Peripheral Giant Cell Granuloma Manifestation in Pregnancy

Abstract
The peripheral giant cell granuloma (PGCG) is a benign oral lesion occurring on the gingiva and alveolar ridge. It is the most common oral lesion and occurs at an average age of 30 years. The upsurge in the levels of estrogen and progesterone in pregnancy leads to a plethora of changes in various parts of human body, including the oral cavity. In the oral cavity, changes are commonly seen on the gingiva. These include pyogenic granuloma, PGCG and also peripheral ossifying fibroma, etc., The etiology of PGCG in our case might be related to hormonal alterations during the gestation period.

Keywords: Mononuclear stromal cells, multinucleated giant cells, myeloid tumor, peripheral giant cell granuloma, pregnancy

Introduction
Peripheral giant cell granuloma (PGCG) is a relatively common reactive exophytic lesion of the oral cavity. The exact etiology of PGCG is not known. It may be due to an irritant or aggressive factor (trauma, tooth extraction, badly finished fillings, plaque, calculus, chronic infections, impacted food, etc.). The influence of hormones has been suggested as contributory factor. Clinically, PGCGs may present as polyploid or nodular lesions, predominantly bluish-red with a smooth shiny or mamillated surface with variable size, rarely exceeding 2 cm in diameter, and are generally soft or rubbery. Most commonly, they are asymptomatic, but due to interference with occlusion they may ulcerate and become infected. They show site predilection for premolar and molar mandibular regions. Treatment is usually by surgical excision and elimination of possible irritant factors.

Case Report
A 27-year-old female patient reported to the Department of Periodontology, Government Dental College and Hospital, Mumbai, with the chief complaint of painless enlargement in the lower left mandibular posterior region. Her history suggested that the lesion was present for the past 3 months of her pregnancy. The lesion remained static in size postpartum, i.e. for the past 1 month. On general examination, the patient was moderately built and well-nourished. Intraoral examination revealed a solitary polyloid growth measuring approximately 1.5 cm × 2 cm in size, interdentally in the region of 46 and 47 with lingual and buccal extension. The surface appeared lobulated, smooth, and shiny and showed ulceration. Color was of normal mucosa with areas of bluish-red discoloration [Figure 1]. On palpation, the growth was pedunculated, soft to firm in consistency, nontender and root pieces of the first molar were present as an irritating factor. There was interference in occlusion by the growth while chewing. There was generalized gingival inflammation, bleeding on probing, and calculus. The patient was advised for routine blood investigation, and all the parameters were in normal range. Radiographically, intraoral periapical radiograph showed root piece of 46 and bone resorption in the molar region. The patient was explained about the treatment plan. Scaling and root planning was carried out under phase I therapy and was treated accordingly with plaque control measures. Although plaque control measures improved the periodontal status dramatically, the lesion appeared static. A complete surgical excision was planned and a written consent was obtained.

Under local anesthesia, incision was taken with 12 number blade [Figure 2] and a full thickness mucoperiosteal flap was raised. A small polyploid growth was noted with lingual extension. On curettage, the lesion was found to be soft and friable. Histopathological examination revealed mononuclear stromal cells, multinucleated giant cells, and myeloid tumor cells [Figure 3]. The patient was advised for periodic follow-up. The patient was followed up after 3 months, and the lesion was found to be static in size and asymptomatic.

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reflected in 46 and 47 regions [Figure 3]. Root planing and flap curettage were done and irrigated with saline. It was followed by removal of the root piece of 46 [Figure 4] and then excision of the growth [Figure 5]. The flap was sutured and a periodontal dressing was placed. Postoperative instructions and medications were given. Excised specimen was sent for histopathological examination.

Microscopic examination of hematoxylin and eosin stained section revealed para keratinized, stratified, and squamous epithelium with a focal area of ulceration. The connective tissue showed dense inflammatory infiltrate predominantly lymphocytes and few plasma cells. Multi-nucleated giant cells and few budding capillaries were seen. In addition, few areas of reactive bone formation were seen [Figures 6 and 7]. All these features were suggestive of PGCG. The patient tolerated the procedure well and healed unremarkably on follow-up. Radiographically, the cupping resorption of underlying alveolar bone could be seen. The treatment is most frequently surgical excision and scaling of adjacent teeth to remove any source of irritation and to minimize the risk of recurrence. The most important thing is to do a regular follow-up as this lesion has a recurrence rate of 10%–15%.[3] Patients with poor oral hygiene and hormonal imbalance are more susceptible to large PGCG and should be examined occasionally.

One week postoperative, healing showed some marginal inflammation with receded gingival margin. The patient was comfortable, and the healing was uneventful. Sutures were removed, and proper maintenance program was initiated. During recall visits of 3 and 6 months, the surgical site appeared normal without recurrence of the lesion [Figure 8].

