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Inflammatory Fibroepithelial Hyperplasia: A Case Report

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Abstract

Inflammatory lesions in gingiva resulting from chronic irritation, commonly caused by plaque accumulation, calculus, or ill-fitting dental prostheses are often observed among population who fail to attend to it immediately resulting in tooth loss. This case report presents a 43-year-old female with localized gingival overgrowth in the left maxillary anterior region. The lesion was firm, non-tender with periodontal attachment loss. Scaling and root planing, followed later by surgical excision of the tissue overgrowth was performed. Histopathological analysis confirmed fibroepithelial hyperplasia with features of early peripheral ossifying fibroma. Postoperative healing was uneventful, with no signs of recurrence at four weeks. Patient was placed on maintenance protocol. Proper diagnosis and histopathological evaluation are essential to differentiate reactive hyperplasia from neoplastic lesions. Management involves eliminating etiologic factors and surgical removal to prevent recurrence. Regular follow-up and oral hygiene maintenance play a crucial role in long-term success. This case highlights the importance of early intervention and comprehensive periodontal care in managing reactive gingival overgrowths.

Keywords: Fibroepithelial hyperplasia, Gingival inflammation, Gingival overgrowth

INTRODUCTION

The oral cavity is subject to a variety of stimuli that can induce a broad spectrum of pathological responses, including developmental, reactive, inflammatory, and neoplastic changes [1]. Among these, reactive hyperplastic lesions such as inflammatory fibrous hyperplasia, pyogenic granuloma, peripheral giant cell granuloma, and peripheral ossifying fibroma are common non-neoplastic conditions characterized by abnormal tissue overgrowth resulting from persistent irritation [1]. These lesions are typically classified histologically as hypertrophy (increased cell size) or hyperplasia (increased cell number), often triggered by chronic irritants such as plaque, calculus, poorly fitting prostheses, or over-contoured restorations [2].

Inflammatory hyperplasia, a reactive process involving the proliferation of inflamed fibrous and granulation tissue, typically varies in size depending on the intensity and type of irritation as well as the degree of inflammation. These lesions are often associated with plaque mediated inflammatory conditions such as gingivitis or periodontitis. Persistent biofilm accumulation in tooth surface with limited self-cleansing and other predisposing iatrogenic factors can act as a nidus for gingival irritation, perpetuating the hyperplastic response [3].

Clinically, these inflammatory lesions may present as localized or generalized gingival enlargement, which can mimic other pathologies, necessitating the importance of thorough clinical examination of periodontium and histopathological evaluation of the affected tissue for an accurate diagnosis. Gingival overgrowth, often referred to in earlier terms such as epulis or fibrous hyperplasia, can have significant functional and psychological impacts, including difficulty with mastication and aesthetic concerns [4]. Management of this condition usually involves eliminating the underlying irritants and, when necessary, surgical excision. Histopathological confirmation is essential to guide appropriate surgical therapy and minimize recurrence risks.

CASE REPORT

CLINICAL PRESENTATION

A 43-year-old female presented to the Department of Periodontology with a six-month history of localised swelling in the left upper anterior region, accompanied by discomfort during mastication. The patient had experienced a similar episode one year prior, which was treated at a private dental clinic with scaling and root planing followed by antibiotics, leading to the alleviation of symptoms. However, the swelling recurred six months later despite treatment. Patient had no reported history of underlying medical conditions or hormonal abnormalities.

On clinical examination, a well-defined, pale pink swelling was noted in the left maxillary anterior region involving tooth number 22 and 23 regions. The lesion extended across the marginal and attached gingiva of 22,

as well as the interdental papilla between 22 and 23 on the facial surface, measuring approximately 8 × 10 × 6 mm [Figure 1]. On palpation, the lesion was firm, leathery, and non-tender. Mild inflammation was present, with no enlargement on the palatal surface. The associated teeth exhibited moderate calculus accumulation, bleeding on gentle probing, a probing depth of 9 mm, and a clinical attachment loss of 6 mm. Exudate was absent. Spacing was observed between 22 and 23, indicating pathological migration.

