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CASE REPORT

Different Surgical Approaches for the Treatment of Gingival Overgrowth of Inflammatory Origin not Amenable to Non-surgical Periodontal Therapy – A Case Series

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ABSTRACT :
Inflammatory gingival overgrowth or enlargement is the most common type of gingival overgrowth seen mostly associated with the presence of local factors that predispose to it. The treatment of such enlargement is necessary as it acts as a niche for plaque accumulation and colonization by various periodontopathogenic bacteria. These overgrowths may cause bleeding on slight manipulation, interfere with occlusion or may get complicated with superinfections. The following two cases describe different surgical approaches for the treatment of inflammatory gingival enlargement. The choice of approach depends on the nature of enlargement, underlying bony topography and patient's esthetic and functional needs.
Key words: Gingival overgrowth, Gingival hyperplasia, Gingivectomy, Inflammation, Dental plaque.

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INTRODUCTION :

Gingival enlargement is defined as an overgrowth or increase in size of the gingiva.[1] Gingival enlargements are broadly classified as follows:[2]

I. Inflammatory enlargement – Acute, Chronic
II. Drug-induced enlargement – Anticonvulsants, Immunosuppressants, Calcium channel blockers
III. Enlargements associated with systemic diseases or conditions – Pregnancy, Puberty, Vitamin C deficiency, Leukemia, Granulomatous disease.
IV. Neoplastic enlargement (gingival tumors) – Benign, Malignant
V. False enlargement.

The most common type is reactive hyperplasia of gingival which is a result of plaque-induced inflammatory gingival disease.[3] The etiology of chronic inflammatory gingival enlargement includes factors that favor plaque accumulation and retention such as poor oral hygiene, anatomic deformities, improper restorations and prosthesis and orthodontic treatment.[4] It is most commonly associated with interdental papilla and marginal gingiva and can be localized as well as generalized to the entire dentition.

The treatment of choice for chronic inflammatory enlargements that do not undergo shrinkage after scaling and root planing that can hinder the patient's ability to suitably perform oral hygiene and cause esthetic and functional problems can be surgical removal through gingivectomy or the flap operation.[2]

CASE REPORT:

Case 1

A 21 year old male patient reported to the Department of Periodontology, with a chief complaint of uneven gingival margins in mandibular anterior region of the mouth. Patient had undergone fixed orthodontic treatment for 2 years and had completed the treatment 1 month back. Initial intraoral clinical examination revealed pseudopockets with asymmetry of gingival margin showing gingival inflammation with plaque and subgingival calculus deposits on teeth 33 to 43. Bleeding on probing was present on all teeth from 33 to 43. Radiographic examination revealed normal interdental bone levels with no signs of bone loss.

Phase I periodontal therapy comprised of supragingival and subgingival scaling and polishing. Patient was prescribed 0.2% chlorhexidine mouthwash twice daily for 15 days. On re-evaluation after phase I therapy, the gingival enlargement did not show considerable reduction in size, but the tissues appeared to be firm in consistency (Fig.1).

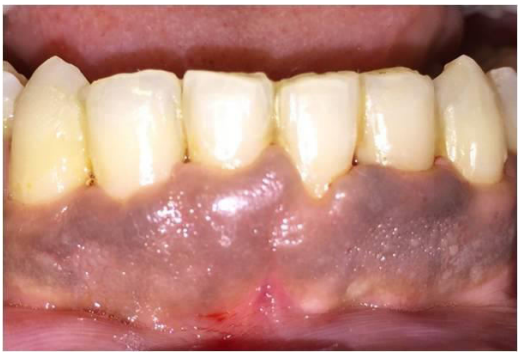

Fig 1: Uneven gingival margins and gingival inflammation w.r.t 33 to 43.

After administration of local anesthesia, bone sounding was performed using UNC 15 periodontal probe. Using pocket marker, bleeding points were marked (Fig.2) and external bevel gingivectomy was performed using scalpel and no. 15 blade taking utmost care to preserve biologic width of 3mm from the gingival margin to the bone crest (Fig.3,4).

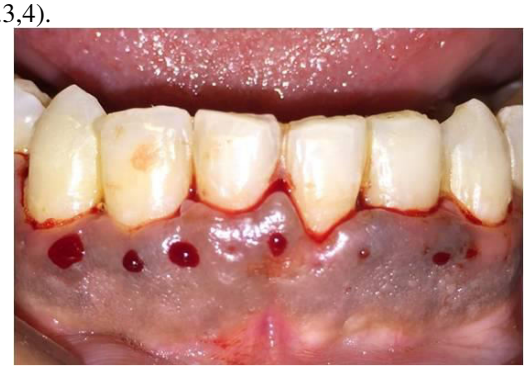

Fig 2: Bleeding points marked using Crane Kaplan pocket marker.

Excised gingival tissue was sent for histopathological examination. The microscopic picture revealed parakeratinized stratified squamous hyperplastic gingival epithelial tissue suggestive of inflammatory hyperplasia (Fig.5). Antibiotics and analgesics were prescribed to the patient. Patient was recalled after 7 days for post-operative evaluation. Stable and healthy gingival margins were seen at 1 month follow-up (Fig.6).

