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RESEARCH ARTICLE

PERIPHERAL GIANT CELL GRANULOMA: AN ENORMOUS APPEARANCE

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ABSTRACT

The peripheral giant cell granuloma, also known as giant cell epulis, PGCL or giant cell hyperplasia, is the most common giant cell lesion in the oral cavity. It does not constitute a true neoplasm, but rather a reactive lesion caused by local irritation or trauma. Moreover, its etiology is still contentious. Previously, the lesion was called peripheral giant cell reparative granuloma. However, its reparative effect has not been proved yet, hence osteoclast activity seems doubtful. This paper presents a unique case of peripheral giant cell granuloma in an 11 year old boy who was referred for evaluation of a gingival mass approximately 2 cm × 1.5 cm in size extending from distal aspect of canine to mesial aspect of first permanent molar and covering the crowns of first premolar and primary second molar. It extended from the free gingival margin on the labial side of the first premolar and primary second molar to the attached gingiva on the lingual surface. Thus, management of such case presents a challenge to the practitioner to carefully excise the lesion and on the same hand protecting the underlying erupting permanent tooth.

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INTRODUCTION

Various types of localized gingival overgrowths are relatively common finding which occur as reactive lesions include pyogenic granuloma, peripheral giant cell granuloma (PGCG) and peripheral ossifying fibroma. In 1962, Gottsegen (Gottsegen, 1962) suggested that PGCG can arise following periodontal surgery. However, the etiology behind such type of lesions is uncertain, but authors suggest it to arise from trauma, microscopic organisms and plaque retainers like calculus, old restorations and prosthetic appliances (Jaffe, 1953). Because of its overwhelming incidence on the gingiva, the condition is associated with two other diseases pyogenic granuloma and peripheral ossifying fibroma. Because of its similar microscopic appearance to the bony lesions called central giant-cell granulomas, some researchers considered peripheral giant-cell granulomas to be soft tissue equivalent (Nedir, 1997). The purpose of this paper is to illustrate a case report on PGCG and also discuss treatment planning, histology and clinical features.

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Case history: An 11 year old boy was referred for evaluation of a gingival mass approximately 2 cm × 1.5 cm in size extending from distal aspect of canine to mesial aspect of first permanent molar covering the crowns of first premolar and primary second molar. (Figure 1) The swelling was first noticed by the patient 3 months back when he observed discomfort while eating, and it was gradually growing in size since then. Recently the lesion has doubled in size and is interfering with normal occlusion. The overlying mucosa resembled features of normal gingiva. It extended from the free gingival margin on the labial side of first premolar and primary second molar to the attached gingiva on the lingual surface. There was no associated lymphadenopathy. On further inspection, it seemed to be a smooth, hyperplastic, well-defined, vascular and sessile lesion. On palpation, it was non-tender, firm in consistency and did not show any blanching. No other oral or cutaneous lesions were noted in this healthy child. Radiographicaly, showed erupting first and second premolar and no other discrepancy was found. (Figure 2) (Figure 3) No systemic abnormalities were detected and hematological reports were noncontributory. Thus, it was decided to excise the lesion. The periodontal treatment plan included patient education and motivation for oral hygiene instructions, scaling and root planing, reevaluation and surgical excision of the

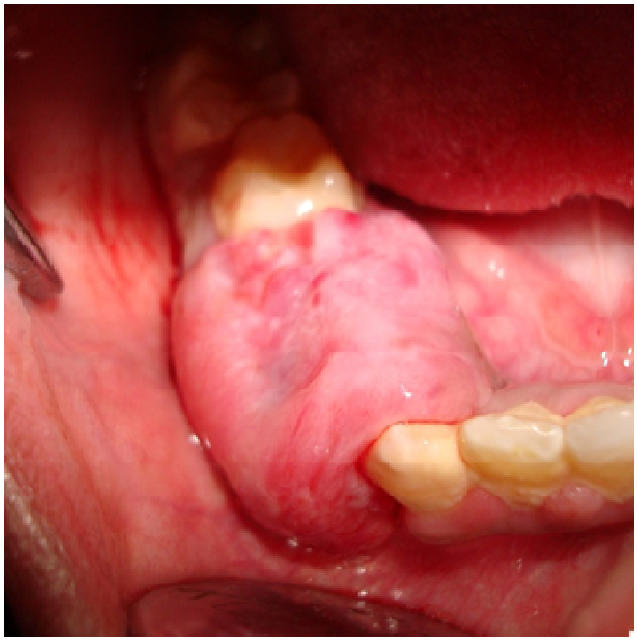

Figure 1. Intraoral swelling in relation to the right permanent lower first premolar and primary second molar

