aspect) since few months.

On examination generalized deposits of calculus and marginal recession of gingiva was also present. Intra oral periapical radiograph revealed bone resorption and widening of Lamina dura with lower second premolar (Fig 1 and Fig 2). Generalized bleeding on probing of the periodontal pockets was present. The lesion present was painless, as well as elastic on palpation, sessile, extending from distal aspect of left first premolar to mesial aspect of left first molar covered by red-white mucosa and measuring about $10 \times 7 \text{ mm}^2$ (Fig 3 and Fig 4).

The provisional diagnosis for the case was moderate chronic periodontitis with pyogenic granuloma. The differential diagnosis could be Fibrous hyperplasia, inflamed irritational fibroma and Hemangioma.

The patient underwent complete blood investigations. Phase1 periodontal therapy was performed, and patient was recalled after 15 days for excisional biopsy. The lesion was excised under local anaesthesia, and flap of that quadrant was raised for complete debridement and root planing in order to prevent recurrence. Obtained tissue specimens were sent for histopathological examination.

On microscopic examination the excised specimen shows single bits of tissues consisting of epithelium and connective tissue. The epithelium was parakeratinized stratified squamous type (Fig 5). At one part of the epithelium there were long pushing rete ridges and at the other part it was flat. The lamina propria adjacent to epithelium showed loosely arranged collagen fibres, few chronic inflammatory cells, many diffusely arranged fibroblast and blood vessels which appeared engorged as well as dilated with blood elements in it. Deeper connective tissue showed many multinucleated giant cells distributed evenly with variable number of nuclei ranging from 4-10 in number with mild to moderate chronic inflammatory cells like plasma cells and lymphocytes (Fig 6). The lesion was diagnosed as Peripheral Giant Cell Granuloma.
Fig 1 - Intra-oral peri-apical radiograph revealing reduction of the level of crestal bone in premolar area.

Fig 2 – OPG revealing generalized bone loss

Fig 3 - Specimen removed by excisional biopsy (about 7 × 10 mm²)
Fig 4 - Showing overgrowth on lingual aspect below mandibular premolar

Fig 5 - Low Power Microscope showing Surface Epithelium

Fig 6 - High Power Microscope showing many Multinucleated Giant Cells
DISCUSSION

The present case was histopathologically diagnosed as peripheral giant cell granuloma. The peripheral giant cell granuloma, also known as giant cell epulis, PGCL or giant cell hyperplasia though is the most common giant cell lesion, it is not a true neoplasm, but rather a reactive lesion caused by local irritation or trauma. Moreover, its etiology is still contentious. These lesions have been described as reddish or purple with a smooth surface and consistency that varies from soft to firm. In the present case, the lesion was reddish in color in accordance to the literature.

The preferential location of the lesion is premolar and molar zone, though Shafer, Giansanti and Waldron suggest that it commonly occurred anterior to molars. The occurrence of PGCG is 2 times more common in females than males. When compared maxilla to mandible, it is more frequent in latter. All the characteristic features present in our study were in accordance to the literature.

The characteristic histopathological features include a non-encapsulated highly cellular mass with abundant giant cells, hemosiderin deposits, inflammation, mature bone or osteoid, interstitial hemorrhage. Incipient lesions may bleed and induce minor changes in gingival contour but large ones adversely affect normal oral function. Interference with occlusion may cause ulceration of the lesion due to which it becomes infected and painful.

As the exact origin of the giant cells remains unclear, several hypotheses were generated to explain their proliferation: osteoclasts, spindle-shaped mesenchymal cells, osteoblasts, foreign body cells, phagocytes reacting to hemorrhage and endothelial cells.

Two members of the tumor necrosis factor (TNF) group: receptor activator of nuclear factor-κβ ligand (RANKL) and osteoprotegerin (OPG) helps Stromal mononuclear cells (monocytes and macrophages) to participate in the formation of multinucleated giant cells. The transmembrane molecule RANKL is produced by osteoblasts/stromal cells and binds to its RANK receptor, which is situated on osteoclast progenitor cells surface. Differentiation of osteoclast progenitor cells into mature osteoclasts is promoted by RANK – RANKL binding. OPG, also produced by stromal cells/osteoblasts, competitively binds to RANKL thereby blocking and neutralizing its binding to the RANK receptor, resulting in the reduction of osteoclastogenesis.

Treatment consists of local surgical excision down to the underlying bone, for extensive clearing of the base (Neville et al, 2009). Removal of local factors or irritants is also required (Regezi et al, 2008). If resection is only superficial, the growth may recur. Exposure of all bony walls following thorough surgical resection responds satisfactorily most of the times. Recurrence rate of 5.0-70.6% (average 9.9%) has been reported in various epidemiologic studies (Mighell et al, 1996).

CONCLUSION

Early and precise diagnosis of the lesion can allow conservative management without destruction of tooth and adjacent bone. Treatment consists of local surgical excision down to the underlying bone, with removal of local factors or irritants. The growth may recur if superficially resected. So complete excision of the lesion along with regular recall visits is the treatment of choice for Peripheral Giant Cell Granuloma.
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REFERENCES