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Case Report

Amlodipine Induced Gingival Enlargement- A Case Report

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ABSTRACT

Gingival enlargement, an over-exuberant response, caused due to a large number of local and systemic factors. Drugs like certain anticonvulsants, immuno-suppressive drugs and a number of calcium channel blockers are one of the common causative factors which ARE a cause of concern for both patient as well as the clinician. Amlodipine is a calcium channel blocker that is widely used because of its safety profile and duration of action for the management of hypertension and angina. This case report describes the management of gingival enlargement in a 55 year old female hypertensive patient taking amlodipine. The treatment aspect included phase one therapy, substitution of the drug, surgical excision and maintenance and supportive therapy.

Keywords: Amlodipine, calcium channel blockers, gingival overgrowth

INTRODUCTION

Gingival enlargement or gingival overgrowth is increase in size of the gingiva. Gingival overgrowth is a well recognized, unwanted effect and one of the most important clinical features of gingival pathology. It has multi factorial etiologies caused by various local and systemic factors that is frequently associated with inflammatory changes in the gingiva.[1] Severe gingival enlargement is often disturbing and can interfere with speech, occlusion and mastication. Drug-influenced gingival overgrowth is defined as gingival enlargement resulting in whole or in part from systemic drug use.[2] Medication mainly implicated are the anticonvulsant such as phenytoin for treatment to control seizure disorders in epileptic patient, calcium channel blockers (ccb) such as Nifedipine for treatment of hypertension or angina pectoris, immunosuppressant such as cyclosporine for treatment to prevent rejection in patient receiving organ transplant.[3] These drugs are having different pharmacologic effect though all of them act similarly on a secondary target tissue, that is, the gingival connective tissue causing common clinical and histopathological findings. An increasing number of medications are associated with gingival overgrowth. Currently, more than 20 prescription medications are associated with gingival enlargement.[4] The pathogenesis of drug influenced gingival enlargement suggest that it is multifactorial including; age, genetic predisposition, pharmacokinetic variables, drug induced alteration in gingival connective tissue homeostasis, plaque induced inflammatory changes and drug induced action on growth factors.[3]

Amlodipine is a third generation dihydropyridine calcium channel blocker

used in the management of both hypertension, and angina and it is known to interfere with the production of collagenase through altered Ca influx into fibroblast which allows collagen accumulation without degradation. *Ellis et al* first reported gingival sequestration of amlodipine and amlodipine-induced gingival overgrowth in 3 adult dentate patients.[5] *Jorgensen*, 1997 had reported the prevalence of amlodipine-induced gingival enlargement as 3.3%.[6]

Clinical manifestations of gingival enlargement frequently appear within one to three months, after initiation of treatment with the associated medications. Gingival overgrowth normally begins at the inter dental papillae and is more frequently found in the anterior segment of the labial surfaces. Gradually, gingival lobulations are formed that may appear inflamed or fibrotic in nature depending on the degree of local factor-induced inflammation. However, the fibrotic enlargement is normally confined to the attached gingiva, but may extend coronally causing the extensive disfigurement of gingiva.[1]The treatment of drug influenced gingival enlargement is a challenge for the periodontist due to difficulty in the selection of proper line of management and its high recurrence rate.[7] This is a case of amlodipine-induced gingival overgrowth which was being treated thorough Phase-1 therapy, substitution of the drug, surgical excision of the residual gingival overgrowth and maintenance and supportive therapy.

CASE REPORT

A 55-year-old female patient came to the department of Periodontics, Govt. Dental College and Hospital, Srinagar with a chief complaint of swollen gums since 2-3 months. Initially the swelling appeared in the upper and lower front tooth region as small bead-like nodular enlargement of the gums which gradually progressed to involve whole of the gingiva to attain the present size. The swelling was associated with foul odor, bleeding, fetid discharge from gums and discomfort on chewing. The medical history of the patient revealed that the patient was hypertensive and taking antihypertensive drug, amlodipine -10 mg/day, single dose orally since 5 years. However, her past dental history was noncontributory. The personal history revealed that she cleaned her teeth once daily with brush and paste, which she discontinued since past 1 month due to enlarged and bleeding gums. Her general physical examination revealed that the patient was well built and her vital signs were within the normal range. There were no significant extraoral findings.

On intraoral examination there was generalized massive gingival enlargement, which was more prominent on the labial aspect of maxillary and mandibular teeth. The marginal and interdental gingiva was enlarged as well as covering almost coronal one-third of maxillary and mandibular teeth. The gingiva was red, erythematous, lobulated with stippling absent. On palpation the gingiva was soft and edematous. There was bleeding on probing and suppuration was seen in relation to 37. The mean plaque index was 2.6 and mean gingival index was 2.1. Grade III mobility was seen in relation to 37and grade I mobility with 31, 32, 41 and42 was present. Patient's oral hygiene on assessment was poor with presence of local irritating factors contributing to the inflammatory component of the gingival enlargement. The probing of gingival sulcus revealed presence of pseudo-pockets.