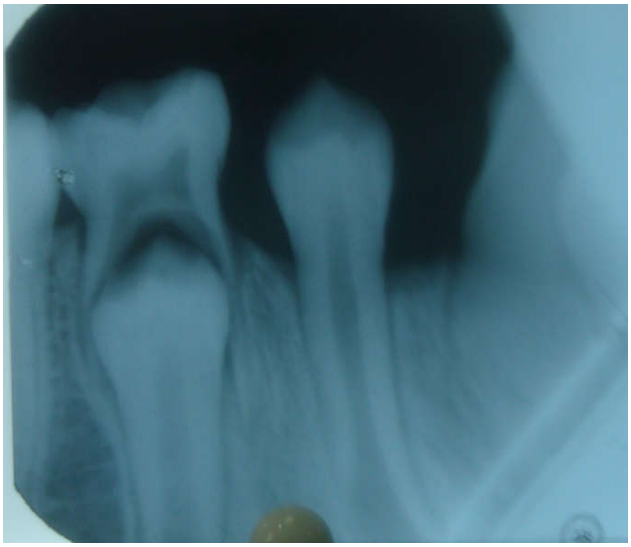

Figure 2. IOPA showing erupted first premolar and erupting second premolar

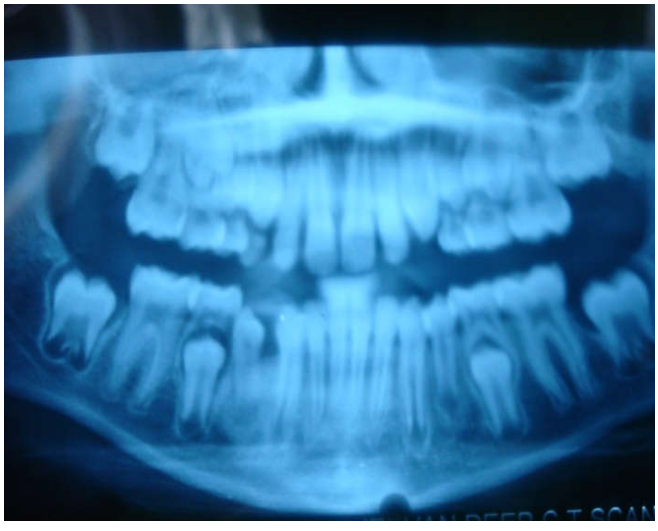

Figure 3. OPG showing presence teeth in the various stages of eruption

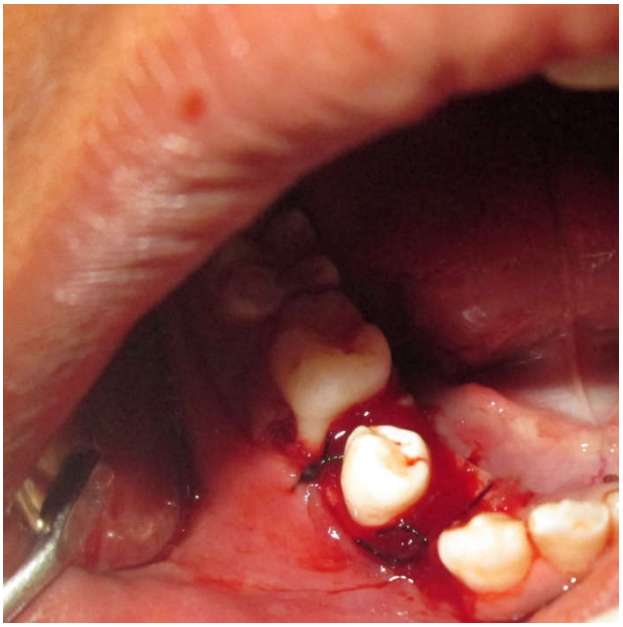

Figure 4: Intraoral view after excision

