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CASE REPORT

FIBROEPITHEIAL HYPERPLASIA ASSOCIATED WITH SEVERE BONE LOSS TREATED WITH PRF & BONE GRAFT – AN EFFECTIVE BIOLOGIC BANDAGE

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ABSTRACT

Tissue enlargement of the oral cavity often presents a diagnostic challenge because a diverse group of pathologic processes can produce such lesions. Benign hyperactive lesion of gingiva is a reactive tumor like growth which arises due to various stimuli such as low grade long duration local irritation and minor trauma.Fibro-epithelial hyperplasia is ahistological variant of fibroma and a proliferativefibrous lesion of the gingival tissue that causesesthetic and functional problems. These lesions are a result of trauma/chronicirritation, or arise from cells of periodontium,periodontal ligament, or periosteum. This article focuses on a rare case of gingival overgrowth associated with severe bone loss which was positively dealt with appropriate surgical periodontal therapy, bone graft & platelet rich fibrin (PRF).

INTRODUCTION

A female patient aged 50 years, reported to the Department of Period ontology with a chief complaint of pain &swelling in the upper front tooth region since 6 months which lead to spacing between the upper front teeth. The swelling started as a small painless growth 6 months back, gradually increasing to the present size and was hindering mastication. On clinical examination, a localised gingival swelling 1X 3 cm extending labiopalatallyin relation to the upper anterior region with moderate supra and sub gingival calculus [Figure. 1].

Figure 1- Localised gingival swelling(1 X 3 cm) extending labio-palatallyin relation to the upper anterior region

The lesion was sessile, ovoid and pinkish. On palpation the growth was non-tender, firm, non reducible and non compressible with no bleeding on probing. Medical and dental histories were non contributory and the routine blood tests were within normal limits. Intra-oral periapical radiographs of the area of interest were obtained, which revealed severe alveolar bone loss between 11 and 21 and widening of periodontal ligament space [Figure. 2].

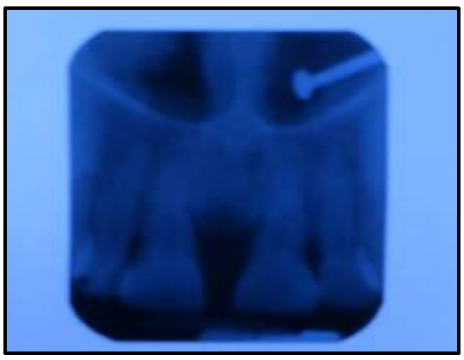

Figure 2- IOPA revealed severe alveolar bone loss between 11 and 21

Based upon clinical and radiographic findings, a provisional diagnosis of Pyogenic granuloma was made. Phase I therapy

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was initiated which included supra gingival scaling and rinsing with 10ml of chlorhexidine mouthwash (0.2%, Hexitidine®) twice daily for 1 week. Excisional biopsy was planned 1 week after the phase I therapy. The patient was explained regarding the procedure and a written consent obtained. The overgrowth was excised enmass with an external bevel incision and excised tissue was sent for histopathological examination [Figure. 3].

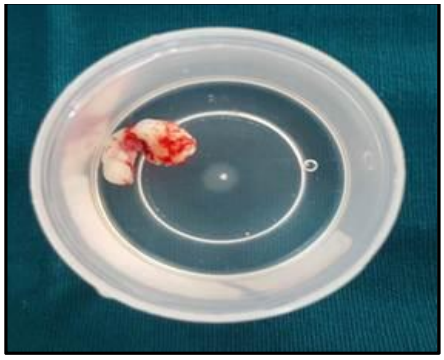

Figure 3 Excision of the overgrowth

The surgical site was then examined for any residual embedded calculus. On examination, labial to palatal cortical bone perforation was seen [Figure.4].

Figure 4 Labial to palatal cortical bone perforation

Hence crevicularincisons were given on either side & flap was raised to make space for the bone graft at the perforation site. Also as the overgrowth was excised enmass exposure of the underlying alveolar bone was inevitable. Hence to prevent further bone loss alloplastic bone material (osseograft) was placed on the labial aspect of the defect [Figure. 5].

Figure 5 Alloplastic bone material (osseograft) placed at the defect area on the labial aspect

PRF was autologously prepared by drawing 10 ml of patient's blood and centrifuging it at 2500 rpm for 10 minutes (Choukroun *et al*.).

The outcome was the fibrin clot containing platelets in the middle of the tube between the red blood cell layer at the bottom and the acellular plasma at the top. The clot was removed from the tube and squeezed between gauze squares to obtain a membrane. The membrane which was thus obtained was placed where the bone exposure was encountered [Figure. 6].

Figure 6 Platelet rich fibrin (PRF) placed at the defect area on the palatal aspect

Figure 7- Post – operative evaluationafter 6 months

A partial thickness flap beyond the muco-gingival junction was raised so as to cover the defect & allow close approximation of both the labial& palatal tissues. 3-0 BBS simple interrupted sutures were given & over which a periodontal dressing was placed (COEPAK ®). Histopathologic report showed hyperplastic stratified squamous epithelium of parakeratinized type and bundles of dense collagen fibers, and few blood capillaries as well as infiltration of chronic inflammatory cells like lymphocytes and plasma cells within the connective tissue stroma. Focal myxoid changes & dystrophic calcifications were also seen in the connective tissue suggestive of inflammatory fibro epithelial hyperplasia with calcifications.

DISCUSSION

Fibrous growths of the oral soft tissues are fairly common and include a diverse group of reactive and neoplastic conditions (Mohammed NA *et al*., 2010).The term "Fibro epithelial hyperplasia"was suggested by Deley *et al*. 1990 which implies a reactive localized tissue response and is therefore a better term to "fibroma" which wrongly implies, a benign neoplastic proliferative fibrous lesion (Tyldesley WR *et al*., 1974). Although the term Fibro epithelial hyperplasia, precisely describes the clinical appearance and pathogenesis of this entity, it is not commonly used. It is most often, a slow

growing welldefinedlesion that occurs at any age but most commonly occurs during the third, fourth and fifth decades of life. Females are twice as frequently affected as males (Mathur LK *et al*., 2010).

One of the important factors that may aggravatethe response of gingival tissue is poor oralhygiene which can result in enlargement ofgingival tissue (Carranza FA *et al*., 2006). In the present case, gingival tissue irritation due to presence of subgingivalplaque and calculus may have led to the development and progression of the overgrowth. The lesion usually presents as painless, sessile, round or ovoid, broad-based swelling, lighter in colour than surrounding tissue due to a reduced vascularity. Their size varies from few millimeters to several centimeters in size and rarely, may cause significant bone loss. In present cases, size of lesions attained a larger size >2.5 cm and caused significant bone loss in the affected area.

As the hyperplasia, in our case, was excised enmass, there was no soft tissue left to achieve primary closure, leaving behind an open wound area with bone exposure. Also there was labial to palatal bone perforation. If left to heal by secondary intention there would be bone necrosis with severe loss of alveolar bone, delaying the healing. Hence, to prevent further alveolar bone loss & accelerate healing, we decided to use bone graft & autologous PRF in the form of biologic bandage to cover the exposed bone.

It was observed that at time of 1week postoperative evaluation, the excision site had completely healed with formation of inter dental soft tissue. In our case the enmassexcision and the adjunct use of bone graft &PRF has resulted in improved gingival architecture which enhancedboth the esthetics and functional outcomes. [Figure. 7].

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