Case Report

Management of Amlodipine Induced Gingival Enlargement

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ABSTRACT

Calcium channel blockers by far are most prescribed drugs for the treatment of cardiac disorders and play a significant role in gingival overgrowth. Nifedipine is most commonly implicated in most of such cases. This article shows the rare case series involving association between amlodipine and overgrowth, as well as brief review of its effect on gingiva and also deals with management of enlargement surgically.

Keywords:amlodipine, enlargement, hypertension

INTRODUCTION

Gingival enlargement represents an overgrowth response to a variety of local and systemic conditions. It is a well-recognized unwanted side-effect associated with three major drugs/drug groups – phenytoin, cyclosporine and the calcium channel blockers. The prevalence of this unwanted effect varies between drugs, and a variety of risk factors have been identified in relation to the expression of drug-induced gingival overgrowth.[1]Calcium channel-blocking agents are used extensively for the management of cardiovascular conditions, including angina pectoris, coronary artery spasm, cardiac arrhythmias-i.e. supraventricular tachycardia-and hypertension.[2]Amlodepine is a third generation dihydropyridine which has been found to be very useful in middle aged to older aged adult patients for various cardiovascular conditions. It functions by inhibiting calcium ion influx across cell membranes of heart and smooth muscles thereby, blocking its intra-cellular mobilization.[3] Once medication-induced gingival enlargement of the gingiva is present, it usually does not respond well to plaque control or scaling and root planing. Therefore hyperplastic gingival tissues need to be surgically excised.[4]

CASE REPORT

The first case was 54 year old lady who gave positive history of hypertension and diabetes and was on Amlodipine 10mg taken daily since last 2 years and Miglitol 50mg (Fig. 1). Intraoral examination revealed generalized pale pink, firm and resilient, with irregular contours having nodules on the gingiva in the maxillary jaw, covering the portions of the crown. Periodontal pockets were present in all the upper teeth and lower anterior teeth along with subgingival plaque and calculus that caused secondary inflammation at some sites.

INVESTIGATIONS

The investigations carried out were complete haemogram, orthopantogram and biopsy. Thehaemogram showed all values within the normal variable range. There was mild bone loss associated, as revealed by orthopantogram. The

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histopathological examination showed hyperplastic parakeratinised epithelium. Rete pegs were found to be elongated in some areas and in some region they were broad and blunt. Underlying connective tissue showed fibrous hyperplasia with coarse collagen fiber bundles and numerous fibroblasts. Chronic inflammatory cells along with few scattered giant cells and capillaries indicating a superimposed inflammation were present.

Management of the gingival overgrowth

The patients were advised to substitute or discontinue the medication in consultation with physician. After obtaining prior consent from the patient and physician, and controlling the other systemic factors, the patients were given intensive oral hygiene instructions. Phase-I periodontal therapy comprising mechanical removal of plaque and calculus as well as patient instructions on oral hygiene maintenance was initiated(Figure 2). The patient was also prescribed an adjunctive rinse of 0.2% chlorhexidine mouthwash twice daily.Following oral prophylaxis and substitution of amlodipine, significant improvement in the gingival tissues was observed. It was then followed by phase II periodontal therapy with an internal bevel gingivectomy and flap operation(Figure 3& 4)).Histopathological examination of the specmen confirms the overgrowth of collagen fibres (Figure 5).

Follow up

Patient is follow up of both the patient is done for 1 year and recurrence is not noted.

