Isolated lesions of gingiva: A case series and review

D. Radha Bharathi, S. Sangamithra, K. V. Arun, T. S. S. Kumar

Abstract

Isolated lesions of gingiva arise in succession to the hyperinflammatory reactions in response to the underlying local irritants. Despite their overlapping clinical and histological features, these lesions are distinctive regarding their biological behavior. Recurrence has been reported after surgical excision because of the incomplete removal of underlying local irritants. This article describes the clinical and histological features of four localized gingival lesions, adding a note on their molecular pathogenesis and surgical management.

Keywords: Gingival lesions, peripheral giant cell granuloma, peripheral odontogenic fibroma, peripheral ossifying fibroma, pyogenic granuloma

Introduction

Localized gingival overgrowths usually manifests as a slow growing nonneoplastic tumor which is a sequelae of nonspecific, exaggerated inflammatory reaction in response to the underlying local irritants. These overgrowths include pyogenic granuloma, peripheral ossifying fibroma (POF), peripheral giant cell granuloma (PGCG), and peripheral odontogenic fibroma, which characteristically differs from plaque-induced inflammatory gingival enlargement.

Despite the similarities in clinical appearance, the histopathology and treatment modality of these lesions are sufficiently diverse enough to classify them as distinctive entities. Commonly being asymptomatic in nature and reactive in character, these lesions pose esthetic and oral hygiene challenges to the patient and usually undergo surface ulcerations as a result of trauma from opposing teeth.

The conventional method of management of these lesions includes surgical excision by scalpel, laser, or electro surgery. However, these lesions are reported to have a high frequency of recurrence owing to the inability to completely remove the lesion by excision alone. It has been well documented that simple excision of these lesions results in remnants of inflamed tissue or local irritants causing recurrence which warrants secondary surgery.

This article aims at presenting four different cases of localized gingival overgrowth and a comprehensive review addressing specific concerns with each of these entities including therapeutic challenges.

Case Reports

Case 1: Peripheral odontogenic fibroma

A 53-year-old male patient reported with an asymptomatic swelling on the lingual surface of 33–37 measuring 2.5 cm × 1.5 cm, which was reddish pink and pedunculated with surface ulcerations [Figure 1]. The patient gave a history of surgical excision of the lesion 6 months back at the same site.

Under local anesthesia, an internal bevel incision was directed to the base of the tumor extending to the adjacent papilla, followed by sulcular incision on the buccal and lingual surfaces. Tumor was excised down to the periosteum and was preserved in formalin for H and E staining. A full-thickness flap was elevated, and thorough debridement of the site was performed. Flap was then approximated and sutured.

Department of Periodontics, Ragas Dental College, Chennai, Tamil Nadu, India

Correspondence: Dr. D. Radha Bharathi,
2E, Pushkar Apartments, ECR-OMR Link Road,
Sholinganallur, Chennai - 600 119, Tamil Nadu, India.
E-mail: drradhabharathi@gmail.com

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Antibiotics and analgesics were prescribed. Suture removal was done after a week.

H and E staining revealed interlacing myxomatous connective tissue fibers with highly cellular connective tissue strands or islands of odontogenic epithelium and mineralized cementoid-like material seen adjacent to the epithelium; hence, the diagnosis of peripheral odontogenic fibroma was made [Figure 1].

The WHO defined the odontogenic fibroma as "the benign odontogenic neoplasm of fibroblastic origin characterized by relatively mature collagenous fibrous tissue and varying amounts of odontogenic epithelium with the potential to occur in either a central or extraosseous location." The extraosseous counterpart is designated as peripheral odontogenic fibroma.[1] Pathogenesis of any odontogenic tumor is hypothesized as an aberrant growth of odontogenic epithelium with or without inductive proliferation of ectomesenchyme which may mimic the complex epithelial–mesenchymal interaction during odontogenesis.[2]

