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### **RESEARCH ARTICLE**

# PERIPHERAL GIANT CELL GRANULOMA: RARELY OCCURRING, FREQUENTLY RECURRING

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#### **ABSTRACT**

Solitary gingival enlargements in children are a relatively common finding and are usually the result of a reactive response to local irritation. Giant cell reparative granuloma (GCRG) is not a true neoplasm but rather a reactive process; its origin could be triggered by trauma or inflammation. Peripheral Giant Cell Granuloma (PGCG) is least commonly diagnosed among various hyperplastic gingival lesions. It accounts for 7% of all benign tumors of the jaw. It is also known as peripheral giant cell reparative granuloma. This is a case report of a 14 year old boy which highlights the importance of depth of excision for successful treatment outcomes.

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## **INTRODUCTION**

Solitary gingival enlargements in children are a relatively common finding and are usually the result of a reactive response to local irritation. Although incipient lesions may bleed and cause minor changes in gingival contour, progressive growth in some cases produces a significant tumescence that compromises normal oral function. Giant cell reparative granuloma (GCRG) is not a true neoplasm but rather a reactive process; its origin could be triggered by trauma or inflammation (Waldron and Shafer, 1966; Jaffe, 19653). GCRGs are classified, according to location, as central or peripheral, occurring, respectively, in bone or gingival soft tissues (Fechner, 1984). Peripheral giant cell granuloma (PGCG) occurs exclusively on gingiva or edentulous alveolar ridge varying in size, maybe sessile or pedunclated and varies from deep red to bluish red in color. The clinical importance of these benign tumors is that they clinically mimic a malignant lesion (Ficarra et al., 1987).

### CASE REPORT

A 14-year-old boy reported to Department of Periodontology with a complaint of swelling in lower left back tooth region since 1 month. No increase or decrease in size of swelling was noticed in this duration.

JSS Dental College and Hospital, JSS University, SS Nagar, Mysore-570015, Karnataka, India There were no associated symptoms of pain or bleeding. There was no sensitivity to hot or spicy foods. Clinically a solitary, well-defined, smooth, shiny, sessile gingival enlargement measuring 2cm X 1cm in lingual aspect of 34, 35 & 36 tooth region was noted. It was pale pink in color with focal red areas. Other intra oral findings included generalized enamel hypoplasia and poor oral hygiene (Fig. 1.).



Fig. 1. Giagival Enlargement on the lingual side of Premolars

On palpation the lesion was non-tender, firm in consistency and on provocation delayed bleeding was noted. Radiographic examination included intra oral periapical radiograph (IOPAR) (Fig. 2), which did not reveal any appreciable changes. The patient underwent phase I periodontal therapy. Patient was recalled 15 days later for re-evaluation wherein no regression in condition could be noted (Fig. 3).

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Fig. 2. Pre-operative Radiograph



Fig. 3. After Phase 1 therapy

The patient was sent for routine blood investigations, which were normal. Surgical excision of the lesion was done a week later. A fresh band of calculus was exposed after excision, which was removed. The excised specimen was sent for biopsy (Fig.4).



Fig. 4. Excised Specimen

Histological diagnosis given for the lesion was Plasma Cell Gingivitis owing to presence of numerous inflammatory cells resembling plasma cells in connective tissue stroma. The clinical and histologic diagnosis did not seem to co-relate. The patient's dietary and oral hygiene history were taken which were inconclusive. The patient was kept on regular follow up. Clinically, 1 week and 1 month post operative healing was satisfactory but the patient was not able to maintain proper oral hygiene (Fig.5). Hence powered toothbrush was prescribed to aid in plaque control. 3 months post operatively the lesion started showing signs of recurrence with initially a pin point

growth at lingual attached gingiva in 35 36 region. The lesion rapidly progressed to a size of 6mm X 4mm within next 3 months. Re-excision was done involving a wider healthy tissue area and the tissue was dissected deep to the bone (Fig. 6 & 7). The area was thoroughly curetted which revealed bony dehiscence measuring 5-6 mm in length on second premolar and first molar. The excised specimen was again sent to lab for histopathology investigation.



