ORIGINAL RESEARCH PAPER

Dental Science

KEY WORDS: Giant cell epulis, giant cell reparative granuloma, giant cell hyperplasia.

PERIPHERAL GIANT CELL GRANULOMA- A CASE REPORT.

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ABSTRACT

Peripheral giant cell granuloma (PGCG) is the most common oral giant cell lesion. It is a benign reactive lesion of gingiva which manifests as a firm, soft, bright purplish- red nodule or as a sessile or pedunculated mass consisting of multinucleated giant cells in a background of mononuclear stromal cells and extravasated red blood cells.
It is localized to attached gingiva, or on alveolar mucosa, but never found on non osseous supported tissue and arises interdentally or from gingival margin. This lesion probably does not represent a true neoplasm, but rather may be reactive in nature, but the cause is not certainly known. This article reports the management of peripheral giant cell granuloma in 25 year old female by surgical excision. There was no residual or recurrent swelling or bony defect apparent in the area of biopsy after a follow-up period of 3 months.

INTRODUCTION:

Peripheral giant cell granuloma (PGCG) is the most common oral giant cell lesion appearing as a soft tissue extra osseous purplish-red nodule consisting of multinucleated stromal cells and extravasated red blood cells[1], vary in appearance from smooth regularly outlined masses to irregularly shaped multilobulated protuberance with surface indentations. It is painless, varies in size, and may cover several teeth. The growth is firm or spongy with the peak prevalence in fifth and sixth decades, 20 to 33% occur within the first two decades of life. PGCG affects mandible (55%) more than maxilla; mandible to maxillary predilection is 2.4:1.[2] PGCG bears a close microscopic resemblance to central giant cell granuloma, and some pathologists believe that it may represent a soft tissue counterpart of the central bony lesion.[3]

Incisor and Canine region is the most common sight, with a slight predilection for the mandible. PGCG is found more commonly in females (65%) than in males (35%).[4] Complete removal leads to uneventful recovery. Early and precise diagnosis allows conservative management without risk to adjacent teeth or bone.

CASE REPORT:

25 years old female who was systematically healthy, reported to the department of Periodontology with chief complaint of enlargement in gums in left lower back region of mouth since 3 years. (Figure: 1)

FIGURE: 1

Clinical Examination:

Deep carious lesion with grade II mobility in left mandibular 1st molar was seen.

Clinical appearance: Smooth, non tender lesion which blend with minimal manipulation.

History and Investigations:

Patient was systemically healthy and not taking any medications. Her haemogram revealed normal limits.

RADIOGRAPHIC EXAMINATION:

Intraoral periapical (IOPA) radiograph shows evidence of **horizontal bone loss** in left mandibular premolar and molar region. (Figure: 2)

Orthopantomogram (OPG) revealed bone loss throughout the mandible but severe horizontal bone loss in left mandibular premolar and molar region with involvement of furcation area in second molar region. (Figure: 3)

Therefore surgical removal of PGCG was expected. The procedure was explained to the patient and an informed consent was obtained.

FIGURE: 2

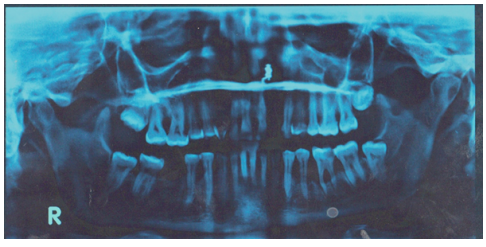

FIGURE: 3

Treatment:

After Scaling and Root Planing (SRP), Root canal treatment of left mandibular 1st molar was initiated. An **excisional biopsy** of the lesion was performed using an internal bevel gingivectomy and sutures were placed. Biopsy specimen was embedded in 10% formalin and sent for examination. Antibiotics and analgesics were prescribed for 5 days. (Figure: 4,5,6,7,8)

FIGURE: 5 - Flap Reflection

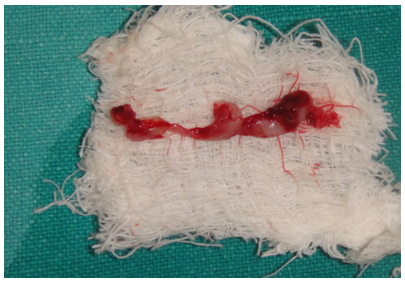

FIGURE: 6 - Tissue Excised

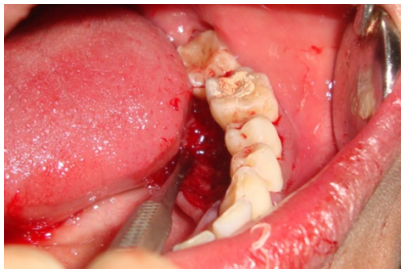

FIGURE: 7- Flap Debridement

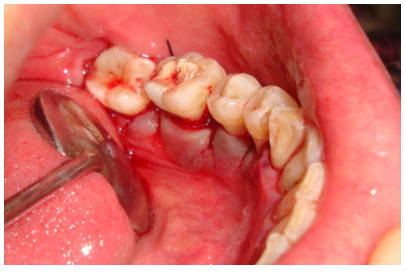

Figure: 8 - Flap Sutured

Histopathological examination:
Parakeratinized stratified squamous epithelium and fibrocellular connective tissue with numerous plump cells proliferating in sheets and whorls. Numerous multinucleated giant cells were seen to be dispersed throughout tissue suggestive of PGCG. Diffuse chronic inflammation was also seen along with numerous blood vessels. (Figure: 9)

