GIANT CELL LESION OF ORAL CAVITY: A CASE REPORT OF PALATAL PERIPHERAL GIANT CELL GRANULOMA

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ABSTRACT:

Peripheral Giant cell granuloma also commonly known as "giant cell epulis" is classified under the giant cell lesions of the oral cavity. It is a non-neoplastic, tumor-like reactive lesion originating from the periosteum or the periodontal ligament or occurring as a result of local trauma or chronic irritation. The present manuscript describes a case of Peripheral Giant cell granuloma involving the palatal mucosa in a 40 year old Indian male patient. The significant clinical and radiographic features, management and prognosis of the same are discussed along with the review of literature. Clinically it mimics vascular disorders like hemangioma, pyogenic granuloma and peripheral ossifying fibroma, thus necessitating the need for a histopathological confirmation.

Key words: Peripheral Giant cell granuloma, Palate, reactive, giant cell epulis

INTRODUCTION:

PGCG is an exophytic lesion of the oral cavity, also known as osteoclastoma, giant cell reparative granuloma, giant cell hyperplasia.^[1] It has been described as a reactive lesion of the jaw occurring on the gingival and alveolar ridge, also believed to be soft tissue counterpart of the central bony lesion by some.^[2] Usually presents as an asymptomatic nodular, sessile or pedunculated growth.^[1]

Radiographic features reveal non specific changes in the bone, sometimes superficial erosion of the underlying bone seen.^[3] be Histologically, may multinucleated giant cells may be seen, the origin of which is unknown; some believe them show to immunohistochemical features of osteoclasts, while others suggest them to from arise mononuclear phagocyte system. Other possible sources include osteoblasts, endothelial cells and spindle cells.PGCG seems to be influenced by hormonal stimulus, especially estrogen.^[4]

*Corresponding Author Address: Dr. Priyanka Bhat, Dept of Oral Medicine, Diagnosis and Radiology, SGT Dental College, Hospital and Research Institute, Gurgaon, Haryana (India).. Email: pbpriyankabhat@gmail.com It is the most common giant cell lesion of the oral cavity which is not a true neoplasm but believed to be a reactive lesion.^[1] The purpose of this case report is to illustrate an example of PGCG, to discuss and compare it's identifying clinical, radiologic and histopathologic picture which can help in it's definitive diagnosis.

CASE DETAIL:

A 40 year old male patient reported to the Department of Oral Medicine and Radiology, SGT Dental College with the complaint of a painless growth in the right upper back region of the jaw since 15-20 days.History revealed that the growth started as small pea sized and progressively increased to the present size over a period of 1 month.

History of bleeding was also given by the patient. Bleeding was initiated on irritation of any kind and mostly on brushing around that area, stopped on its own after a few minutes. No pain or pus discharge associated with it. The patient was systemically healthy, and we also ruled out any history of fever, loss of appetite or loss of weight. No similar growth was observed by the patient in any other part of body.

His intraoral examination revealed a bluish red, raised, smooth-edged nodular mass in the right upper jaw arising from interdental gingival extending to involve the palatal mucosa wrt 13,14 region measuring 1 cm in diameter, with distinct margins and erythematous overlying mucosa with a lobulated surface .The growth was sessile, soft in consistency firm in consistency, non-compressible, non fluctuant, non reducible, sessile, non tender, bleeding present on manipulation. No discharge present. Blanching seen in some areas on palpation. Grade II mobility was present wrt 13,14. Based on the history and clinical examination, a provisional diagnosis of pyogenic granuloma was arrived at. The differential diagnosis included Peripheral giant cell giant cell granuloma, cental giant granuloma, peripheral ossifying fibroma was given.

Routine blood tests were found to be normal. Intraoral periapical (IOPA) radiograph, bone involvement beneath the lesion was seen that presented in the form superficial bone eroson. Peripheral cupping seen wrt 13,14 region. Horizontal bone loss seen with loss of lamina dura irt 15. The lesion was completely excised to periosteum level under L.A. and complete curettage was done. There were no complications in the immediate postoperative period. Biopsy specimen was embedded in 10% formalin and sent to department of pathology.

The specimen biopsied was processed for routine hematoxylin and eosin staining and 4-5 micron thick sections were prepared and examined under light microscope at 40X. The sections revealed well-circumscribed, unencapsulated cellular mass containing oval to spindleshaped fibroblasts, multinucleated giant cells, numerous capillaries and areas of hemorrhage. The multinucleated giant cells were of variable shapes and sizes containing open-faced nuclei ranging from 5 to 7 in number Many giant cells were found in association with and within blood vessels. The overlying epithelium was stratified squamous epithelium which was mildly hyperplastic. In addition the connective tissue also revealed certain foci of giant cells The histopathologic features were suggestive of peripheral giant cell granuloma.

The patient was kept under regular follow up and no recurrence was reported.

DISCUSSION:

PGCG is an ambiguous lesion first described by Jaffe as "giant cell reparative granuloma" to differentiate these from the giant cell tumors as he per his belief of the former lesions representing a local reparative reaction rather than a true neoplasm.^[5] Later Bernier and Cahn gave it the term "peripheral giant cell reparative granuloma".^[6] This terminology is currently not being used as the reparative nature of PGCG has not been proved. Currently, the term peripheral giant cell granuloma is universally and widely accepted.^[7]

The present report is regarding a case of PGCG successfully treated with excision and curettage. The clinical and radiographic 6months follow-up indicated no recurrence and suggested that the chosen management of surgery along with the scrupulous oral hygiene maintenance are adequate for treating prevent its recurrence.

