CASE REPORT

Amlodipine-induced Gingival Enlargement

Kritika Jangid, BDS, Jaiganesh Ramamoorthy, MDS, N.D. Jayakumar, MDS
Chennai, India

ABSTRACT

Background: Amlodipine, a calcium channel blocker, used as an antihypertensive drug has been uncommonly associated with gingival enlargement. It is a serious concern for both the patient and the clinician because of the unesthetic appearance, formation of ecological niches for periodontopathic bacteria, and in severe cases cause difficulty in mastication. Drug-induced gingival hyperplasia is more commonly associated with nifedipine, which is also a calcium channel blocker. Although amlodipine is relatively a safe drug of choice and is also a gold standard for the management of hypertension, this case report intends to make the practitioners aware of this side effect of amlodipine and choose for an alternative drug when needed. (J Clin Prev Cardiol. 2014;3(3):99-101)

Keywords: amlodipine; calcium channel blockers; gingival enlargement; gingival hyperplasia.

Background

Amlodipine is a long-acting di-hydropyridine calcium channel blocker (CCB). It is a commonly used drug and is regarded as the gold standard in its efficacy in reducing hypertension and to treat anginal chest pain. It has a long half life of around 35–50 hours; hence it offers 24 hours of blood pressure control enhancing patient compliance. Currently drug-induced gingival enlargement has been associated with three class of drugs namely anticonvulsants, calcium channel blockers, and immunosuppressants. The most common calcium channel blocker known to cause gingival enlargement is nifedipine. It is because of this side effect and longer duration of action that amlodipine has been preferred over nifedipine. However, the prevalence of amlodipine-induced gingival enlargement has been reported as 3.3% (1). Hence care must be taken by the prescribing clinician about this adverse effect and may opt for an alternative drug in susceptible cases.

Case Description

A 55-year-old female patient reported to the Department of Periodontics, Saveetha Dental College, Chennai, with a complaint of swelling of gums for the past 1 year and its progressive nature. History revealed that the patient is a known hypertensive for the past 5 years and is on medication for the same. She had been prescribed 5 mg amlodipine once a day in the morning since then. Patient first noticed slight gingival enlargement around one and a half year back but did not report to the concerned physician or the dentist. The enlargement has been progressive in nature since then up to the present size (Figs. 1–3). On general examination, the patient is normal built.

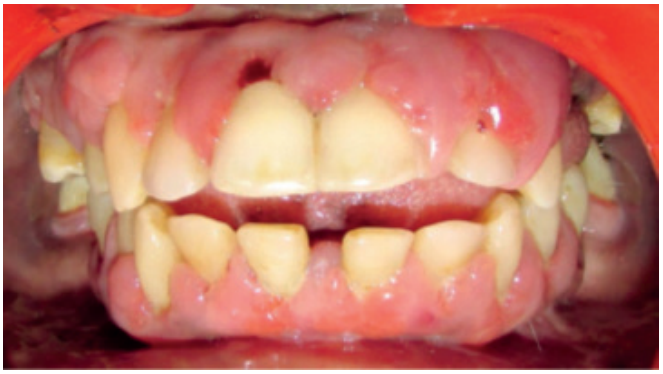

Figure 1. Anterior view at first visit.

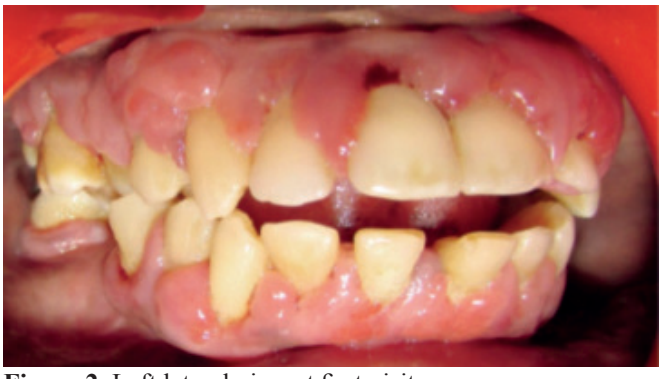

Figure 2. Left lateral view at first visit.

From: Department of Periodontics, Saveetha Dental College, Chennai, India (K.J., J.R., N.D.J)

Corresponding Author: Dr. Kritika Jangid, BDS
Department of Periodontics, Saveetha Dental College, 162 Poonamallee High Road Chennai 600077, India.

