SURGICAL MANAGEMENT OF GINGIVAL ENLARGEMENT: A CASE SERIES

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ABSTRACT

Introduction: Gingival enlargement is defined as an overgrowth or increase in size of gingiva. Gingival enlargement is associated with multiple factors including inflammation, medications, neoplasia, hormonal disturbances and heredity. A case of gingival enlargement should be treated in a step-wise manner, including consultation with the patient’s physician, substitution of the drug, nonsurgical therapy, surgical therapy (if needed), and supportive periodontal therapy after every 3 months.

Case series: This case series presents diagnosis and management of amlodipine induced gingival hyperplasia and idiopathic gingival enlargement. Drug-induced gingival enlargement and idiopathic gingival enlargement was diagnosed and managed by scaling and root planing followed by gingivectomy with 6 months follow up.

Conclusion: Conventional gingivectomy with oral hygiene measures and regular followup is the treatment of choice for such presentation.

Key words: Drug- induced enlargement, idiopathic gingival enlargement, gingivectomy

INTRODUCTION

Gingival enlargement is defined as an abnormal growth of the gum tissue. It is associated with multiple factors including: inflammation, hormonal, drug use, neoplasm, genetic, systemic and idiopathic.(1,2,3) Gingival enlargement is one of the side effects associated with the administration of several drugs.(4) Currently, more than 20 drugs are associated with gingival enlargement. Drugs having side effect of gingival enlargement can be broadly divided into three categories: anticonvulsants, calcium channel blockers and immunosuppressant’s. Amlodipine-induced gingival enlargement is comparatively less prevalent among calcium channel blockers. Since pathogenesis of gingival enlargement is not well-understood, it is still a challenge for the periodontists to diagnose and manage the case effectively.

Idiopathic gingival hyperplasia has been described in several case reports. The clinical presentation of the gingiva is pink, firm, fibrotic and nonhemorrhagic. Severity varies and may cover part/all of the crowns of the erupted teeth. General histological findings include normal overlying epithelium, rete pegs extending deep into underlying connective tissues with some areas of hyperplasia, proliferating dense fibrous CT with increased cellularity and coarse collagenous fiber bundles and hyperkeratosis and acanthosis with elongated papillae.(5)

The purpose of this paper is to present a case series of a patients with amlodipine induced gingival enlargement and idiopathic gingival hyperplasia and their management with 6 month follow up.

Case 1

A 47-year-old male was referred to the Department of Periodontics with complaints of gingival enlargement with foul odor, bleeding and fetid discharge from gums since 1 year. Gingival tissues were pale pink, enlarged, firm, and fibrotic. Generalized bleeding on probing was present. General examination revealed normal built. A diagnosis of generalized drug-induced gingival enlargement superimposed with periodontitis was made. With the consent of the patient and her physician, complete professional oral prophylaxis was performed, along with a prescription of a 0.2% chlorhexidine mouthwash (10 ml BID for 7 days). With the patient’s and physician’s
consent, phenytoin was substituted with losartan 50mg. After completion of Phase I treatment, a quadrant-wise gingivectomy was performed under local anesthesia. BP blades were used for incisions on the facial and lingual surfaces. Orban periodontal knives were used for interdental incisions. Co-pack was given in all quadrants to reduce patient discomfort.

**Case 2**

A 18-year-old female patient reported to the Department of Periodontics with a chief complaint of swollen gums involving her upper front teeth since last three years preventing proper speech, and mastication, causing poor esthetics. She did not give any history of drugs intake, fever, anorexia, weight loss, seizures, hearing loss, nor having any physical or mental disorder. Also familial and postnatal history was non-contributory. Extraoral examination revealed symmetry of the face and there were no findings of lymphadenopathy. Intraoral examination revealed, generalized diffuse type of gingival enlargement involving both maxillary arch. Gingiva was pale pink and firm in consistency. No signs of acute inflammation were present. In addition, halitosis was accentuated, and generalized probing pocket depth in the range of 7 and 10 mm. After completion of Phase I treatment, gingivectomy was performed under local anesthesia. BP blades were used for incisions on the facial and lingual surfaces. Co-pack was given in all quadrants to reduce patient discomfort. Healing was uneventful with no signs of recurrence for 6 months.
DISCUSSION

Gingival hyperplasia, with its potential cosmetic implication and tendency to provide niche for further growth of microorganism, possess a serious concern to patients and clinicians. Calcium channel blockers are considered as potential etiological agent for inducing gingival enlargement. Lafzi et al. (2006) had reported rapidly developing gingival hyperplasia in patient receiving 10 mg/day of amlodipine within 2 month of onset. The prevalence of amlodipine-induced gingival overgrowth was reported to be 3.3% (Jogersen, 1997). The underlying mechanism of gingival enlargement still remains to be fully understood. However, two main inflammatory and non-inflammatory pathways have already been suggested. The proposed non-inflammatory mechanisms include defective collagenase activity due to decreased uptake of folic acid (7) blockage of aldosterone synthesis in adrenal cortex and consequent feedback increase in ACTH level (8) and upregulation of keratinocyte growth factor (9). Alternatively, inflammation may develop as a result of direct toxic effects of concentrated drug in crevicular gingival fluid and/or bacterial plaques (10). This inflammation could lead to the upregulation of several cytokine factors such as TGF-β1 (11).

Gagliano et al. (12) suggested that gingival hyperplasia of different etiologies may have different mechanisms of overgrowth. The likely mechanism of idiopathic gingival hyperplasia may be increased collagen deposition as a consequence of post translational mechanisms. The increase in collagen cross-links renders it less susceptibility to MMP degradation, favoring its accumulation in the gingival connective compartment. The cellular and molecular mechanisms that lead to this condition are not clear. Few authors observed that the proliferation rate is lower in HGF fibroblasts compared to normal gingiva controls. (13) But, recent studies have shown that fibroblasts from these types of gingival enlargement proliferate faster than those of normal gingiva. (14) Recently, role of sex hormones have been suggested in gingival enlargement. (15) According to few reports the increase in collagen synthesis and other extracellular matrix components, such as fibronectin and glycosaminoglycans, (14) and decreased levels of matrix metalloproteinases (MMP-1 and MMP-2) may be involved in gingival enlargement.

Management of gingival hyperplasia depends on the cause of the condition. Drug-induced gingival hyperplasia may improve with substitution of other drugs that rarely affect the gingiva, such as losartan. In general, reinforcement of good home care oral hygiene regimens and periodic professional surgical excision of gingival tissue are the treatments of choice. The present series describes a patients with an idiopathic gingival hyperplasia and drug induced gingival enlargement and their management.
CONCLUSION

Successful treatment of drug induced gingival enlargement and idiopathic gingival enlargement depends on the proper identification of etiologic factors and improving oral hygiene status, esthetics, and function through elimination of local factors and surgical excision of the overgrowth.

ACKNOWLEDGEMENT

Authors acknowledge the immense help received from the scholars whose articles are cited and included in references of this manuscript. The authors are also grateful to authors / editors / publishers of all those articles, journals and books from where the literature for this article has been reviewed and discussed.

REFERENCES