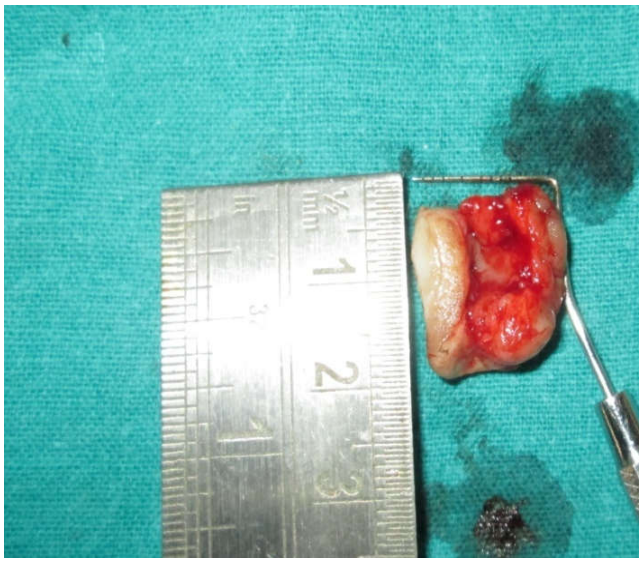

Figure 5. Excised tissue specimen measuring 2 cm × 1.5 cm

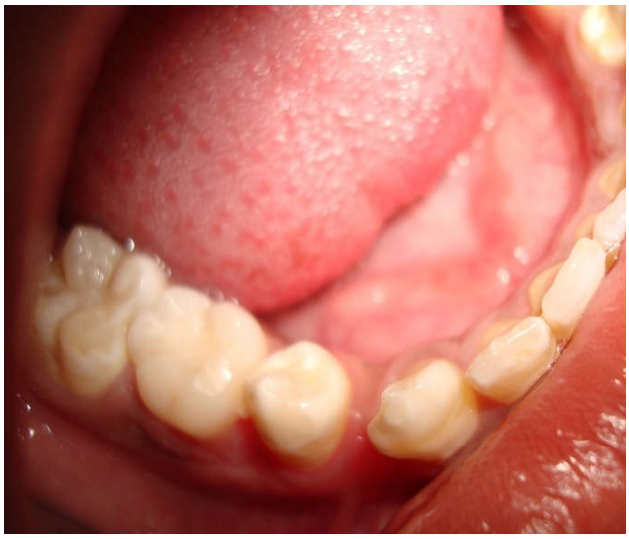

Figure 6. Five months postoperative photo showing uneventful healing

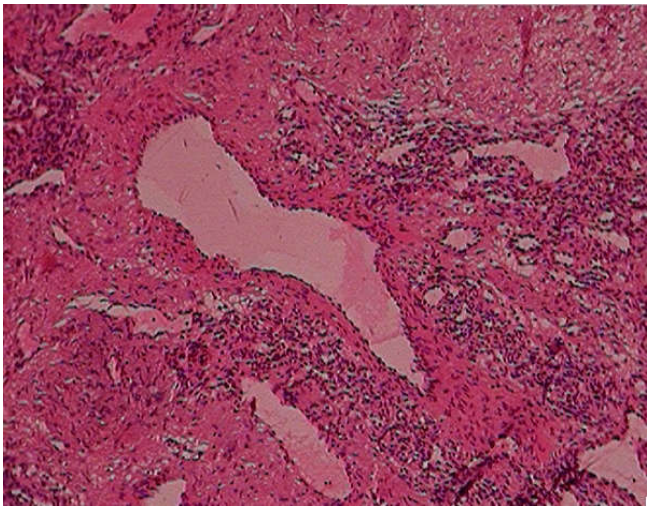

Figure 7. Hematoxylin and eosin staining section of lesion showing multinucleated giant cells