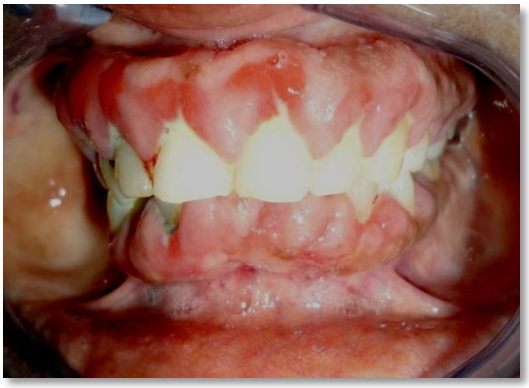

Figure 1. Enlargement before Phase I Periodontal therapy

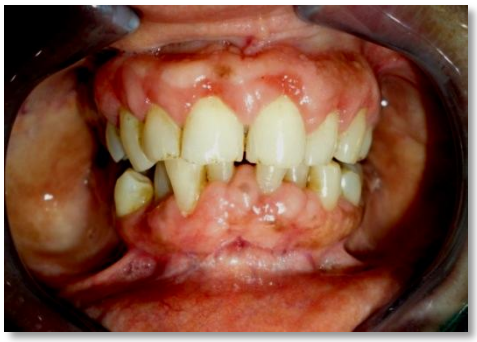

Figure 2. After Phase I Therapy & Drug Substitution

Figure 3. Internal bevel gingivectromy

Figure 4. Flap Reflection & Debridement

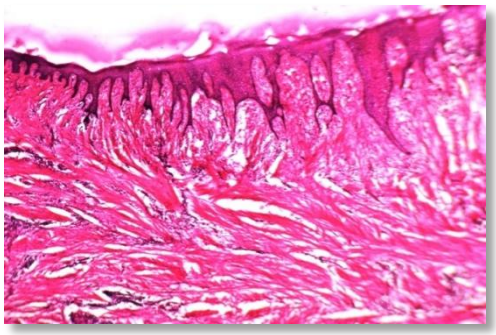

Figure 5 Histopathology showing Overgrowth of collagen fibres

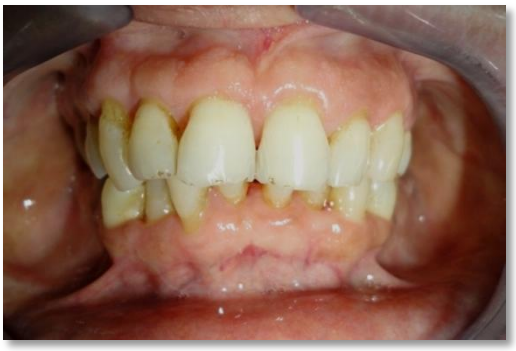

Figure 6. Post Treatment after 1 year

DISCUSSION

This article presentscase reportsof Amlodipine induced gingival overgrowth and its management accordingly. Accurate determination of prevalence rates in each drug category is extremely difficult due to differences in the reported prevalence rates. These differences may be due, at least in part, to assessment of enlargement by medical versus dental personnel, differing indices of overgrowth, focus on institutionalized versus outpatient populations, type of systemic condition being treated, age of the patients, other medications administered simultaneously, poorly controlled underlying periodontal conditions, and other factors.

The underlying mechanism for the pathogenesis of this gingival over-growth remains to be fully understood. These drugs which affect intracellular calcium metabolism or transport may in some patients stimulate gingival fibroblasts to cause increased deposition of extracellular matrix components, such as glycosaminoglycans.[5] The other proposed non-inflammatory mechanisms include defective collagenase activity, blockage of aldosterone synthesis in adrenal cortex which is also calcium dependent and causes a consequent feedback increase in ACTH level[6], and up-regulation of the keratinocyte growth factor.[7]Inflammatory mechanisms include direct toxic effects of the concentrated drug in gingival crevicular fluid and/or bacterial plaques. This inflammation could lead to up-regulation of various cytokines such as TGF-β1.[8]

Cases in which there is no underlying bone loss, external bevel gingivectomy and gingivoplasty is done. In cases with presence of attachment loss and underlying osseous defects, a procedure called internal bevel gingivectomy with flap operation is performed so as to be able to resect the gingival overgrowth and in addition treat the underlying osseous defects.[9,10] All the three cases presented had some degree of bone loss and hence internal bevel gingivectomy was performed. The patients were then maintained on follow-up for a period of upto one year, no recurrences were seen.

CONCLUSION

Management of the patients with drug induced gingival overgrowth thus aims at removing the etiologic factor first by either discontinuation or substitution of the drug after physician consultation. Patients should be placed on a program of meticulous plaque control along with frequent quarterly professional cleanings. Surgical treatment to excise and remodel the gingival contour should beconsidered whenever gingival overgrowthcauses aesthetic and functional problems. However, it should be taken into consideration that these treatment modalities, although effective, do not necessarily prevent recurrence of the lesions.

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