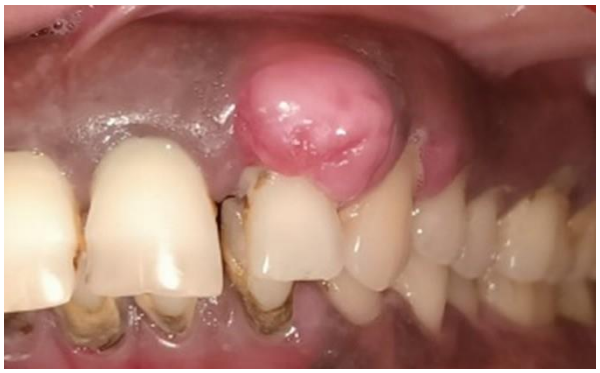

Figure 1: Pre-operative image

Radiographic analysis revealed horizontal bone loss extending beyond the middle third of the roots of 21 and 22, angular bone loss extending to the middle third in relation to 22 and 23 [Figure 2]. A provisional diagnosis of pyogenic granuloma was made. Blood investigations showed no abnormalities. Periodontal management planned with Phase 1 therapy, comprising scaling and root planing, followed by Phase II surgical excision of the lesion.

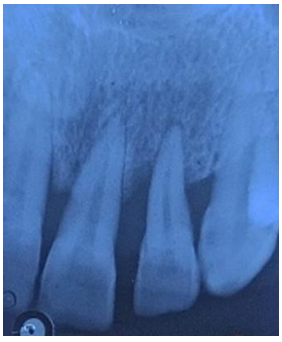

Figure 2: Pre-operative Radiograph

MANAGEMENT

NON-SURGICAL PHASE

The lesion was thoroughly examined. Phase 1 therapy was carried out, including thorough scaling and root planing in two sittings. The patient was advised to use 10ml of 0.2% chlorhexidine and instructed to swish for 30 seconds twice daily. This stage of therapy was done to eliminate local factors and reduce inflammation.

SURGICAL PROCEDURE

After administering local anesthesia to the surgical site 22 and 23, an external bevel incision was made at the base of the lesion, using a No. 15 scalpel blade [Figure 3]. Care was taken to excise the tissue in toto, and it was preserved in 10% formalin for histopathological examination. Following this, a sulcular incision was made and flap reflected. Any residual calculus was removed, thorough planing of root surface and soft tissue debridement was performed [Figure 4]. Tissue tags and granulation tissue were excised. The surgical site was carefully examined to ensure complete removal of the lesion, to minimize the risk of recurrence. The site was irrigated copiously with saline. The wound was closed using 3-0 silk sutures and primary hemostasis was achieved. Periodontal pack (Coe-Pak™) was placed.

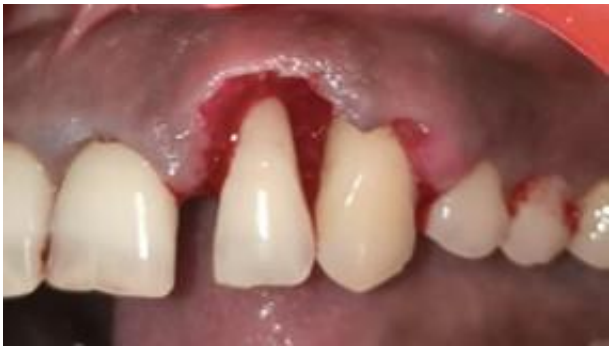

Figure 3: Post- surgical image

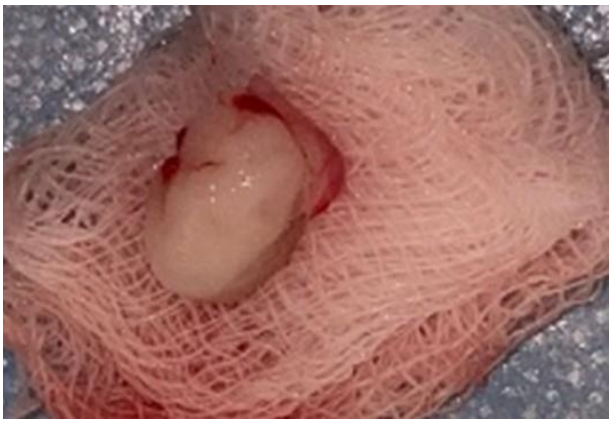

Figure 4: Excised lesion

Post-operative instructions were given to the patient, advising them to avoid spicy food, apply cold packs extra-orally in case of swelling, and begin warm saline rinses 24 hours post-surgery, performed 3–4 times daily. The patient was prescribed amoxicillin 500 mg and Imol (analgesic), both to be taken three times daily for three days, along with the use of 0.2% chlorhexidine mouthwash twice daily for one week.

