

Management of Peripheral Giant Cell Granuloma In A Medically Compromised Patient-A Case Report

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1. INTRODUCTION

Oral growths are frequently encountered by clinicians in their routine life. Some growths are limited to gingiva only while some are linked with osseous lesions. Even, the clinical appearance of some lesions looks so similar that diagnosis becomes really difficult. Some lesions are reactive in nature while some are neoplastic. Among the reactive lesions, Peripheral Giant cell granuloma is a rare clinical entity which is considered as a separate entity due to presence of Multinucleated Giant cells.¹ There are many synonyms for this lesion namely giant cell epulis, Giant cell reparative granuloma or Giant cell hyperplasia.² This clinical entity is limited to soft tissues only and like its other counterpart, Central giant cell granuloma usually does not invade bone.³ The central Giant cell granuloma and peripheral Giant cell granuloma.¹ According to his concepts, peripheral giant cell granuloma is limited to soft tissues while central giant cell granuloma being aggressive in nature involves bone also.² There are certain aggravating factors which include presence of local irritants namely calculus, bacterial plaque, ill-fitting dentures or even the tooth extractions.² It is not merely limited to middle and old age group but children are also affected with average age of 38-42 years.¹ However presence of certain factors like Xerostomia, deficient oral hygiene or presence of dental implants do play an important role in the progression of disease.^{3,4} The appearance of the lesion can be either nodular or poly-ploidal in nature with presence of bluish red or smooth shiny surface.⁴ Even size is also variable within average range up to 2 cm and can even reach up to 5 cm.⁵ The site of occurrence is not fixed but as per findings in a systemic review, there is predilection at the anterior portion of the mandible (60%).⁶

The treatment options include the excision of the tissue in a complete manner to avoid reoccurrence. The prime aim should not only be the excision only but removal of the etiological agent also. However presence of any medical condition like hypertension, hyperthyroidism or diabetes mellitus do create a hindrance in management of the lesion. Even an association has been found between this entity and hyperparathyroidism which was associated with renal failure also.⁷ There can be chances of intra-operative & post-operative hemorrhage that increase the chances of reoccurrence. The aim of this case report is to highlight the sequence of events that are required in the management of peripheral Giant cell granuloma in a medically compromised patient.

2. CASE REPORT

A 33 year old male with a history of gingival overgrowth reported in the OPD of Department of Periodontology MMCDSR Haryana , Ambala. There was a history of trauma 3-5 months due to tooth brushing which resulted in massive gingival enlargement. On clinical examination, there was presence of edentulous region in the 46 tooth region (figure-1). The pedunculated lesion was evident with soft consistency .Also there was complete adherence of the lesion which was confined from 46-47 tooth region (edentulous) with prominence at the facial surface. The colour of the region was light pink resembling the adjacent mucosa while some areas showed reddened appearance with hemorrhagic tendency along with

presence of spontaneous bleeding on slight provocation. The size of the lesion was quite enormous ranging from 1.5 x 1 (cm)-figure-3. On radiographic examination, there was absence of any kind of calcification or cortical resorption (figure-4). However, prominent bone loss was present which was localised in that specific region. It was decided to perform biopsy of the gingival over growth. However, shockingly medical history revealed presence primary hypertension from almost 3 years for which patient was not taking any medication. Pre-operatively, the blood pressure was recorded which surprisingly, came out to be 210/140 mm hg. Patient was deferred for the treatment and advised to consult physician regarding immediate starting of the medications. Patient reported 30 days back after getting operated for hypertension. Again, the blood pressure was recorded which was now within the normal range. Patient was informed about the details and complications associated with the biopsy procedure and a written informed consent was taken from the concerned physician also prior to the surgical protocol.

After proper investigations, the patient was given written and verbal information on the nature, risks and benefits of the procedure and a written signed, informed consent was obtained prior to the treatment. The procedure was performed under local infiltration (2 % lidocaine, with adrenaline 1:2,00,000).It was completely excised from the base (figure -2,3). Further, it was followed by curettage and debridement of the remaining tissue to prevent chances of any reoccurrence. Dressing was given on the excised tissue region and post -operative instructions were reinforced. Wounds were left open to heal by granulation and secondary epithelization; therefore, no sutures were required.

Patient was recalled after 1 week,1 month and after 6 months for post-operative follow up (figure 6-8). Healing was completely uneventful with no chances of any post-operative infection or reoccurrence.



