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CASE REPORT

PERIPHERAL GIANT CELL GRANULOMA: A CASE REPORT

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ABSTRACT

Peripheral giant cell granuloma appears as a solitary purplish-red nodule, having vascular or hemorrhagic appearance, with or without surface ulceration. It may be a sessile or pedunculated lesion on the gingiva or the alveolar crest, common in relation to incisors and first molars. It occurs in response to local irritation from the connective tissue of the gingiva, periodontal ligament or periosteum of the alveolar ridge and may occur in periimplant tissues and edentulous arches. They are more common in females than males, more frequently seen in mandible than maxilla. Their growth potential is very high, and may penetrate interdentally to involve the adjacent cortical bone. An accurate diagnosis based on the histological observation is vital for its management. Considering its rapid and penetrating nature of its growth, surgical excision including its base is recommended to prevent the recurrence.

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INTRODUCTION

Localized gingival overgrowth, historically termed as epulides, are a common finding in clinical practice and may often present a diagnostic challenge due to similar clinical presentation. Various types of localized gingival overgrowth are identified, amongst which, the most prevalent one is peripheral fibroma (56 - 61%), followed by pyogenic granuloma (19 - 27%), peripheral ossifying fibroma (10 - 18%) and peripheral giant cell granuloma (1.5 - 7%) (Zhang *et al*., 2007; Kfir, 1980). These occur as reactive response of the gingiva to chronic, low-grade irritation caused by plaque and calculus, periodontal disease, poor dental restorations, ill-fitting dental appliances or extractions (Shafer, 1983). Clinically, peripheral giant cell granuloma appears as a solitary purplish-red nodule, having vascular or hemorrhagic appearance, with or without surface ulceration. It may be a sessile or pedunculated lesion on the gingiva or the alveolar crest, common in relation to incisors and first molars. It occurs in response to local irritation from the connective tissue of the gingiva, periodontal ligament or periosteum of the alveolar ridge. It may even be reported post periodontal surgeries, in periimplant tissues and edentulous arches (Shafer *et al*., 1983; Gottsegen , 1962; Hernandez , 2009-5). They occur commonly in females, at fourth to sixth decades of life, more frequently in mandible (Katsikeris, 1988). Their growth potential is very high, and may penetrate interdentally to involve the adjacent cortical bone resulting in mobility and separation of the adjacent teeth (Bodner, 1997). Radiographic features are nonspecific; however, widening of the periodontal ligament space, resorption of alveolar crest in the interdental region and resorption of associated teeth may be discerned on periapical radiographs (Chaparro-Avendaño, 2005). When edentulous areas are involved, the cortical bone exhibits a concave resorption beneath the lesion, typically known as "cuffing" resorption (Shafer, 1983). The diagnosis is confirmed histologically based on the presence of multinucleated giant cells embedded in a highly fibrillar connective tissue stroma containing large number of ovoid or spindle shaped young fibroblasts and spicules of bone or osteoid. Complete surgical excision following removal of the local irritants is the preferred treatment modality in order to reduce the recurrence (10-15%) (Shafer, 1983).

CASE REPORT: A 38 year old male reported with a localised painless purplish-red, sessile, bi-lobulated gingival overgrowth in relation to 14 and 15, extending from the interdental papilla on the buccal aspect to the palatal aspect,

the size being 11 mm x 15 mm (Figure 1 A, B). The growth started as a small swelling approximately 6 months back. It was resilient in consistency, with no blanching on digital pressure. Surface ulceration was seen on the palatal aspect and revealed blood on aspiration. 14 was found to be non-vital on electric pulp testing and Grade II mobile. The growth was asymptomatic, though tended to bleed during brushing or mastication, if accidentally bitten on. No history of trauma to that area was reported. Extra-oral examination revealed no facial asymmetry or lymphadenopathy. Patient was systemically healthy; however, he gave history of central giant cell granuloma in relation to 35-37 region and had undergone partial mandibulectomy 3 years ago. No sign of recurrence was observed.

Intra-oral periapical radiograph revealed widening of the PDL space in relation to 14 and 15 with erosion of interdental bone and external root resorption on the distal aspect of 14 (Figure 1 C). Routine blood investigations were found to be within normal range. Based on the history, clinical and radiographic findings, provisional diagnosis was made as Peripheral giant cell granuloma. Pyogenic granuloma and peripheral fibroma were considered for differential diagnosis due to the similarity of their clinical presentation. Phase I periodontal therapy followed by extraction of 14 and surgical excision of the growth was planned. Written consent was obtained from the patient. Accordingly, scaling and root planing was performed. 14 was extracted and growth was excised along with the surrounding normal tissues using Kirkland's gingivectomy