Patient was subjected to complete hemogram and all the parameters were found to be within normal range. Intra-oral periapical radiograph showed moderate generalized bone loss, with periapical radiolucency in relation to 37. On the basis of the patient's history and clinical features, a clinical diagnosis of amlodipine-induced gingival enlargement was made.

The treatment of the patient was started with preliminary phase constituting the extraction of 37. Then the patient was subjected to non-surgical phase which included the planned sessions of scaling and root

planing. Patient was instructed to maintain good oral hygiene with the use of 0.12% chlorhexidine oral rinses twice daily. Patient was referred to a physician regarding drug substitution or withdrawal of the drug. The physician substituted the drug Ramipril 5 mg, once daily. Patient reported after 1 month with regression in the size of gingival enlargement. The patient was then kept on maintenance phase for next six months. After six months there was a marked reduction in then enlargement with minimal of fibrotic component left. Finally, surgical excision of gingival hyperplastic tissue was planned employing the techniques of gingivectomy/gingivoplasty to restore the normal shape and contour of the gingiva. Under local anesthesia, the enlargement was removed by blade and scalpel. There were no post-operative complications and the healing was uneventful. The patient was followed-up regularly and at the end of 6 months patient remains asymptomatic with no signs of recurrence.

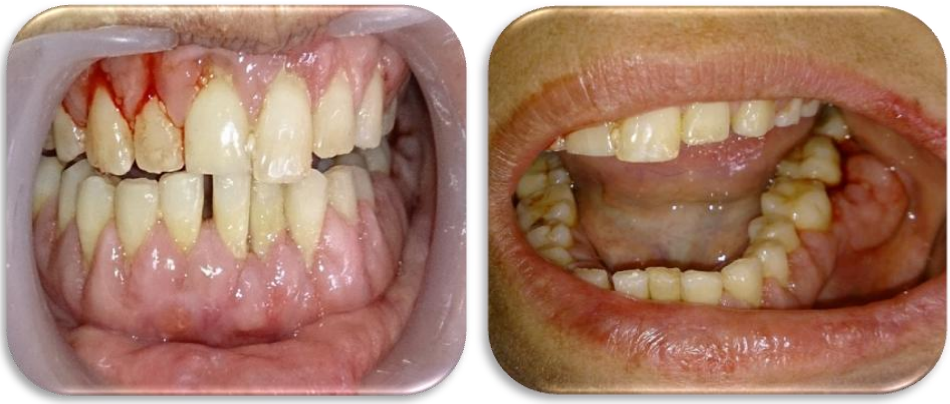

Fig. 1: Preoperative View Showing Gingival Overgrowth

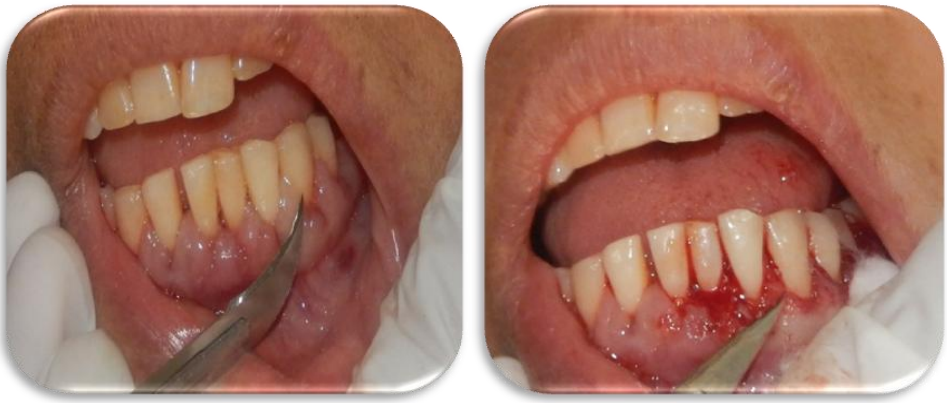

Fig. 2: Gingivectomy By Scalpel And Blade

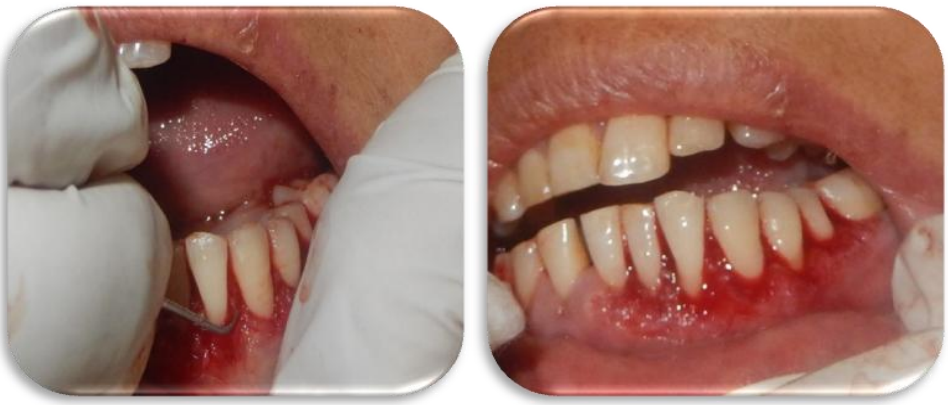

Fig. 3: Root Planning after Gingivectomy

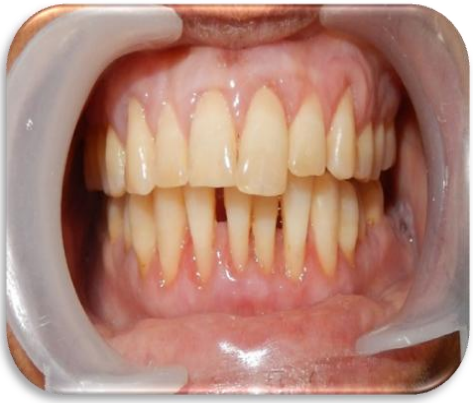

Fig. 4: 3 Month Post Operative

DISCUSSION

Gingival enlargement or gingival overgrowth, a common clinical condition that occurs due to many reasons such as inflammation, drug intake associated conditioned, neoplastic or false enlargements. It has potential cosmetic implications and also provides new niches for the growth of microorganisms that can pose serious concern for both the patients and clinician. The drug induced enlargement is caused by anticonvulsant (phenytoin), calcium channel blockers (amlodipine) and immunosupressants (cyclosporine). Lafzi *et al.*, had reported rapidly developing gingival hyperplasia in patient receiving 10 mg/day of amlodipine within 2 month of onset.[8] The prevalence of amlodipine-induced gingival overgrowth was reported to be 3.3% .[6]