PGCG may be encountered at any age, especially during the first through sixth

decades of life ^[1], however, the highest and commonest incidence (40%) is in the fourth to the sixth decades of life. ^[2,8,9] A slight female predilection has been reported in a large number of studies with the male: female ratio. 1:1.5.^[10] A female predilection of 60% has been reported.^[1] However, PGCG was more common among men (M/F 1.4:1) in a study by Zarei et al.^[11] Similar male predilections have been reported by Bhaskar SN et al.^[12] Salum FG et al.^[13], Chaparro- Avendano AV et al ^[14] and Peralles PG et al.^[15] The size of the lesion is usually smaller than 2 cm in diameter, although larger ones may be seen occasionally; diameter as large as 5 cm have been reported.^[16] Gradual growth in some cases produces an important tumor mass that adversely affects normal oral function.^[14] Bodner et al conducted a study according to which following factors contribute to the growth of PGCG are: compromised systemic health, poor oral hygiene ill fitting dentures and oral dryness.^[16]

Clinically it presents as a painless nodular mass soft in consistency, usually red to red bluish in color. The lesion appears blue-purple in color due to extensive hemorrhagic areas and hemosiderin deposition at the periphery. It varies in appearance from smooth, well demarcated regularly outlined mass to irregularly shaped, multilobulated protruberance with surface indentation. Ulceration of the margin is occasionally seen, secondary to trauma.^[1]

PGCG is seen in the anterior or posterior region of the gingival or the soft tissue

covering the edentulous alveolar ridge. Usually found in the gingival margin between teeth anterior to the permanent molars, with the premolar molar region of the jaw being the most common site of occurrence.^[17] 5% cases were reported on the palate in a study by Maryam et al.^[18] In the given case, the growth involved the palatal gingiva in a 40 year old male.

The mandible is affected slightly more often than the maxilla, the reported proportion being 2.4:1.24.^[14,19] However, the lesion in our case involved the maxillary arch. Upon palpation, lesion may either be soft or hard, depending on the composition collagen and/ or inflammatory components.^[10] In our case, the lesion was an exophytic, sessile nodular growth which was bluish-red in color, firm in consistency and was found on the palatal gingiva in relation to right maxillary canine region involving the interdental papilla.^[1]

Peripheral giant cell granuloma develops within soft tissue, radiographically presenting as "cupping" resorption of the underlying alveolar bone or the leveling effect.^[1,14] In some cases radiographic findings may point toward the possible irritational factor, in contrast to the granuloma.^[20] central cell giant Radiography thus becomes an important tool for determining whether the lesion is of gingival (i.e. peripheral) origin or bony (central) origin. Cupping resorption with evidence of superficial erosion of the crestal bone was evident in the present case.

Lesions mimicking PGCG makes the microscopic examination mandatory for definitive diagnosis. Factors such as history, duration, tendency of spontaneous bleeding on slight provocation, bone resorption helps in differentially diagnosing PGCG from pyogenic granuloma and hemangioma. Hemangioma's origin being congenital and color ranging from bright red to blue, the lesion exhibits brisk bleeding and blanching on palpation.^[21] The presence of a non vital tooth with pus exudates rules it out from parulis. Radiographic examination plays a vital role in ruling out peripheral Ossifying fibroma (POF) and central giant cell granuloma (CGCG); radiopaque calcifications at centre of the lesion being present in POF and radiolucency crossing midline in CGCG.^[21]

PGCG histopathologically shows three main features; first, presence of numerous young, proliferating fibroblasts; vascularized fibrocellular stroma with numerous capillaries, abundant multinucleated giant cells.^[22]

Treatment consists of surgical excision locally, down to the underlying bone, extensive clearing of the base being of utmost importance to avoid recurrence the growth. Elimination of irritant, local factors is also required.^[23,24] Recurrence rate of 5.0-7.6% (average 9.9%) has been reported in various epidemiologic studies ^[10] owing to superficial clearance of the lesion, while studies by Eversole and Rovin reported recurrence as 11%.^[25] Other treatment modalities includes cryosurgery using cryoprobe or liquid nitrogen and lasers. Laser surgeries offer advantages like bloodless field and minimal post operative discomfort.^[24]

CONCLUSION:

Thorough knowledge of biologic behavior, etiopathogenesis and going deep into the molecular aspects of this notorious lesion will aid the clinician, researchers and academicians in the categorization, **REFERENCES:**

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detection and definitive diagnosis. Thus target therapy with optimal patient care can be provided leading to reduction in the occurrence and reoccurrence rates.

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Vinod VC. et al., Int J Dent Health Sci 2015; 2(1): 248-254

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FIGURES:

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Figure 1: Intraoral presentation of the lesion



Figure 2: Intraoral presentation of the lesion

Vinod VC. et al., Int J Dent Health Sci 2015; 2(1): 248-254



Figure 3: IOPA radiograph showing Peripheral cupping wrt 13,14 region. Horizontal bone loss seen with loss of lamina dura irt 15.



Figure 5: Histopathological view 10X magnification



Figure 4: Histopathological view 40X magnification showing a giant cell