PERIPHERAL GIANT CELL GRANULOMA – A CASE REPORT

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ABSTRACT

Peripheral giant cell granuloma (PGCG) is a non-neoplastic lesion representing a local hyperplastic reaction. It is a tumor-like pathologic condition arising on the buccal or lingual attached gingival or alveolar mucosa and the crest of the edentulous alveolar ridge, of uncertain etiology; it is probably a reactive lesion caused by chronic local irritants or trauma rather than a true neoplasm. PGCG is thought to originate from elements of the periodontal ligament or from the periosteum. Clinically, it appears as a sessile or broadly pedunculated, bluish to purple-red, fleshy or firm swelling with a frequently ulcerated surface. It may be difficult to distinguish PGCG from pyogenic granuloma and peripheral odontogenic tumors. The clinical appearance of all these lesions is similar, the definitive diagnosis with histological examination is mandatory. Treatment of choice is surgical excision with total removal of the base of the lesion. This case aims to present a case of PGCG in a 11 year old girl in which surgical resection of the lesion was done to restore functions and esthetics.

KEYWORDS: Giant Cell, Exophytic, Peripheral Granuloma

INTRODUCTION

Peripheral giant cell granuloma is a reactive, exophytic lesion of the oral cavity, also known as giant-cell epulis, osteoclastoma, giant cell reparative granuloma, or giant cell hyperplasia. It is the most frequent giant cell lesion of the jaws, and originates from the connective tissue of the periosteum or from the periodontal membrane, in response to local irritation or chronic trauma. Jaffe first suggested the term giant cell granuloma for the central lesion of the jaw bones. Bernier and Cahn proposed the term peripheral giant cell reparative granuloma for the peripheral lesion. Today the term peripheral giant cell granuloma is universally accepted. It is more frequent in women than in men, with a slightly higher prevalence in the 30- to 70-year-old-age group, and affects largely the lower jaw (55%) than in the upper jaw (the reported proportion being 4:1).

Cases of PGCG have been documented in children, where the lesion appears to be more aggressive, with resorption of the interproximal crest area, displacement of the adjacent teeth and multiple recurrences. Clinically, it manifests as a soft to firm, bright nodule or as a sessile or pedunculated mass, which is predominantly bluish red with a smooth shiny or mamililated surface, localized in the gingival tissue or alveolar processes of the incisor and canine region, though according to Pindborg the preferential location is the premolar and molar zone. The lesion range in size from small papules to enlarged masses, though reportedly rarely exceeding 2 cm in diameter, and are generally located in the interdental papilla, edentulous alveolar margin, or at marginal gum level. It is basically asymptomatic, in fact pain is not a common characteristic, and lesion growth in most cases is induced by repeated trauma such as, with occlusion in which case it may ulcerate and becomes infected.

Although the pathogenesis of oral cavity PGCGs is still uncertain, local irritants such as calculus, bacterial plaque, periodontitis, periodontal surgery, ill fitting dentures, overhanging restorations and tooth extractions, are suggested as the etiological causes. These soft tissue lesions rarely affect the underlying bone, though the latter
Fig. 1. Intraoral photograph showing an exophytic lesion extending from mandibular left deciduous canineto right deciduous canine

Fig. 2. Periapical radiograph showing loss of interproximal bone.

Fig. 3. Mandibular Occlusal view showing reactive expansion of cortical bone

Fig. 4. Postsurgical view

Fig. 5. Photomicrograph showing giant cells with basophilic cytoplasm and multiple nuclei, lying within the vascular spaces. Extravagated red blood cells are also seen. (Haematoxylin and Eosin).

Fig. 6. Healing after 6 months
may suffer erosion. Treatment comprises surgical resection, with extensive clearing of the base of the lesion to avoid relapses.

**Case Report**

An eleven year old female patient reported to the department of Pediatric dentistry, People’s Dental Academy, Bhopal, with a chief complaint of growth in the lower front region, which bled frequently and interfered with eating. Her medical history was non-contributory. History revealed that growth was noticed 3 months back during the routine dental examination. Recently the lesion has doubled in size, is interfering with normal masticatory functions and bleeds on slight probing.

