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Case Report- Peripheral Giant Cell Epulis

Dr.Vijay Mehetre*,Dr. Varsha Ranmare,Dr.Alkesh Shende***, & Dr.Priyadarshani Gir******

**Senior lecturer, Department of periodontology,A.C.P.M dental college, Dhule*
***P.G Student, Department of periodontology,M.G.V's K.B.H dental college, Nashik*
****P.G Student, Department of periodontology,A.C.P.M dental college, Dhule*
*****P.G Student, Department of oral and maxillofacial surgery,A.C.P.M dental college, Dhule*

ABSTRACT :

Peripheral giant cell lesions (PGCL) are reactive, extraosseous (soft gum tissue) and exophytic,is a non-neoplastic, tumor-like reactive lesion. It originates from theperiosteum or periodontal membrane following local irritation or chronic trauma.Early and definite diagnosis correlating clinical, radiologic and histopathologic examination is important for conservative management of such lesion thus eliminating potential risk to adjacent hard tissue structures.

Keywords :-*periphearl giant cell lesion,giant cells,local irritation,reactive.*

INTRODUCTION:

Peripheral giant cell granuloma is one of the reactive hyperplastic lesion accounting 7% of all benign tumors of the jaw. It is the most common oral giant cell lesion appearing as a soft tissue extra-osseous purplish-red nodule consisting of multinucleated giant cells in a background of mononuclear stromal cells andextravasated red blood cells. It is occurred in response to local irritation such as tooth extraction, inadequate dental restorations, ill-fitting dentures, plaque, calculus, food impaction and chronic trauma .[1]

It is more frequent in women than in men, with a slightly higher prevalence in the 30- to 70-year-old-age group, and largely affects the lower jaw (55%) than in the upper jaw. Cases of PGCG have been documented in children, where the lesion appears to be more aggressive, with infiltration of the interproximal crest area, displacement of the adjacent teeth and multiple recurrences.[2]

Case history:-

A 37 years old female patient reported to dental clinic with the chief complaint of swelling in upper right anterior region of jaw since 6 months The swelling had reached the present size with no evidence of further growth since past one month. It was associated with pain and difficulty in eating with negative history of trauma or any other associated significant history.

On inspection, the solitary swelling was 2 x1cm in size extending from permanent right maxillary lateral incisor to permanent right first premolar region[Figure 1].. Thelesion was sessile, irregular with overlying surface erythematous and ulcerated. On palpation the lesion was soft and tender with tendency to bleed.

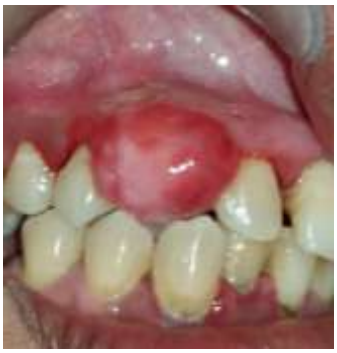

Fig.1

Surgery (excisional biopsy) was planned under local anesthesia (LA). The overlying mucosa was incised and undermined. Lesion was separated from the adjacent tissue by blunt dissection and removed in one piece [Figure 2]. Primary closure was done with 3-0 silk suture [Figure 3]. The specimen was sent for histopathologic examination. Sutures were removed after 1 week. There was no evidence of recurrence till 5 months of follow-up

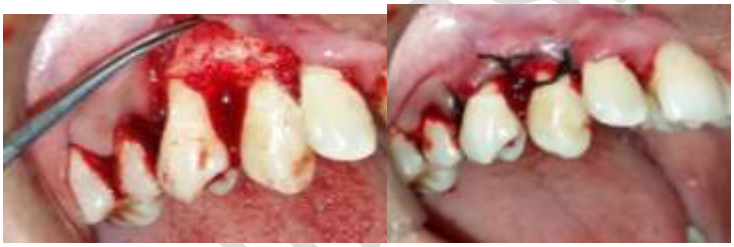

Fig.2 Fig.3

Discussion:- The PGCG occurs throughout life, with peaks in incidence during the mixed dentitional years and in the age group of 30–40 years.It is more common among females (60%). The mandible is affected slightly more often than the maxilla.[3]Lesions can become large, some attaining 2 cm in size. The clinical appearance is similar to that of the morecommon pyogenic granuloma, although the PGCG often is more bluish-purple compared with the bright red color of a typical pyogenic granuloma. Recently, the PGCG associated with dental implants has also been reported.[4]

Although the PGCG develops within soft tissue, "cupping" superficial resorption of the underlying alveolar bony crest issometimes seen. At times, it may be difficult to determine whether the mass is a peripheral lesion or a central giant cellgranuloma eroding through the cortical plate into the gingival soft tissues.[5]

On the other hand, Gunhan et al[6] in their study on 26 PGCG cases suggested that these lesions could be influenced by sex hormones, and the giant cells were to be a potential target for estrogen (but not progesterone) action. In rare cases, giant cell granulomas are oral manifestations of hyperparathyroidism, when multiple lesions are identified and the patient suffers recurrences in spite of adequate treatment. The lesions typically associated with hyperparathyroidism appearcentrally in bone and are referred to as brown tumors. In the lower jaw, these intrabony lesions can perforate the cortical layer, spreading towards the soft

tissues and imitating a peripheral lesion. A parathyroid tumor or chronic renal failure primarily or secondarily can give rise to increased parathyroid hormone (PTH) production, which in turn favors the formation of giant cell lesions. Children with hypophosphatemic rickets (subclinical hyperparathyroidism) are also at anincreased risk of developing such lesions. Histologically, brown tumors cannot be distinguished from giant cell granulomas.[7]

The treatment of PGCG comprises surgical resection with elimination of the entire base of the lesion and suppression ofthe etiologic factor. If resection is only superficial, the growth may recur. Most lesions respond satisfactorily to thorough

surgical resection, with exposure of all the bone walls. When the periodontal membrane is affected, extraction of theadjacent teeth may prove necessary to insure full resection though this is initially contraindicated.

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