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Case Report

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# Drug induced gingival enlargement in a hypertensive patient: A case report

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## Abstract

Gingival enlargement caused due to a large number of local and systemic factors. Drugs like certain anticonvulsants, immuno-suppressive drugs and some calcium channel blockers are the common causative factors which are a cause of concern for both the patient as well as the clinician. Amlodipine is a calcium channel blocker that is widely used because of its safety profile and longer duration of action for the management of hypertension and angina. This case report describes the management of gingival enlargement in a 47 year old female hypertensive patient taking amlodipine. The treatment aspect included phase one therapy, the substitution of the drug, surgical excision and maintenance and supportive therapy.

**Keywords:** Amlodipine, calcium channel blockers, gingival overgrowth, drug induced gingival overgrowth

## Introduction

Gingival enlargement or gingival overgrowth is an increase in the size of the gingiva. Gingival overgrowth is a well-recognized and unwanted condition that is one of the most important clinical features of gingival pathology. It has multi-factorial etiologies caused by various local and systemic factors that are frequently associated with inflammatory changes in the gingiva. The factors causing enlargement of gingiva can be divided into five groups, namely inflammatory gingival enlargements, drug-induced gingival hyperplasia (DIGH), GO associated with systemic conditions, GO associated with systemic diseases and hereditary gingival fibromatosis. Drug-induced gingival enlargement mainly results from drugs that are intended for disorders of non-dental origin. The class of drugs usually implicated in gingival enlargement, in the form of their adverse drug reactions, includes immune suppressants, anticonvulsants, and calcium channel blockers (CCBs). Genetic factors, drug dosage and local factors can affect the development and severity of DIGH. DIGH, depending upon its severity may cause difficulty in chewing, altered phonetics, oral hygiene deterioration, and can lead to the disfigurement of the gingival tissue. Occasionally, it may lead to increased mobility and migration of the teeth due to alveolar bone loss. CCBs inhibit the influx of calcium ions through cell membranes and act on vascular smooth muscles, the cardiac nodes (sinoatrial and atrioventricular nodes), and the cardiac myocytes. They act on L-type calcium channels of these tissues, thereby causing coronary and peripheral arterial vasodilation, reduced heart rate, reduction in myocardial contractibility and oxygen utilization by the myocardium, and slow conduction at atrioventricular nodes.[1] Thus, CCB is used for the management of hypertension, cardiac arrhythmias, angina pectoris, and coronary artery spasms. The degree of fibrosis and inflammation depends on the dose, duration, type of drug, oral hygiene and individual susceptibility including genetic factors and environmental influences. The first case of GO associated with the calcium channel blocker nifedipine was reported in 1984.[2] Through interference with calcium metabolism, calcium channel blockers decrease calcium levels in gingival fibroblasts and T cells, thereby affecting T-cell proliferation or activation and collagen biosynthesis.[3] In addition to fibroblast metabolism and function, inflammatory regulation of tissue turnover is a major factor in DIGH pathogenesis. Fibroblast functions such as proliferation, differentiation and production of the extracellular matrix are affected by levels of cytokines and growth factors. GO lesions are characterized by increased levels of Interleukin-6 (IL-6), IL-1, platelet-derived growth factor subunit B (PDGFB), fibroblast growth factor 2 (FGF2), transforming growth factor (TGF), and connective tissue growth factor (CTGF).[4] Macrophages are the main source of these cytokines.

## Case Description

A 47 year old hypertensive female patient visited the Department of Periodontics, at Dr. R Ahmed Dental College and Hospital, with the chief complaint of gingival swelling for the last 1 month. The patient gave a history of antihypertensive drug (Amlodipine 10mg/day) for the last 6 months. On intraoral examination, a generalized gingival overgrowth was observed with poor oral hygiene, predominantly involving the interdental papillae. Gingival enlargement was severe in the first quadrant, almost covering the entire crown of teeth 12 (Figure 1). The enlarged gingiva of 12 was 10×9 mm in diameter, firm and non-tender in consistency, pale pink in colour and did not bleed easily on probing while the hard tissue examination revealed increased

mobility in relation to 12 (Figure 2, Figure 3). Panoramic radiographs revealed generalized bone loss (Figure 4). The patient underwent phase 1 therapy along with meticulous oral hygiene instructions and was sent to the physician concerned regarding drug substitution. The patient was evaluated after 1 week and a marked improvement was noted except in the upper right quadrant. Drug substitution was done by the physician and the patient was under Telmisartan 40mg /day. Gingival overgrowth removal surgery was planned and an informed consent was taken from the patient. An external bevel gingivectomy procedure was planned that included pocket measurement followed by pocket marking with a pocket marker. An external incision was given apical to the base of the pocket towards the coronal direction (Figure 5, Figure 6, Figure 7). Complete excision of lesion done followed by periodontal pack placement (Figure 8, Figure 9, Figure 10). The Excised tissue was stored in a formalin solution and was sent for histopathological evaluation. Histopathology revealed stratified squamous epithelium with hyperplasia and acantholysis. The underlying fibro-collagenous connective tissue showed dense mixed inflammatory infiltrate with congested blood vessels (Figure 11). The patient was recalled for follow-up after 10 days and again at an interval of 6 months.