The presence of epithelial cell rests of malassez (ECRM) may be attributed to the proliferation of odontogenic epithelium seen in the lesion. ECRM has been shown to be immunoreactive for bone morphogenic protein (BMP2 and BMP4) which regulate the expression of Msx-1 and Msx-2 gene thereby regulating the epithelial-mesenchymal interactions suggesting the possibility of calcifications post tooth eruption.[3]

Case 2: Peripheral ossifying fibroma

A 46-year-old male presented with a swelling which was measuring 2 cm × 1 cm extending from buccal to the lingual surface in the 43–44 region. The swelling was clinically akin to peripheral odontogenic fibroma and was excised with open flap debridement leaving no residues of the underlying local factors. H and E staining showed dense connective stroma with cementoid-like areas and inflammatory cells [Figure 2]. The lesion was diagnosed as POF.

POF originates from the periosteum which undergoes chronic irritation resulting in the metaplasia of connective tissue, where the undifferentiated mesenchymal cells in the periodontal ligament differentiate to form cementoid-like material or dystrophic calcifications.

Evidence of origin of POF from periodontal ligament has been well documented with the positive expression of Runx2 and BMP2.[4]

Case 3: Pyogenic granuloma

A 38-year-old female patient presented with an asymptomatic lobulated mass in 11–12 buccal region measuring about 1.5 cm, which bled upon pressure and was compressible in nature. As the swelling was restricted to interdental papilla and did not show any radiographic bone loss, simple excision was performed. H and E staining revealed prominent endothelial proliferation and ulcerated epithelium with chronic inflammatory cells [Figure 3]. The lesion was diagnosed as pyogenic granuloma.

Termed by Crocker in the year 1903, pyogenic granuloma is the most common of all reactive lesions and has a high female sex predilection.[5] Chronic irritation precedes the inflammatory reaction and the increase in inflammatory cells *in situ*. It has been proposed that immunolocalization of basic fibroblast growth factor (FGF) which is an angiogenic protein released by macrophages increases by multifold, leading to granuloma formation.[6]

In female patients, there is an increased expression of vascular endothelial growth factor and FGF accompanied by female sex hormones resulting in the formation of pyogenic granuloma which undergoes remission postpartum due to increase in apoptotic factors such as tumor necrosis factor α.[7]

Case 4: Peripheral giant cell granuloma

A 35-year-old female patient presented with a reddish pedunculated swelling measuring about 1.5 cm in the buccal aspect of 23–24 region with ceramic crown placement in relation to 23 [Figure 4]. The swelling was ulcerated and bled spontaneously. Flap was raised, and the tumor was excised. Bone loss extending up to the middle-third of the root corresponding to the cupping resorption of PGCG was observed, and osteoplasty was done using bone files. Flap was approximated and sutured after thorough debridement of the region.

H and E staining revealed numerous multinucleated giant cells lying in vascular stroma with dense inflammatory infiltrate. The periphery of the section showed deposition of hemosiderin; hence, the diagnosis of PGCG was made.

The pathogenesis of PGCG could be attributed to the increased secretion of monocyte chemoattractant protein-1 and macrophage colony stimulating factor by the stromal cells which induces the migration of monocytes that fuses to form multinucleated giant cells.[8]

Discussion

The prevalence of reactive lesions among other inflammatory lesions in the gingiva has been well documented. Of these lesions, the incidence of pyogenic granuloma is reported to be higher (42%) followed by POF (18%) and PGCG (10%). Majority of the swelling was reported to be present in the maxillary or mandibular anterior region, and there was a slightly