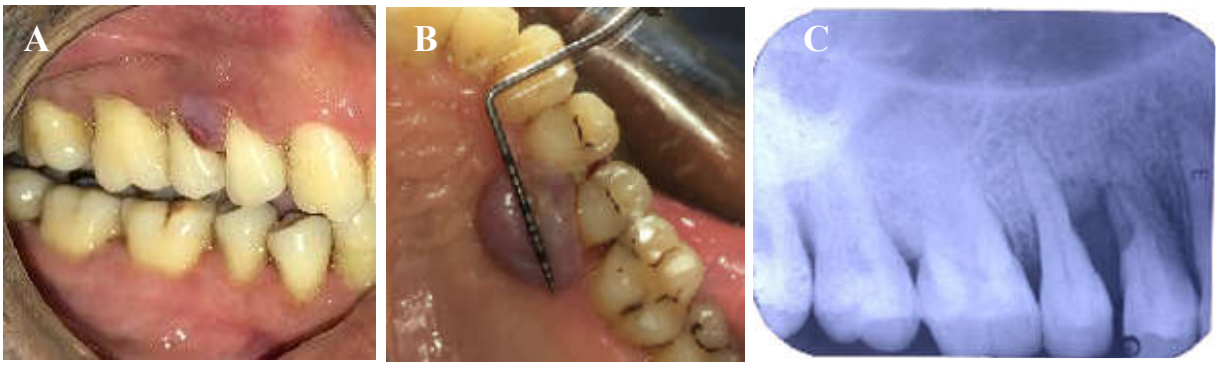

Figure 1 . (A) Intraoral view of gingival overgrowth seen in the interdental papilla between 14 and 15; (B) Palatal aspect of the overgrowth: 11 x 15 mm in size; (C) Intraoral periapical radiograph reveals horizontal bone loss in between 14 and 15 and external root resorption in 14.

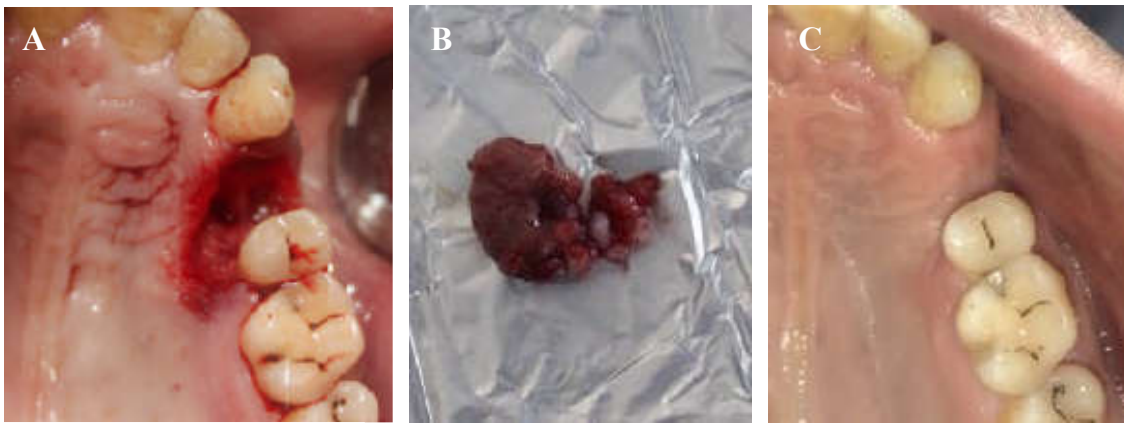

Figure 2. (A) Fresh wound immediately after extraction of 14 and excision of the growth; (B) Excised soft tissue; (C) Postsurgical palatal view showing no signs of recurrence (6 months)