Clinical examination revealed reddish, sessile nodule measuring 2 x 2 x 2 cm involving the mandibular anterior gingiva and alveolar mucosa on both buccal and lingual side. There is smooth lobulated without any ulceration. Although the non tender lesion bleeds easily with minimal manipulation, it does not blanch when palpated. The adjacent mandibular incisors exhibit class I mobility with interproximal separation and lingual displacement(Fig.1). A periapical radiograph revealed superficial erosion of the alveolar bone interproximally (Fig.2) and occlusal view depicted reactive expansion of cortical bone (Fig.3). No other oral or cutaneous lesions were noted in this child.

Clinical provisional diagnoses in this case were PGCG, pyogenic granuloma, peripheral ossifying fibroma and the lesion was completely excised surgically (Fig.4). The lesion was then sent for histopathological examination to confirm the diagnosis. The microscopic examination of haematoxylin stained tissue section revealed a covering of parakeratinised stratified squamous epithelium with proliferation. Underlying connective tissue stroma was delicate with abundant proliferating blood vessels with extravasated erythrocytes. There were diffused distribution of large amounts of giant cells and inflammatory cells throughout the connective tissue stroma (Fig.5). A confirmed diagnosis of peripheral giant cell granuloma was made.

Healing was uneventful, and no recurrence was observed after 6 month follow up (Fig.6). During this period the patient did not report any complaints and no other treatment was needed.

**Discussion**

Solitary gingival enlargements are relatively common finding and usually with numerous etiological factors. The lesion grows rapidly and reaches a significant size within several months of its initial diagnosis.

PGCG is more common in the fifth and sixth decades of life than in children, and females are more commonly affected than males. In this paper we reported a case of PGCG located on buccal and lingual attached gingival of the mandibular anterior region in a young female.

Gingival lesions in children that mimic the PGCG are the pyogenic granulomas, parulis, and hemangiomas. The pyogenic granuloma presents as a soft, friable nodule that bleeds freely with minimal manipulation. Another erythematous nodule of the gingiva is the parulis, which is associated with an entrapped foreign body, a gingival pocket or a non vital tooth. Pain and expression of purulent exudates with fluctuation in lesion size help to differentiate this inflammatory disease from the PGCG. Another diagnostic hypothesis is hemangioma based on a red or blue discoloration of the soft tissue nodule although many hemangiomas are congenital lesions, some vascular malformations increase in size during childhood. Brisk bleeding, increased warmth of the tissue and blanching upon palpation are characteristic of this vascular entity.

To establish a definitive diagnosis, histopathological examination is required and it is characterized by highly vascularized connective tissue with extravasations of multinucleated giant cells and erythrocytes. Bernier and Cahn believed giant cells to be phagocytic response to hemorrhage in a preexisting granulation tissue. Some authors consider giant cells to be derived from osteoclasts while others believe that they are derived from mononuclear histiocytes. Flanagan, et al gave definitive evidence of the resemblance of the multinucleated giant cells to osteoclasts and it has been on the basis of excavation of giant cells from bone in-vitro. This property is not exhibited by macrophages and other mononuclear phagocytes which are morphologically, functionally and histogenetically related to osteoclasts. Although incipient lesions may bleed and cause minor changes in gingival contour, progressive growth in
some cases produce a significant tumescence that compromises normal oral function. Early detection of the PGCG results in more conservative surgery with less risk of tooth and bone loss. Management of this gingival lesion includes surgical excision and elimination of any local contributing factors. Recurrences of the PGCG have been reported in 5% to 70.6% of cases. This great variation is probably attributable to the surgical technique used, since recurrences re-excised up to the periosteum have not recurred thereafter.

CONCLUSION

In conclusion, in pediatric dentistry careful examination of oral mucosa is important to identify reactive lesions such as PSCG. These lesions are not clinically aggressive or invasive, but some of them may interfere with the eruption of teeth, produce tooth movement and lead to bone resorption as seen in this case report. Early detection of the PGCG results in more conservative surgery with less risk for tooth and bone loss.

References

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