Fig.-1 .Pre-operative lesion ranging facially from 46-47 tooth region (edentulous).



Fig.-2 .Post-operative view after complete excision of lesion .



Fig.-3 . Excised massive overgrowth.

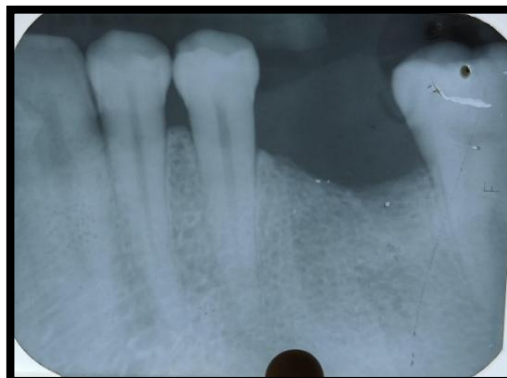


Fig.-4 .Radiographic examination showing localised Bone loss with respect to 46 tooth region.



Fig.-5. H&E examination showing multinucleated giant cells.



Fig.-6 .Post-operative 1 month examination showing uneventful healing.



Fig.-7 .Post-operative 6 month examination (lingual view)



Fig.-8 .Post-operative 6 month examination (facial view)

3. HISTOPATHOLOGICAL EXAMINATION

Haematoxylin & Eosin (H & E) stains revealed presence of para-keratinized stratified squamous epithelium overlying the connective tissue component. The connective tissue was fibrovascular in nature with high degree of cellularity. Numerous blood vessels engorged with RBCs were noted along with presence of multinucleated giant cells spreading over the connective tissue. Also, focal areas showed presence of extra-vascular RBCs.

4. DISCUSSION

Peripheral giant cell granuloma (PGCG) being a rare entity, is assumed to be considered as a reactive lesion rather than a neoplasm. The triggering factor for this lesion is considered to be local irritation or trauma, but the cause is not clearly justified.⁸ The incidence is also variable with striking rate of 64.6% (maxilla) with preference at the anterior region (5.3%).

Even some authors have suggested this lesion to be of reparative nature but have not found any relationship with the presence of osteoclastic activity. Contradictory to this, some have recommended the involvement of osteoclasts due to attachment of membrane like receptors like calcitonin which were clearly demonstrated in in-vitro studies.^{9,10} Additional to these findings,

some authors have emphasized the importance of mononuclear phagocyte system for the etiology of peripheral giant cell granuloma.¹¹ The PGCG is considered to bear a very close microscopic resemblance to the central giant cell granuloma and is often considered to be its soft tissue counterpart.¹²

Hence, differential diagnosis is must to confirm this entity from other lesions. Merely on the basis of clinical features, it is not easy to differentiate this lesion from other similar entities like pyogenic granuloma, fibrous epulis, peripheral ossifying fibroma, inflammatory fibrous hyperplasia, peripheral odontogenic fibroma, hemangioma caverosum and papilloma. Microscopic examination is must for the definitive diagnosis.¹³ Clinically this lesion has a lot of similarities with pyogenic granuloma and peripheral ossifying fibroma but contrary to the latter two clinical conditions, the former shows a striking bone resorption along with preference on the edentulous region while Pyogenic granuloma has it preference on gingiva followed by tongue and buccal mucosa.^{14,15} Peripheral ossifying fibroma can occur on palate also.¹⁵ On microscopic examination, the lesion shows some association in the form of attachment with periodontal ligament or mucoperiosteum. On comparison with giant cell epulis, there is presence of stratified squamous epithelium which is not observed in peripheral giant cell granuloma.¹⁴ Cherubism is another giant cell lesion showing similarities with Peripheral giant cell granuloma. The absence of perivascular cuffing can help differentiate the latter and its counterpart Central giant cell granuloma from cherubism.¹⁷ Also, presence of foreign body type giant cells and absence of stromal tumor cells differentiate this lesion from a Giant cell tumour or GCT.¹⁸ Solid aneurysmal bone cyst has large blood spaces while there is Normal serum calcium, parathyroid hormone, alkaline phosphatase and phosphorous levels distinguish this lesion from brown tumor of hyperparathyroidism. Radiographic examination generally shows no findings as because the nature of lesion appears to be as a soft tissue mass.¹⁶ However, the PGCG develops within soft tissue and shows **cupping** or **superficial resorption** of the underlying alveolar bony crest and clinically it is really difficult to determine whether the soft tissue peripheral lesion came out as peripheral or a central giant cell granuloma which first erodes through the cortical plate and then invades the gingival soft tissues. The most striking feature of Peripheral giant cell granuloma is presence of a non-encapsulated highly cellular mass with abundant multinucleated giant cells, inflammation, interstitial hemorrhage, abundant calculus and tooth mobility.^{8,15}