**Discussion**

PGCG is not a true neoplasm, but rather a benign hyperplastic reactive lesion caused by local irritation or chronic trauma. PGCG originates from the periodontal ligament or mucoperiosteum.[3] The etiology of PGCG is still unclear. Some investigators suggested that a history of trauma might be related to the development of the lesions. Other possible factors that have been implicated, but not definitely proven, include hormonal disturbances, primary hyperparathyroidism, tooth extraction, poor dental restorations, food impactions, ill-fitting dentures, orthodontic therapy, dental plaque, and calculus.[4]

Caillouette and Mattar[5] in their study found that PGCG and central giant cell granuloma are under the influence of the ovarian hormones. Chambers and Spector,[6] suggested

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**Figure 1:** Preoperative view of the lesion

**Figure 2:** Incision

**Figure 3:** Full thickness flap reflection

**Figure 4:** Removal of the root piece
PGCG to be enhanced by pregnancy rather than being “pregnancy-dependent.” The responsiveness of the gingiva to these hormones along with the immunosuppressive actions of the hormones may have contributed to the growth of the lesion. Shirani and Arshad stated that the ovarian hormones play no part in the development/growth of these lesions, but the evidence suggests that they may influence them only secondarily and their study result confirmed the role of poor oral hygiene in the initiation of PGCG. Furthermore, they concluded that the secondary influences of sex hormones with a background of poor oral hygiene or other stimulation factors are important in etiology of PGCG. In their study, they found that PGCG has a predilection to females and occurs more frequently in first four decades of life when hormonal changes are prominent. Age and sex predilection in our case is in accordance with the previous researches. The case included in the present study showed the characteristic clinical appearance of PGCG, i.e., nodular growth, soft, or rubbery on palpation, with a smooth, shiny, or mamillated surface. Most commonly, the cases reported in literature had lesion size varying from 1.0 to 2.0 cm in diameter, but masses more than 5 cm have rarely been reported. A radiograph revealed bone resorption in the molar region in this case. PGCG is a soft tissue lesion that presents on gingival and alveolar mucosa; radiographic features are thereby nonspecific. Occasionally, bone involvement beneath the lesion has been seen that presents as superficial bone resorption and thus, it can be discerned easily on periapical radiographs. Widening of the periodontal ligament space is also seen which is often accompanied by the mobility of associated teeth. Alveolar crest region or margin at interdental bone level of the lesion and associated teeth also exhibits resorption. PGCG being a type of reactive lesion, radiographs occasionally demonstrate irritating factors such as sub-gingival calculus. Radiographs are also important in distinguishing whether the lesion is of gingival (i.e., peripheral) origin or of bone (central) origin that spread toward the surface.

Histologically, as in our case, PGCG consists of a nonencapsulated mass of tissue composed of a delicate reticular and fibrillar connective tissue stroma containing large numbers of ovoid or spindle-shaped young connective...
tissue cells and multinucleated giant cells. Capillaries are numerous, particularly around the periphery of the lesion, and the giant cells sometimes may be found within the lumina of these vessels. Foci of hemorrhage, with the liberation of hemosiderin pigment and its subsequent ingestion by mononuclear phagocytes as well as inflammatory cell infiltration, are also characteristic features. Spicules of newly formed osteoid or bone are often found scattered throughout the vascular and cellular vascular lesion. Small to medium-sized vessels were seen at the periphery of the lesion admixed with chronic inflammatory infiltrate [Figures 5 and 6].

**Differential diagnosis**

Differential diagnosis of PGCG holds importance as there are a variety of other lesions on the list that mimics PGCG; and that are associated with a difference in their treatment and their prognosis. The spectrum of focal proliferative growths occurring on gingival tissue that have a close resemblance with PGCG includes pyogenic granuloma, hemangiomma, central giant cell granuloma, peripheral ossifying fibroma, and metastatic carcinomas that should be differentiated from nonossifying fibromas, which differ in consistency and coloration; central giant cell granulomas, which are expansive and destructive intraosseous lesions that can perforate the cortex; chondroblastomas and metastatic carcinomas, which when localized in the gingiva, may provoke irregular bone destruction below the exophytic lesion and hemangiomma, which, unlike PGCG lesions, are pulsatile and disappear under pressure. Pyogenic granulomas are difficult to differentiate from PGCG lesions on clinical grounds but may be distinguished histopathologically. There was no sign of malignancy in our case as indicated by the lack of nuclear or cytological atypism on histopathological examination. The surgical resection with the elimination of the entire base of the lesion and removal of the underlying etiologic factors has yielded successful results in the treatment for PGCG. The superficial resection may lead to recurrence of the growth. When the adjacent teeth are periodontally involved, their extraction may prove necessary to ensure full resection, although this is initially contraindicated. In our case, the lesion was surgically resected, and the involved root piece was extracted because it was periodontally involved and had a poor prognosis. There has been reported a wide variation of 5% to 70.6% in the recurrence rate of PGCG. In our case, no recurrence was noted at 1 year postoperatively.

**Recurrence**

The recurrence rate of 5%–70.6% (average 9.9%) has been reported in various epidemiologic studies. A recurrence rate of 5% has been reported by Giansanti and Waldron, whereas a study by Eversole and Rovin showed a recurrence of 11%. Recurrences are believed to be related to lack of inclusion of the periosteum or periodontal ligament in the excised specimen. A re-excision should be performed for these cases. PGCG lesions are self-limiting. Hence, recommended management of PGCG aims at the elimination of the entire base of the growth accompanied by eliminating any local irritating factors.

**Conclusion**

The hormonal influences during pregnancy may lead to the development of PGCG in patients with poor oral hygiene. The precise diagnosis of PGCG, based on the clinical, radiological, and histological findings is necessary so that extensive damage to the underlying bone and consequent need for tooth extraction can be avoided. Thorough knowledge and identification of etiopathogenesis and biologic behavior of this lesion will lead to a reduction in occurrence and recurrence rates of PGCG. Digging into a hidden molecular aspect of this lesion will aid us to design target therapies against PGCG thereby providing optimal patient care. A definite diagnosis of PGCG on the basis of clinical, radiographical, and histopathological examination allows us to do conservative management with minimal risk to the adjacent hard tissues.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

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