Tel: +91 8800131239 E-Mail: doctor.kritika@gmail.com

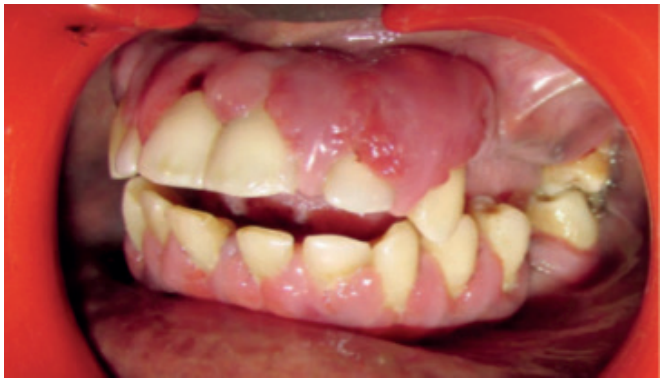

Figure 3. Right lateral view at first visit

Intraoral examination reveals poor oral hygiene, which adds to the inflammatory component to the firm and fibrotic drug-induced gingival enlargement. A generalized nodular enlargement of the gingival is seen from the interdental papilla, marginal gingiva to the attached gingiva. The enlargement does not progress to the oral mucous membrane. Because of the presence of plaque and calculus due to poor oral hygiene, the marginal gingiva and the interdental papilla showed erythematous with soft and edematous consistency. The nodules on the attached gingival, however, showed coral pink color with firm and fibrotic consistency (Figs. 1–3).

Orthopantomograhic examination showed generalized marginal horizontal bone loss up to the middle third of the teeth due to long-standing inflammation and subsequent periodontitis.

Hematological investigations of hemoglobin percentage, fasting and post-prandial blood sugar, bleeding time, clotting time, total white blood cell count, and differential count were all found to be in normal range.

Management

At the first visit, after a detailed case history and hematological investigation, a request letter to the concerned cardiologist was given for the change of drug. The patient reported back after 10 days with the prescription of the concerned cardiologist. The drug was changed to alphadopa 250 mg BD. Also, phase 1 therapy of complete supra and subgingival scaling was done in this visit. The patient was asked to report after 10 days. On the subsequent visit, the patient showed reduced signs of inflammation along with regression of the gingival enlargement to some extent (Figs. 4–6).

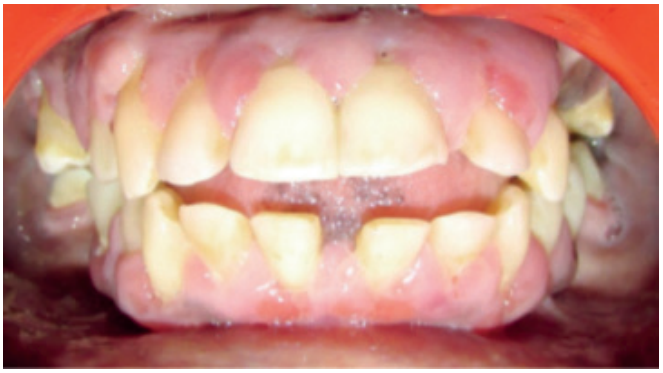

Figure 4. Anterior view after scaling and change of drug.

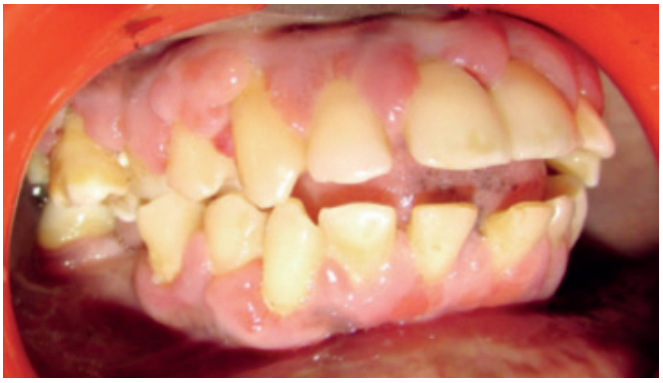

Figure 5. Left lateral view after scaling and change of drug.