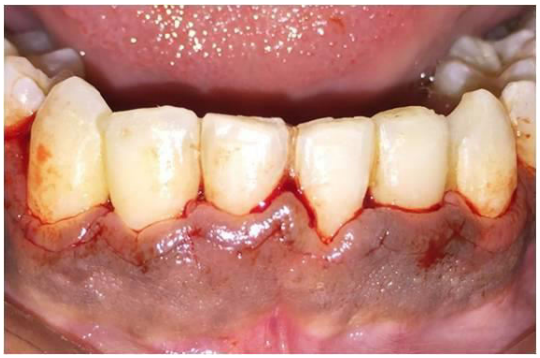

Fig 3: External bevel incison placed using no. 15 blade.

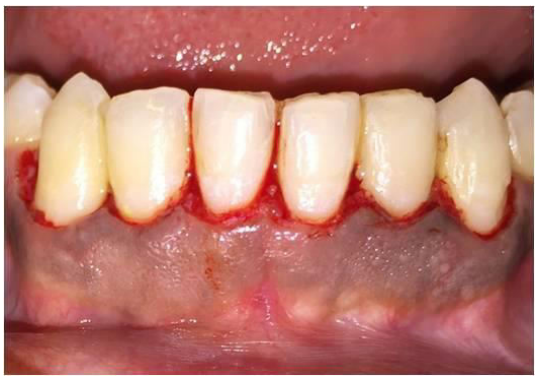

Fig 4: Immediate post-operative intraoral photograph.

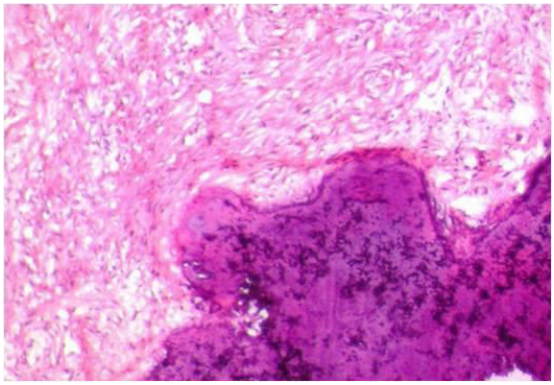

Fig 5: Histopathological picture showing parakeratinized stratified squamous hyperplasticgingival epithelial tissue suggestive of inflammatory hyperplasia.

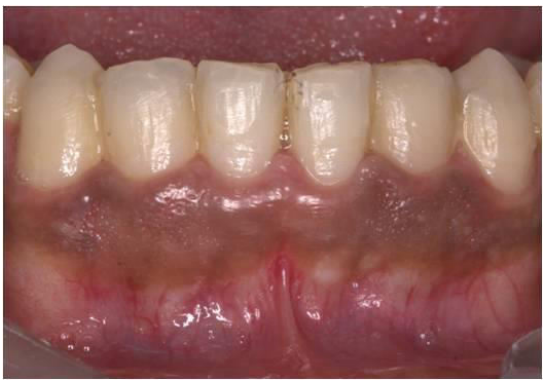

Fig 6: 6 months post-operative intraoral photograph of case 1.

CASE 2

A 23 year old male patient reported to the Department of Periodontology, with a chief complaint of bleeding gums in mandibular anterior region of mouth. Intraoral clinical examination revealed gingival inflammation with papillary gingival enlargement with 33 to 43 (Fig.7). Bleeding on probing was present on all teeth from 33 to 43. Radiographic findings did not reveal any abnormalities with interdental bone levels.

Following phase I periodontal therapy, Normal probing depths were present on all the teeth and the gingival tissue was firm and resilient (Fig.8). Horizontal bone sounding revealed slight buttressing of the buccal cortical bone. Using pocket marker, bleeding points were marked and internal bevel gingivectomy was performed using scalpel and no. 15 blade. Sulcular incisions were placed to reflect full thickness mucoperiosteal flap to correct underlying osseous architecture (Fig.9). 4-0 black silk sutures were placed using simple interrupted suturing technique and periodontal dressing was given (Fig.10). Antibiotics and analgesics were prescribed to the patient. Uneventful healing was observed at 7 days follow-up. Stable and healthy gingival margins were seen at 3 months follow-up (Fig.11).

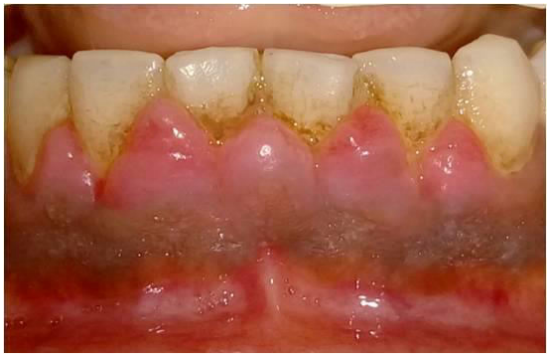

Fig 7: Gingival inflammation with papillary gingival enlargement with 33 to 43.