MAINTENANCE PHASE

The patient was recalled for follow-up at regular interval. During the first week follow-up, healing appeared satisfactory, and sutures were removed [Figure 5]. Patient was motivated to maintain proper oral hygiene. At the two-week follow-up, there were no signs

of inflammation or residual swelling. By four-weeks, no signs of recurrence were observed with a reduction in probing depth [Figure 6]. The patient was placed in maintenance protocol and advised bi-monthly review. But the patient did not turn up for her review and didn't respond to our calls. After five-months from the baseline, the patient turned up for review. Though no recurrence of the soft tissue lesion was observed, the tooth exhibited an increase in mobility with radiographic evidence of angular bone loss extending upto apical third of root in relation to 21and 22 and horizontal bone loss upto middle third in relation to 22 and 23 region [Figure 7 and Figure 8].

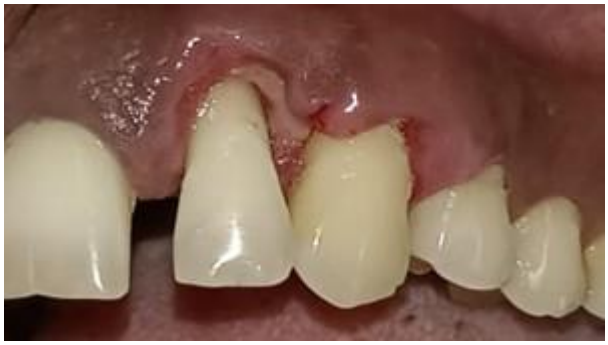

Figure 5: One-week post-operative image

Figure 6: Four-week post-operative view

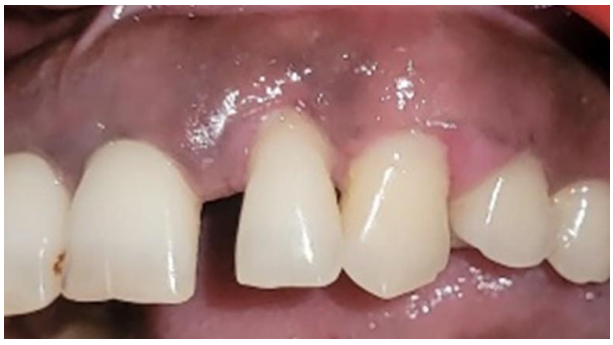

Figure 7: Fifth-week post-operative view

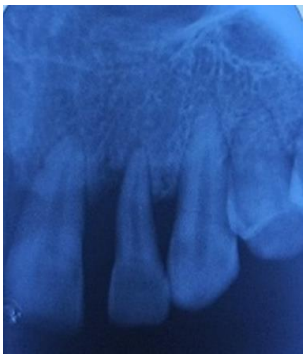

Figure 8: Post-operative Radiograph

HISTOPATHOLOGICAL PRESENTATION

Histopathological analysis of the H&E-stained section revealed parakeratinized stratified squamous surface epithelium associated with inflamed cellular fibrovascular connective tissue containing diffuse chronic inflammatory cell infiltrate [Figure 9]. These findings were suggestive of fibroepithelial hyperplasia with ossifications, with clinical and histological features indicative of an early peripheral ossifying fibroma [4][5]. Peripheral ossifying fibroma lesions have hyperplastic stratified squamous epithelium with slender finger-like rete ridges extending into the fibrocellular connective tissue stroma. There is also infiltration of chronic inflammatory cells similar to one observed in our specimen.