The mechanisms by which calcium antagonists induce gingival hyperplasia have yet to be fully explained. Two main pathways have been suggested - inflammatory and non-inflammatory pathway. Among the several proposed mechanisms, the best hypothesis so far under non-inflammatory mechanisms is that calcium antagonists inhibit the influx of calcium ions that is needed for degradation and synthesis of collagen.[9] The accumulated collagen and other extracellular matrix not degraded are suggested to produce gingival hyperplasia. In addition to this mechanism, the importance of good oral hygiene for prevention of gingival hyperplasia is emphasized.[10] Inflammation may develop as a result of direct toxic effects of concentrated drug in crevicular gingival fluid and/or bacterial plaques. This inflammation could lead to the upregulation of several cytokine factors such as transforming growth factor beta 1.[11]

The treatment options for drug-induced gingival enlargement should be based on the medication being used and the clinical presentation of the individual case. The clinician should emphasize plaque control as the first step in the treatment of drug-induced gingival enlargement. Although the exact role played by bacterial plaque in drug-induced gingival enlargement is unclear, there is evidence that elimination of local factors and regular maintenance of good oral hygiene decrease the degree and severity of the gingival enlargement and improve the overall gingival health.[12] In this present case, there was reduction in the overgrowth after the initial Phase I therapy was performed .The drug discontinuation or substitution also has to be considered .This requires consultation with the patient's physician. It may take from 1 to 8 weeks for resolution of gingival overgrowth. Consideration may be given to the use of another class of antihypertensive medications, which are known to be not associated with the gingival enlargement. In the present case, substitution of the drug amlodipine by Ramipril 5 mg, once daily along with Phase-1 therapy resulted in clinically significant improvement in four weeks time.

The timing and need for surgical intervention has to be assessed carefully for aesthetic/cosmetic needs before any functional consequences are present. The classical surgical approach has been the external bevel gingivectomy. However, a total or partial internal gingivectomy approach has been suggested as an alternative. In the present report, external bevel gingivectomy followed by

gingivoplasty was carried out by blade and scalpel under local anesthesia. The postoperative results were found to be extremely satisfactory both esthetically and functionally. Patient was recalled every month until first 3 months and then at the end of 6 months. Regular oral hygiene reinforcement and scaling was carried out during the maintenance phase.

CONCLUSION

This is a case of amlodipine induced gingival enlargement treated by phase I therapy along with drug substitution followed by gingivectomy. The gingival enlargement caused by drug intake is likely to increase in the years to come. Strictly maintaining good oral hygiene, switchover to alternative drugs and surgical therapy if required, remains the main stay of available treatment modalities in drug induced gingival enlargements.

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