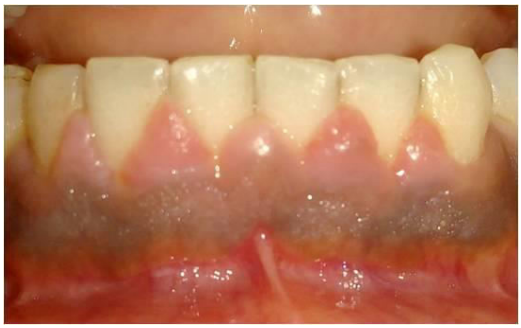

Fig 8: Following phase I periodontal therapy, firm and healthy gingival tissue.

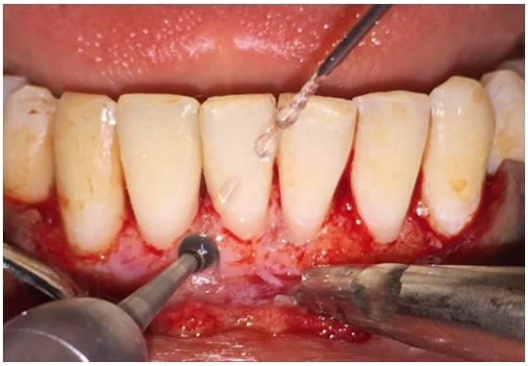

Fig 9: Osseous resection performed using round carbide bur.

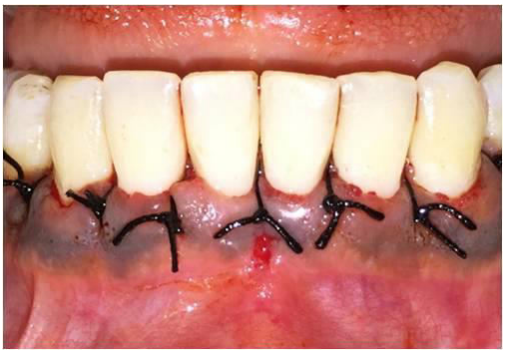

Fig 10: 4-0 Black silk sutures placed using simple interrupted suturing technique.

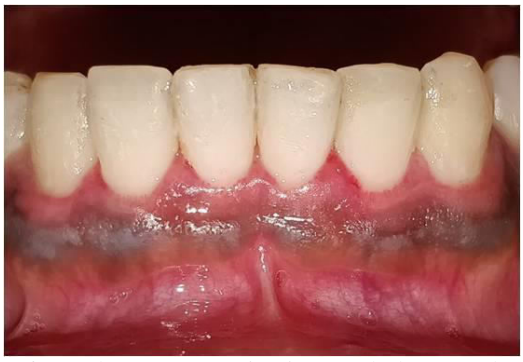

Fig.11: 6 months post-operative intraoral photograph of case 2.

DISCUSSION:

Chronic irritation to the gingival tissues can induce inflammation, which produces granulation tissue of endothelial cells and chronic inflammatory cells followed by proliferation of fibroblasts and manifest as an overgrowth or enlargement.[5] The definitive diagnosis of inflammatory gingival enlargement can only be ascertained through histopathological findings. Ramfjord S in 1953 studied histopathological features of inflammatory gingival enlargement and stated that peripheral zones of irritation and inflammation, found in the crevicular areas and at the base of protruding lesions act as a stimulus for continuous growth. The fibrotic hyperplastic gingival tissues can persist even following elimination of the initial source of irritation may predispose to further inflammation.[6]

Zanatta FB in 2014 suggested a positive co-relation between fixed orthodontic therapy and gingivitis and gingival enlargement. They also concluded that anterior gingival enlargement is associated with gingival inflammation and excess resin around brackets.[7] The mechanism of such enlargement as suggested by Trackman in 2004 is increased production by fibroblasts of amorphous ground substance with a high level of glycosaminoglycans. Increases in mRNA expression of type I collagen and up-regulation of keratinocyte growth factor receptor could play an important role in excessive proliferation of epithelial cells and development of gingival enlargement.[8] Pinto AS 2017 concluded that the occurrence of gingival enlargement increased as the duration of orthodontic treatment increased and showed that patients undergoing orthodontic treatment had a 20 to 28-fold increased risk for gingival enlargement than without orthodontic appliances.[9]

Treatment options of inflammatory gingival enlargement are mostly aimed at reducing bacterial plaque, which include mechanical removal of bacterial deposits by non-surgical periodontal therapy and antiseptic mouthrinses.[10] The surgical treatment should only be contemplated after appropriate and meticulous non-surgical periodontal therapy. When deciding between gingivectomy and flap surgery, underlying osseous topography plays a crucial role. Even in the absence of periodontal pockets, reflecting a flap to gain access to correct underlying osseous architecture is needed in few cases. Intraoral peri-apical radiographs and bone sounding is important part of diagnosis in these patients.

CONCLUSION:

Thorough dental and medical history of the patient, proper assessment of the type, nature and extent of enlargement and knowledge of etiologic factors and risk factors is the key for successful diagnosis of gingival enlargement. Reinforcement of effective oral hygiene maintenance is of utmost importance in such set of patients. Oral hygiene education and positive patient motivation should be performed in order to obtain predictable outcomes.

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