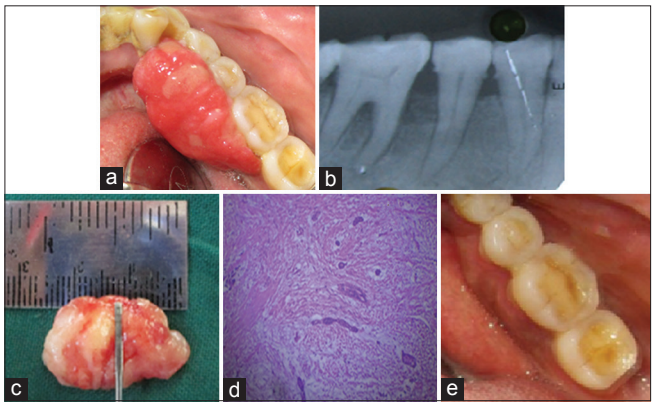

Figure 1: (a) Clinical view of peripheral odontogenic fibroma; (b) intra oral periapical radiograph reveals crestal bone loss in 35–36 region; (c) excised tissue measuring 2.5 cm × 1.5 cm; (d) H and E showing odontogenic epithelium and dystrophic calcifications; (e) clinical post operative view after 1 year

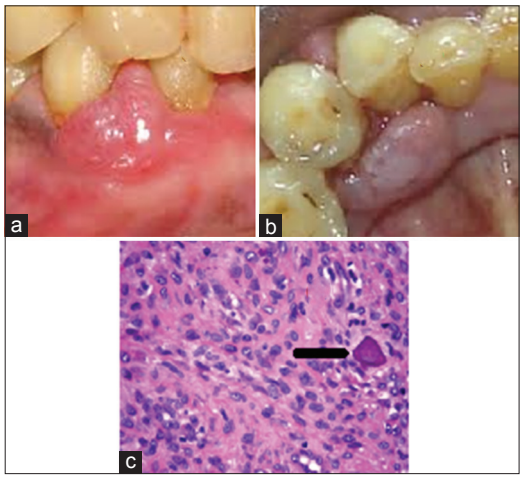

Figure 2: (a) Buccal view of peripheral ossifying fibroma; (b) lingual view of peripheral ossifying fibroma; (c) H and E showing cementoid such as calcifications

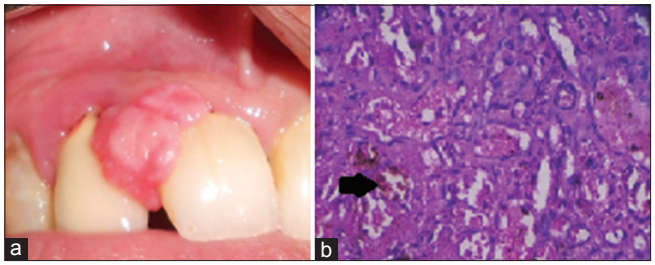

Figure 3: (a) Clinical view of pyogenic granuloma; (b) H and E showing dense inflammatory cells and endothelial proliferation

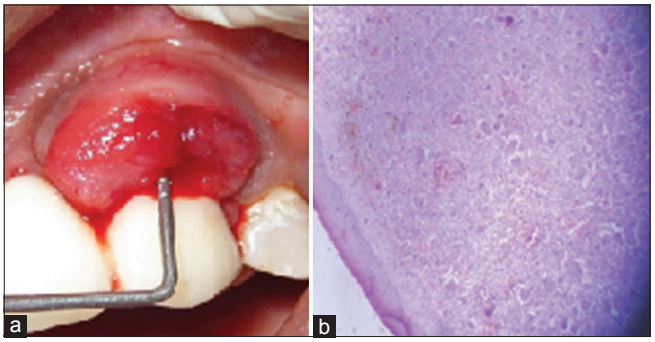

Figure 4: (a) Clinical view of peripheral giant cell granuloma; (b) H and E showing multinucleated giant cells with dense inflammatory infiltrate

high female sex predilection with the presentation in the second and third decade of life.[5] The underlying molecular pathogenesis discussed along with case description, clinical, and histological features, would aid in better understanding of these lesions and establishing an appropriate treatment protocol.

Conclusion

The recurrent nature of these lesions validates their inherent neoplastic potential; therefore, in conjunction to the interpretation of the clinical and histological features, understanding their biological behavior and proliferative potential assists in treating them effectively thereby preventing recurrence. Hence, raising a flap, wherever necessary, not only aids in the complete elimination of the remnants but also establishes favorable gingival contour and mucogingival complex that enables better oral hygiene maintenance.

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Conflicts of interest

There are no conflicts of interest.

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