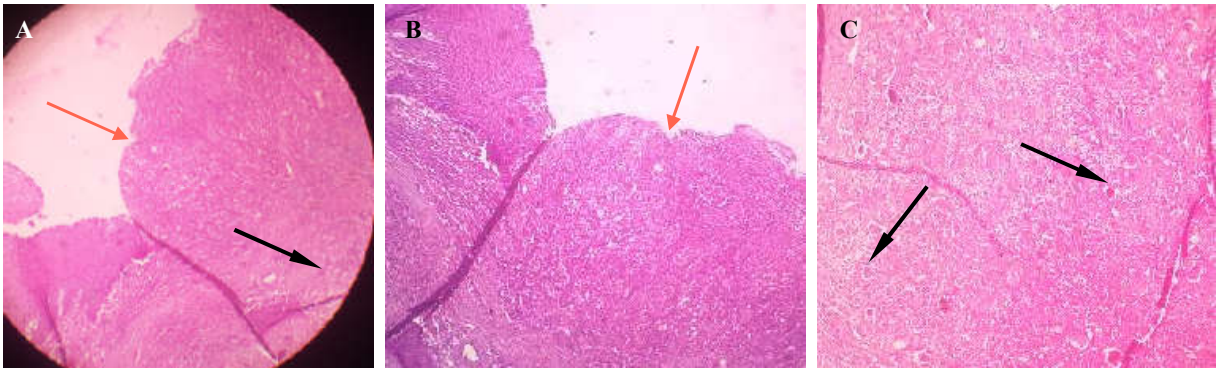

Figure 3 : (A) H & E staining of biopsied specimen: hyperplastic stratified squamous epithelium with ulcerated surface and multinucleated giant cells, denoted by red and black coloured arrows, respectively; (B) Hyperplastic stratified squamous epithelium with ulcerated surface denoted by red coloured arrow; (C) Connective tissue stroma showing scattered multinucleated giant cells of varying size denoted by black coloured arrows

knife, and interdental bone was curetted under local anaesthesia (Figure 2 A, B). Hemostatic gel-sponge was placed into the socket and flap margins were approximated with interrupted sutures. Periodontal dressing (Coe-pack) was placed and the patient was recalled after 7 days for suture removal. The excised tissue was sent for histopathological examination. The patient was followed up regularly upto 6 months post-surgically and no evidence of recurrence was observed (Figure 2 C). Histopathological examination of the tissue section revealed ulcerated, hyperplastic stratified squamous epithelium. Underlying connective tissue stroma was moderately collagenous, with numerous young fibroblasts and scattered giant cells of varying sizes along with diffuse chronic inflammatory cells (Figure 3 A, B, C).

DISCUSSION

Peripheral giant cell granuloma is a benign exophytic lesion, initially thought to be of reparative nature, and was named Giant cell reparative granuloma by Jaffe in 1953 (Jaffe, 1953). In contrast, Waldron and Shafer described that histologically this lesion showed features of benign giant cell tumors of bone with no reparative characteristics (Waldron, 1966). Bhaskar *et al.,* (1959) subdivided giant cell granuloma into central and peripheral. Central giant cell granuloma occurs within the bone, while peripheral giant cell granuloma originates in gingiva or edentulous alveolar processes (Bhaskar, 1959). The central variant is rare in nature, making up 7% of total benign lesions of the jaws. They may occur either as slowly growing asymptomatic swelling or an aggressive lesion that manifests with pain, local destruction of bone, root resorption, or displacement of teeth. Central giant cell granuloma has also been reported with conditions such as Cherubism, Noonan Syndrome, Jaffe-Campanacci Syndrome, Neurofibromatosis type 1, or Ramon Syndrome. Radiographically, it may appear as a large, unilocular or multilocular radiolucency with well-defined or ill-defined margins and varying degrees of expansion of the cortical plates (Chuong *et al*., 1986; Valentine, 2011). Peripheral giant cell granuloma is also known as peripheral giant cell tumor, osteoclastoma, reparative giant cell granuloma, giant cell epulis and giant cell hyperplasia (Chaparro-Avendaño, 2005).

Eversole and Rovin (1972) postulated that pyogenic granuloma, peripheral giant cell granuloma and peripheral ossifying fibroma share similar clinical and histologic features, though their response to irritation varies. Peripheral giant cell granuloma occurs as a result of more exaggerated response of periosteum to the irritants than that associated with the formation of pyogenic granuloma, which is a more common lesion. In addition, peripheral giant cell granuloma is found to be associated with xerostomia, which may be attributed to the reduced cleansing action of saliva that facilitates accumulation of local irritants (Bodner, 1997; Eversole, 1972). It has also been reported as an oral manifestation of hyperparathyroidism, X-linked hypophosphatemic rickets, though very rare (Flaitz, 2000; Parbatani *et al*., 1998). Marked female predilection suggests a possible hormonal influence on its causation. Surgical excision along with its base is the treatment of choice. Sometimes, extraction of the adjacent teeth may be necessary to ensure complete resection in some cases (Patil *et al*., 2014). Improper eradication of underlying source of irritation (Kfir, 1980) or lack of inclusion of the periosteum or periodontal ligament during excision may lead to recurrence (Regezi *et al*., 2009) which demands further re-excision (Shafer, 1983; Neville, 2009). The present case is still under observation even after six months of surgical removal of growth and shows no sign of recurrence.

CONCLUSION

An accurate diagnosis of gingival overgrowth through clinical, radiographic and histopathological examination is vital for its management. Due to the rapid growth pattern and tendency to resorb bone with resultant tooth movement, the treatment should include the surgical excision of the growth including its base in addition to elimination of etiologic factors. Post-surgical regular follow-up is essential to prevent recurrence of the growth.

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