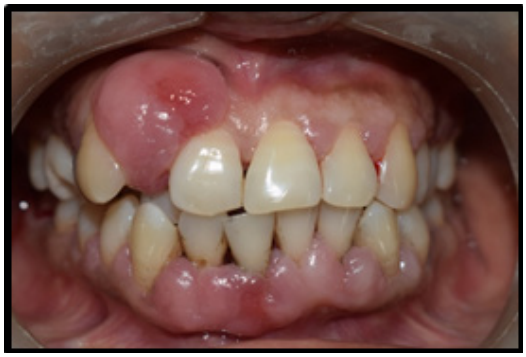

**Figure 1** A case of drug induced gingival enlargement.

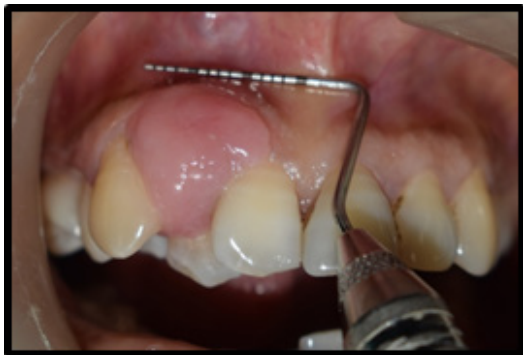

**Figure 2** Measuring Gingival Enlargement with UNC 15 Probe.

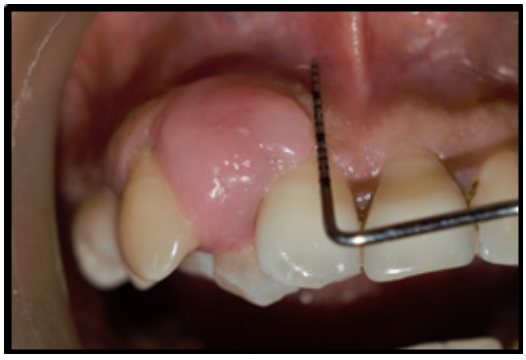

**Figure 3** Measuring Gingival Enlargement with UNC 15 Probe.

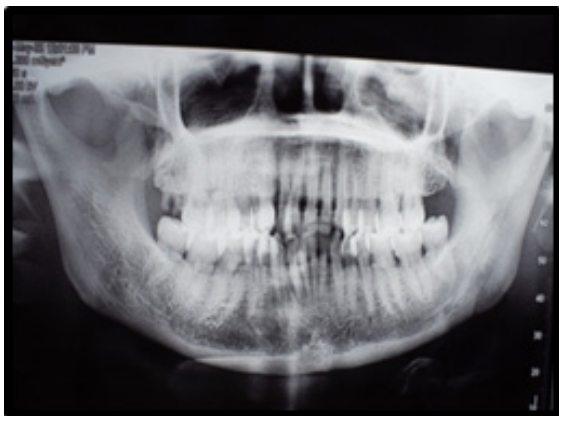

**Figure 4** OPG reveals interdental bone loss w.r.t 34.

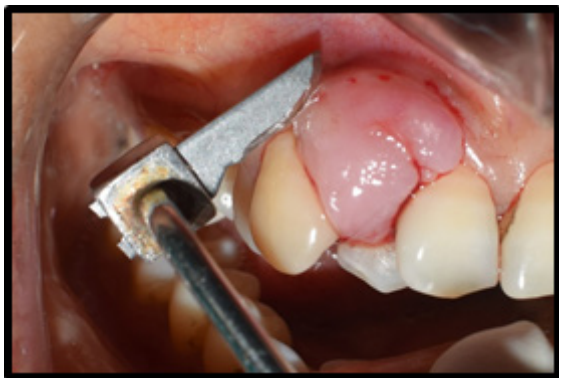

**Figure 5** External bevel incision given.

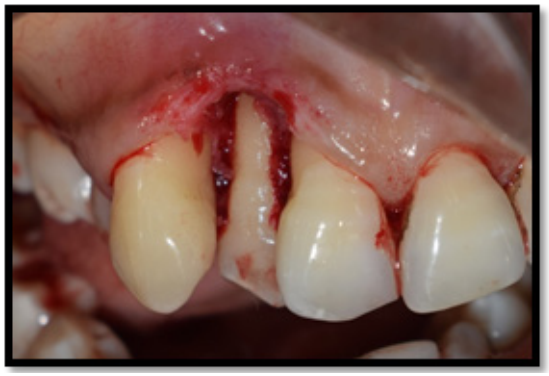

**Figure 6** The surgical wound.

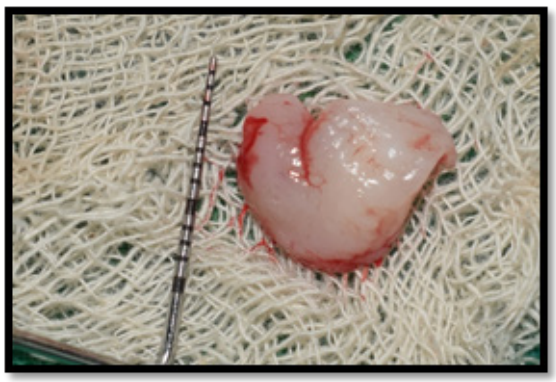

**Figure 7** The excised mass.



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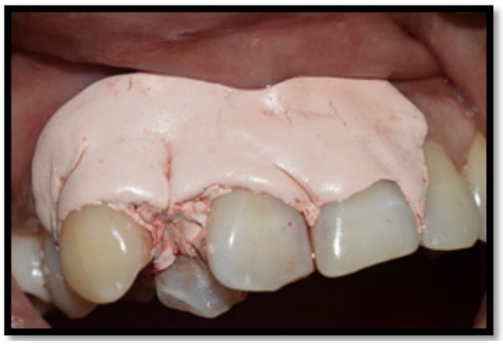

**Figure 8** Periodontal pack applied.

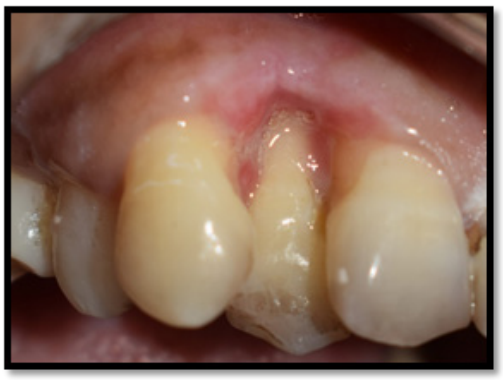

**Figure 9** 10 days post-operative view.

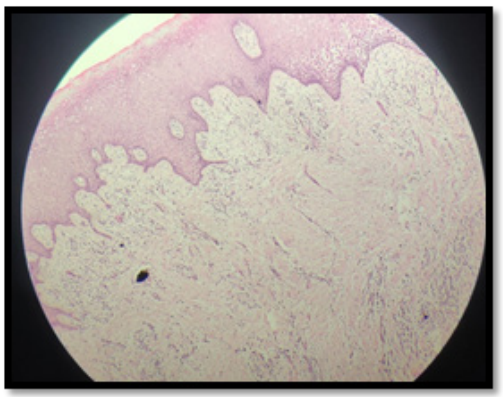

**Figure 10** h/p shows pronounced hyperplasia of collagen tissue.

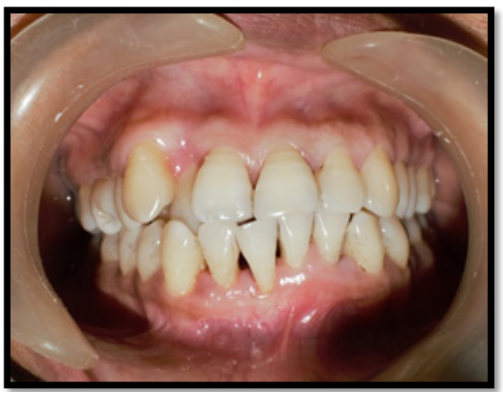

**Figure 11** 6 month post-operative view.

## Discussion

Amlodipine is a very common drug used on hypertensive patients. Lafzi et al. reported rapidly developing gingival hyperplasia in patients receiving 10 mg/day of amlodipine within 2 months of onset.[5] 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. Among the several proposed mechanisms, the best hypothesis so far under the non-inflammatory pathway is that calcium antagonists inhibit the influx of calcium ions that are needed for the degradation and synthesis of collagen.[7] The accumulated collagen and other extracellular matrices that are not degraded are suggested to produce gingival hyperplasia. In addition to this mechanism, the importance of good oral hygiene for the prevention of gingival hyperplasia is emphasized.[8] Inflammation may develop as a result of direct toxic effects of concentrated drugs 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 (TGF-β1). In this case, there was a reduction in the overgrowth after the initial Phase I therapy and drug substitution. Esthetics considerations had to be taken into account in this case. So external bevel gingivectomy was done which is supported by a similar study done by Khairat et al.[9] The postoperative results were extremely satisfactory both esthetically and functionally. The patient was recalled after 10 days and then again at the end of 6 months. Regular oral hygiene reinforcement and scaling were 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. A satisfactory result was achieved. Strict maintenance of good oral hygiene, substitution of drugs and surgical therapy if required, remain the mainstay of available treatment modalities in drug-induced gingival enlargements.

## Acknowledgments

None.

## Conflicts of Interest

None.

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