3 months post operatively: no residual or recurrent, swelling was seen and lesion healed uneventfully. (Figure: 10)

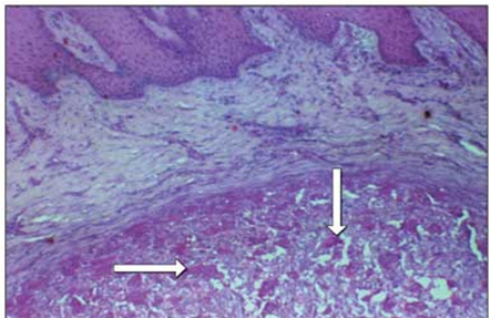

FIGURE: 9 – Histopathological Findings

FIGURE: 10 - 3 Months Post Operative Image

Figure:11(A) Pre Operative

Figure:11(B) 3 Months Post Operative

DISCUSSION
Giant cell granuloma is a benign, non odontogenic, moderately rare tumors of the oral cavity.Peripheral giant cell granuloma(CGCG) has history of more than a hundred years[5]. It originates from periosteum or periodontal membrane following local irritation or chronic trauma such as poor dental restorations, dental extraction, plaque, and calculus accumulation. Recurrence rate: 5 to 70.6%.Pain is rare and in most cases the lesion is induced by constant trauma. A possible hormonal influence has been postulated by ***Whitacker and Bouquot***.[6]

Jaffe HL through his research affirmed that the giant cell tumors occurring at other areas of the body were poles apart from the giant cells found in the jaws and termed them as giant cell reparative granuloma.[7]

Nedir et al,[8] said that surgical excision revealed superficial resorption of cervical region of involved tooth. Root resorption although extremely rare, may be associated with PGCG. In rare instances, PGCG is an oral manifestation of hyperparathyroidism without obvious central bony involvement.

Berneir and Cahn suggested that these lesions should be called as either a peripheral or central giant cell reparative granuloma.[9] ***Bhaskar et al.*** in 1959 subdivided giant cell granuloma into central and peripheral type. A non- neoplastic lesion by nature, the giant cell granuloma is distinctive in its histologic makeup. It contains multinucleated giant cells embedded in a stroma environment composed of mononucleated stromal cells along with ovoid to spindle- shaped nuclei.[10]

Sidhu MS explained that giant cell granulomas occurring within the bone are called central giant cell granuloma (CGCG) and those occurring on edentulous alveolar processes or gingivae are called PGCG. All though the CGCG is rare in nature, making up 7% of total benign lesion of the jaw, it is at times uncompromising in nature, especially in young patients.[11] While contrary to that, ***Katsikeris N*** and fellow researchers stated that PGCG is a more common giant cell lesion of the jaw and can arise either in response to local irritation or from the connected tissue of the gingiva, periodontal membrane or from the periosteum of the alveolar ridge.[12]

Adhlakha VK discussed that histopathology of the CGCG centres around three main features- presence of numerous young proliferating fibroblast, vascularised fibrocellular stroma with numerous capillaries and abundant multinucleated giant cells.[13]

Kaya GS said that the spectrum of focal proliferative growth

occurring on gingival tissue that has a close resemblance with PCGC includes pyogenic granuloma, hemangioma, CGCG, peripheral ossifying fibroma and metastatic carcinomas[14]. In some cases a detailed examination of this lesion reveals vertically oriented bony spicules at the base of the lesions; this can be attributed to the foci of bone metaplasia. PGCG being a type of reactive lesion, radiograph occasionally demonstrate irritating factors such as subgingival calculus[14].

A recurrence rate of 5% has been reported ***Giansanti and Waldron***[16]. While the study by ***Eronat Nand his colleagues*** showed a recurrence of 11%[17].

Regezi JA stated that recurrence is believed to be related to lack of inclusion of the periosteum periodontal ligament in the excised specimen.[18] A re- excision must be performed for these cases. PGCG lesions are self limiting hence a recommended management aims at elimination of the entire base of the growth accompanied by eliminating any local irritating factor[19].

DIFFERENTIAL DIAGNOSIS:

Central giant cell granuloma, located within the jaw itself, exhibit a more aggressive behavior and only radiological evaluation can establish a distinction. Osteoblastic sarcoma, differentiated by the uniformity of the stromal cells and by the lack of dysplasia in these cells. In young persons, however, numerous mitotic figures and active proliferation of stromal cells make this distinction difficult. It is indistinguishable it is differentiated from the rare extraosseous brown tumor of hyperparathyroidism.

CONCLUSION

PGCG is a worldwide designated and acknowledged terminology. The lesion is usually asymptomatic and is not clinically aggressive or invasive however repeated trauma due to certain occlusion forces can lead to its growth. The factors responsible for PGCG growth are compromised systemic health, poor oral hygiene, oral dryness and ill fitting dentures. A definite diagnosis of this lesion on the basis of clinical, radiographic and histopathological examinations allow us to manage it with minimal risk to adjacent hard tissues, therefore excising and eliminating the local contributing factors.

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