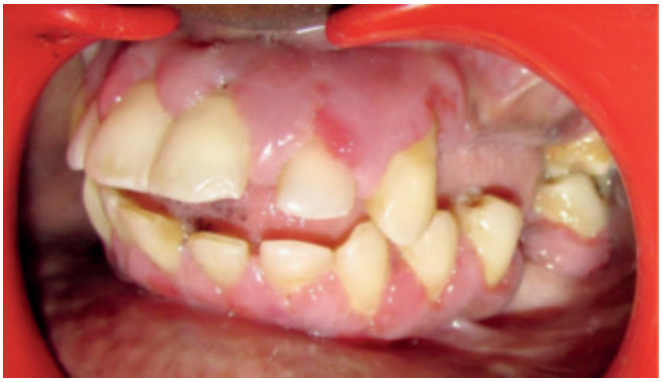

Figure 6. Right lateral view after scaling and change of drug.

Discussion

Amlodipine is a third generation dihydropyridine calcium antagonist, which is structurally similar to nifedipine. The advantages of amlodipine over nifedipine are long duration of action favoring patient compliance and less incidence of gingival hyperplasia. Amlodipine, however, in some individuals has uncommonly been reported to show instances of gingival enlargement.

Gingival enlargement has been classified under inflammatory, drug-induced, idiopathic, associated with systemic diseases and neoplastic. Drug-induced

gingival enlargement has been reported to be caused by anticonvulsants, calcium channel blockers, and immunosuppressants. To diagnose a drug-induced gingival enlargement, a proper case history from the patient about the drug history plays the most important factor. Additionally, drug-induced gingival enlargements have a nodular appearance of the interdental papilla, which extends to the attached gingiva. It is firm and fibrotic in consistency and is usually painless unless superadded acute or chronic infection is present. Because of the painless nature of the gingival enlargement, it is usually ignored by the patient unless the patient is very concerned about his/her esthetics or the enlargement grows to an extent to interfere with occlusion of the teeth.

The exact pathophysiology of amlodipine-induced gingival enlargement has not been clearly understood. Two possible mechanisms have been implicated as inflammatory and noninflammatory pathways. The proposed noninflammatory pathway includes defective collagenase activity due to decreased uptake of folic acid and upregulation of keratinocyte growth factor. Another hypothesis given by Nyska *et al*. is that the calcium antagonist induces blockade of aldosterone synthesis, which is a calcium-dependent pathway in zona glomerulosa (2). This may in turn by a negative feedback mechanism stimulates pituitary secretion of ACTH, which causes zona glomerulosa hyperplasia which stimulates androgens that may act on gingival cells and matrix to produce hyperplasia (2). The possible inflammatory mechanisms are the role of proinflammatory cytokines – IL-1β, IL-6, transforming growth factor-β (TGF-β). Calcium channel blockers have a synergistic effect with IL-1β on gingival fibroblasts to stimulate collagen protein synthesis. Calcium channel blockers also stimulate IL-6, which in turn stimulates the fibroblasts for a positive feedback mechanism to produce collagen and glycosaminoglycans (3). They also stimulate TGF-β to stimulate fibrosis and cause enlargement.

Patients benefit from a thorough supra and subgingival scaling and root planning. The change of drug from calcium channel blockers to a relatively safe antihypertensive is mandatory. In the present case, amlodipine was substituted by alphadopa, which is an alpha adrenergic agonist. If symptoms are not satisfactory, a gingivectomy using conventional scalpel or laser may be performed.

Conclusion

Amlodipine, a commonly used antihypertensive drug, can uncommonly lead to gingival enlargement. It presents as a firm fibrotic growth seen on the gingiva. Careful management by substituting the calcium channel blocker to a relatively safe drug along with oral prophylaxis serve as the first-line treatment of amlodipine-induced gingival enlargement.

Source of Funding

Nil

Conflict of Interest

None declared

References

1. Jorgensen MG. Prevalence of amlodipine-related gingival hyperplasia. *J Periodontol*. 1997;68:676–8.
2. Nyska A, Shemesh M, Tal H, Dayan D. Gingival hyperplasia induced by calcium channel blockers: mode of action. *Med Hypothes*. 1994;43:115–8.
3. Seymour RA, Thomason JM, Ellis JS. The pathogenesis of drug-induced gingival overgrowth. *J Clin Periodontol*. 1996;23:165–75.
4. Marshall RI, Bartold PM. A clinical review of drug induced gingival overgrowths. *Aust Dental J*. 1999;44:219–32.
5. Lafzi A, Farahani, Shoja MM. Amlodipine induced gingival hyperplasia. *Med Oral Patologia Oral y Cirugia Bucal*. 2006;11:480–2.