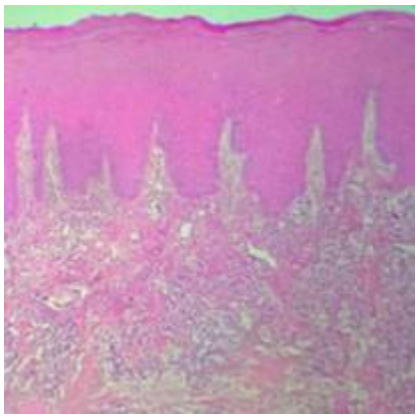

Figure 9: Histopathology

DISCUSSION

Gingival overgrowths are commonly referred to as "epulis," a term historically used to describe reactive hyperplastic lesions [6]. Most intraoral lesions labeled as fibromas are not true neoplasms but represent fibrous overgrowths resulting from chronic irritation. The term inflammatory hyperplasia describes a broad range of nodular growths in the oral mucosa that histologically represent inflamed fibrous and granulation tissue. The size of these reactive hyperplastic masses depends on the intensity and type of irritant stimulus and the degree of inflammation. In this patient, the gingiva appeared firm and fibrotic with no spontaneous bleeding. Factors such as plaque accumulation, calculus, and trauma from

occlusion may have triggered tissue proliferation, while the chronic nature of these irritants likely contributed to the fibrotic transformation of the gingiva.

Fibrous hyperplasia is more frequently observed in older age groups compared to pyogenic granuloma and peripheral fibroma with calcification [7][8]. Cooke reported that fibrous hyperplasia cases peaked in the fourth decade, while Brown and Darlington observed the highest number in the third decade [9][10]. Daley et al. proposed that pyogenic granuloma undergoes a gradual transition where its vascular component is replaced by fibrous tissue over time, eventually being diagnosed as fibrous hyperplasia or fibroma [6]. Furthermore, Al-Rawi found that fibrous hyperplasia of the gingiva shares the same female gender predominance, age range, and site distribution as gingival pyogenic granuloma [11]. These observations suggest that fibrous hyperplasia represents the fibrous maturation of pyogenic granuloma, especially in long-standing lesions.

Management involves complete excision of the lesion along with the removal of the gingival connective tissue base. Alternative treatment modalities include the use of electrocautery, Nd: YAG laser, flash lamp pulsed dye laser, cryosurgery, intralesional injection of ethanol or corticosteroids or sodium tetradecyl sulfate sclerotherapy [12][13][14]. Microscopically examining excised tissue is critical, as other benign or malignant tumors may present with a similar clinical appearance.[15]. To minimize recurrence, eliminating contributing factors such as plaque, calculus, defective restorations, open contacts, or trauma from parafunctional habits is essential. While recurrences are rare, it was observed in patients as reported in few studies.[9][16].

With regard to this case report, the patient was exhibiting poor compliance from the beginning of the management and needed persuasion to get the lesion excised. The patient did not report to the recall visits as required and showed resistance to adhere to maintenance protocols. At the fifth month review the patient was again not willing for any further periodontal management and hence was explained the poor prognostic status of 22 and 23 and placed under a recall program of every 1-2 months in accordance with Merins classification to monitor periodontal health and prevent potential recurrence of the lesion [17].

It is important to differentiate fibroepithelial hyperplasia from focal epithelial hyperplasia, a viral condition caused by human papillomavirus (HPV). Unlike fibroepithelial hyperplasia, focal epithelial hyperplasia affects only the epithelium and spares the connective tissue [18]. Although reactive hyperplastic lesions are considered self-limiting, they may occasionally transform into neoplastic growths under prolonged irritation, emphasizing the importance of timely intervention [19]. Additionally, fibrous gingival growths may occasionally result from systemic conditions or drug-related factors, emphasizing the need for a detailed medical and dental history in these cases. Such distinctions are critical for accurate diagnosis and appropriate treatment planning.

CONCLUSION

Inflammatory fibrous hyperplasia is a benign soft tissue response to local irritants, such as calculus, sharp teeth, broken fillings, and excessive plaque. Clinical diagnosis can be challenging; thus, histopathological confirmation is essential for differentiation from other forms of gingival overgrowths. Complete surgical excision down to the periosteum is the preferred treatment to prevent recurrence. Close post-operative follow-up and patient education on maintaining oral hygiene are crucial to achieving long-term success.

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