lesion under local anesthesia. Scaling and root planing was performed for elimination of local etiological factors. After 1 week of scaling and root planing, a reevaluation and complete surgical excision was performed (Figure 4). Lesion was separated from the adjacent tissue by blunt dissection and removed in one piece (Figure 5). The excised tissue was placed in 10% neutral buffered formalin and sent for the histopathologic examination. Sutures (3-0 silk) and periodontal dressing were placed. Patient was given post-operative instructions and was prescribed with analgesic (tablet ibuprofen-400 mg tds every 4-6 h as needed for pain) and antimicrobial rinse (0.2% chlorhexidine gluconate twice-a-day for 1 week). He was recalled, after 1 week for follow-up and suture removal. There was no evidence of recurrence till 5 months of follow-up (Figure 6). Histological examination showed highly cellular mass with abundant multinucleated giant cells distributed thoroughly. Numerous giant cells of various shapes and sizes, containing 8–15 nuclei, were seen with proliferating and dilated endothelial lined blood capillaries with extravasated red blood cells. Chronic inflammatory cells were present, and neutrophils were mainly encountered in the lesion. Additionally, there were mesenchymal cells that are ovoid and spindle-shaped. Diagnosis of PGCG was totally based on histological examination results (Figure 7).

DISCUSSION

PGCG presents as a firm, soft, pedunculated or sessile nodule with various sizes which range from small papules to enlarged masses; though they are generally less than 1.5 cm in diameter.[4] In present case size of the lesion was >1.5 cm. This lesion was uniquely large and required special care during surgical excision because of presence of underlying unerupted permanent tooth. The color can range from dark red to purple or blue commonly with ulcerated surface (Katsikeris *et al*., 1988). The preferential location of the lesion is premolar and molar zone, though Shafer (Rajendran, 2006). Giansanti and Waldron suggests that it generally occurs in the incisor and canine region (Giansanti *et al*., 1969). The lesions have been reported to be 2 times (approx 60%) more common in females than males and more frequent in the mandible than the maxilla. As the exact origin of the giant cells remains unclear, several hypotheses have been proposed to explain their proliferation: osteoblasts, phagocytes reacting to hemorrhage, endothelial cells, spindle-shaped mesenchymal cells, foreign body cells and osteoclasts (Itonaga, 2003). it is assumed that giant cells may be osteoclasts remaining from the physiological resorption of deciduous teeth. Other authors claim that giant cells may simply constitute a reactive component of the lesion and they may be derived from mononuclear cells originating from the bone marrow. As it has been demonstrated by immune-histochemistry, giant cells have membrane receptors for calcitonin, which characterizes osteoclast activity. Another possibility is that they are formed by mononuclear cells from the phagocytic system (Etoz, 2010). There are no pathognomic clinical features whereby these lesions can be differentiated from other forms of gingival enlargement. The differential diagnosis of PGCG includes pyogenic granuloma, peripheral ossifying fibroma, and peripheral cemento-ossifying fibroma, all of which present with similar clinical and radiographic findings. Another lesion, with very similar clinical and histological characteristics, is central giant cell granuloma, which are located within the jaw itself and exhibit a more aggressive behavior.

Only radiological evaluation can establish the distinction between central and peripheral forms of giant cell granulomas. As to treatment, the local surgical resection of the lesion is regarded as the most suitable approach. However, relapses may occur due to inadequate surgical technique, mainly when the surgeon does not effectively curette the periosteum subjacent to the lesion or small portions of the lesion remain within the tissues and proliferate afterwards, which promotes recurrence (Amaral, 2011). To avoid recurrence after treatment, in addition to complete simple excision with extensive clearing of the base of the lesion, the source of irritation needs to be eradicated (Katsikeris, 1988). This case shows that the proper management of a PGCG lesion requires excluding other pathologies prior to diagnosis, which is confirmed by the histopathological analysis of the excised lesion. Surgical excision with bone resection removed the lesion with no signs of recurrence.

Conclusion

Although the PGCG is a relatively common pathology in today's dental practice but presents a challenge to get diagnosed at early stages. So, early and definite diagnosis of PGCG on the basis of clinical, radiographic, and histopathological examination is essential to minimize risk to the adjacent structures.

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