Among the various cells present in the clinical entity, fibroblasts are the basic element of peripheral giant cell granulomas. There are many young fibroblasts which later form multinucleated giant cells which contain abundant eosinophilic cytoplasm. This appears to be non-functional owing to the biological processes of phagocytosis and bone resorption.¹⁴ On further examination, there are two types of giant cells which are mainly found. First one is metabolically active cells or Type I and the second is dying cell or Type II. The origin of these cells has not been defined yet. However, there are a lot of similarities among both these type of cells.¹²

Histologically, PGCG is composed of nodules of multinucleated giant cells along with abundant spindle-shaped mesenchymal cells and extravasated red blood cells. The giant cells may contain only a few nuclei or up to several dozen. Among these nuclei, some are large and vesicular while others are small & pyknotic nuclei. The origin of the giant cell is still unknown. unknown.⁸ For etiology, various hypotheses had been proposed to explain the nature of multinucleated giant cells. First one states that osteoclasts left from physiological resorption of teeth or reaction to injury to periosteum.⁵ Even evidences suggest that osteoclasts possess receptors for calcitonin and which were easily excavated through bone (in vitro) (Flanagan et al 1988).^{1,5} In one of the supportive study by **Lim & Gibbins (1995)**, it was observed that multinucleated giant cells reacted strongly against a monoclonal antibody (MB1) which reacted with lymphocytes along with T cells and monocytes.¹⁷ The MB1 antibody was earlier shown to be expressed by osteoclasts in fetal bone and further, re-expression of the blood vessels in the lesion by endothelial cell marker factor VIII related antigen failed to show any presence of blood vessels. However, they were observed on the periphery of the lesion with vascular nature extremely. As already proven about the reactive nature of giant cells, there can be chances of their active response which is for shorter duration and compared to stromal cells, they disappear very early.^{8,18}

Another study by **Wulling et al (2001)** revealed that the stromal cells secrete a variety of cytokines and differentiation factors namely Monocyte chemo-attractant protein-1 (MCP1), Osteoclast differentiation factor (ODF) and Macrophage-colony stimulating factor (M-CSF).¹⁹ These molecules are considered as monocyte chemo-attractants and are essential for functioning of osteoclasts. It was found in a study by **Liu et al (2003)** that the receptor activator of NF-kappa B ligand (RANKL) which is an essential component to perform osteoclastogenesis, along osteoprogenin (OPG) are completely necessary for formation of multinucleated giant cells.²²

Treatment options include complete excision of the lesion from the base. In addition, removal of local irritants is must to avoid high reoccurrence rate, owing to almost 10%. Also, in addition there can be chances of esthetic and functional soft tissue defects on account of incomplete removal of the growth.¹ Also, it has been suggested that additional curettage after excision reduces the chances of re-occurrence in almost 85 % of cases.²³

As per our knowledge, this is a first case report of patient with primary hypertension. Case have been reported with malignant hypertension with pregnancy & hyperparathyroidism in which hypertension was a secondary feature.^{23,24} However no direct association was found in these studies which could be attributed to the occurrence of Peripheral giant cell granuloma.

Similarly in our case also, no hypothesis could be suggested by linking the occurrence of Peripheral giant cell granuloma and Hypertension. However, every precaution was taken to prevent excess intraoperative and postoperative hemorrhage as per the latest guidelines for management of hypertensive patients with dental complications/ co-morbidities.²⁵ Fortunately, the healing was also uneventful and no reoccurrence was noted post operatively.

5. CONCLUSION

Oral growths usually do not have the malignant potential. But sometimes reoccurrence along with local irritation can trigger the chances of transformation of normal lesion to malignancy. Presence of any systemic disease can prolong the healing and affect the final recovery. Hence clinicians should aim to completely excise the tissue and manage the associated systemic condition as per the latest clinical protocol.

6. CONFLICT OF INTEREST

The Authors declare no conflict of interest.

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8. ETHICAL COMMITTEE APPROVAL

Not required.

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