Fig. 5. Once month post-operative



Fig. 6. Re-excision



Fig. 7. Bony dehiscence

Histopathology revealed Para keratinized stratified squamous epithelium. Connective tissue stroma consisting of fibres, fibroblasts, numerous blood vessels and extravasated RBC'S (Fig. 8). Numerous irregularly shaped giant cells were seen with multiple nuclei. Areas of ossification consisting of numerous bony trabeculae with plump fibroblasts were also noted (Fig. 9). Hence the diagnosis of PGCG was given

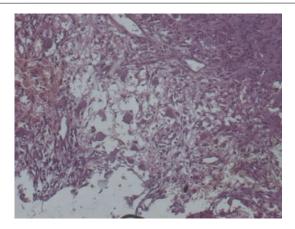


Fig. 8. Blood vessels & extravasated RBCs

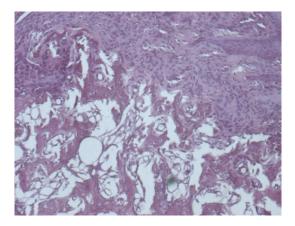


Fig. 9. Giant cells and areas of Ossification

### DISCUSSION

PGCG is least commonly diagnosed among various hyperplastic gingival lesions. It accounts for 7% of all benign tumors of the jaw. It is also known as giant cell epulis or peripheral giant cell reparative granuloma (Pour et al., 2008). Jaffe first suggested the term "giant cell reparative granuloma" for the similar central lesion of the jaw bones (Jaffe, 1953) to help differentiate them from the giant cell tumor (Motamedi et al., 2007) as he believed the former lesion to represent a local reparative reaction rather than being a true neoplasm (Pour et al., 2008). Bernier and Cahn proposed the term "peripheral giant cell reparative granuloma" for the lesion. (Katsikeris et al., 1988) The latter terminology is currently not being used, as the reparative nature of the lesion has not been proved (Carranza et al., 2009). Today, the term peripheral giant cell granuloma is universally accepted (Katsikeris et al., 1988). PGCG most frequently occurs due to local irritation from abnormal tooth morphology, faulty restorations, improperly fabricated prosthesis and poor oral hygiene (Bhaskar et al., 1971). In the present case the patient had enamel hypoplasia and the oral hygiene maintenance was poor. Patient was given oral hygiene instructions and powered toothbrush was provided for better brushing. PGCG is most commonly seen in mandibular region with female predilection and premolars being the most common site. It varies in appearance from smooth, well demarcated (Bhaskar et al., 1971) regularly outlined mass to irregularly shaped, multilobulated protuberance with surface indentation. The color of the lesion often varies from dark red to purple or blue. Upon palpation, one may note a lesion that is either soft or hard, depending on the composition of collagen and/or inflammatory components (Reichart *et al.*, 2000). Though the peripheral giant cell granuloma develops within soft tissue, "cupping" resorption of the underlying alveolar bone is sometimes seen radiographically (Regezi *et al.*, 2011; Neville *et al.*, 2009). Microscopically, the lesion arises from, or is at least attached to the periodontal ligament or mucoperiosteum (Bonetti *et al.*, 1990). The most characteristic histologic features include presence of non-encapsulated highly cellular mass with abundant giant cells, inflammation, interstitial hemorrhage, hemosiderin deposits and the presence of mature bone or osteoid (Katsikeris *et al.*, 1988).

Fibroblasts are the basic element of peripheral giant cell granuloma. Calcified tissue which is found in some of the lesions varies from small amorphous foci to well developed trabeculae. In present case besides other features a welldefined osteoid tissue surrounded by osteoblasts was present. Treatment consists of local surgical excision down to the underlying bone (Dayan et al., 2011), for extensive clearing of the base (Kfir et al., 1980). Removal of local factors or irritants is also required (Regezi et al., 2011). If resection is only superficial, the growth may recur, with an average of 9.9% cases showing recurrence (Reichart et al., 2000). In the present case initially the tissue dissection was not done deep to the bone. A layer of connective tissue was left behind on the bone. Signs of recurrence were noted as early as 3 months and progression in the size of lesion was also quite rapid. The lesion was re-excised deep down to the bone with thorough curettage. The 6-month follow-up of the case has shown no recurrence indicating that the given treatment along with maintenance of a good oral hygiene is sufficient to treat PGCG.

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