Conservative management of Amlodipine induced gingival enlargement: A Clinical Report

Zeba Jafri[1], Nafis Ahmad[2,*]

[1,2]Assistant Professor, Faculty of Dentistry, Jamia Millia Islamia, New Delhi- 110025

***Corresponding Author:**
Email: khannafis08@gmail.com

Abstract

There are varieties of drugs which can modify the response of gingiva to plaque, clinically representing as gingival enlargement. Amlodipine is one of the drugs known to have shown gingival enlargement as its side effect though there are only a few reported cases. It is the newer generation channel blockers which are used for treatment of cardiovascular diseases like hypertension and angina. Its complementary drug Nifidepine, which also is a calcium channel blocker, has frequently shown to be associated with gingival enlargement but with amlodipine the incidence is rare. The paper presents a reported case of gingival enlargement induced by drug amlodipine in a hypertensive patient with a conservative approach in its management.

Key Words: Gingival enlargement, Amlodipine, hypertension, Calcium channel blockers

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Introduction

Drug induced gingival enlargement mainly occurs as a side effect with three main groups of drugs e.g. calcium channel blockers, immunosuppressants and anticonvulsants. Among the calcium channel blockers, Nefidipine, a first generation dihydropyridines, has shown the incidence of induced gingival enlargement as its unwanted side effect to be as high as 47.8%.[1] Amlodipine a newer agent of dihydropyridine, has also been shown to induce gingival enlargement but with lesser prevalence of upto 3.3%.[2,3]

Although, both the drugs are dihydropyridines and structurally similar but as amlodipine is more polar with pka value 8.7, the drug may not pass through cell membrane without an active transport mechanism. Thus majority of amlodipine will be tissue bound and stay inactive rather than circulating freely in the blood.[2] comparatively; Nefidipine is extremely lipophiilic and will dissolve readily within the cell membrane and pass into the cytoplasm. Amlopdipine is a 2nd generation calcium channel blockers which has shown longer action and weaker side effects compared to the first generation nifidipine.[4] It is used for the treatment of hypertention and angina. This drug was first encountered for causing gingival enlargement as side effect, by Seymour et al in 1994.[5] Many studies have reported gingival enlargement caused by amlodipine when taken 10 mg OD [5,6] over a varied period of time.

The clinical manifestations of drug induced gingival enlargement mainly presents as enlarged interdental papillae resulting in lobulated or nodular morphology.[7]

Case Report

Medical and dental history: A 52 year old patient reported to the Department of Peiodontology with a complaint of swelling gums that initiated around 5 months back and slowly progressed to an extent that it gave an unaesthetic appearance and difficulty in eating food. The patient's medical history revealed that he was hypertensive and was under medication for the same with amlodipine 10 mg OD from last 18 months.

Extra and Intra-oral examination: Patient was well built and normal in appearance. Intra orally the patient showed significant generalized gingival enlargement with nodular appearance (Fig. 1 a, b, c). The interdental papillae and attached gingiva, both facially and lingually were involved. Enlargement was seen more in the mandibular arch than the maxillary arch. The consistency was firm and the growth appeared partially fibrotic. There were moderate plaque and calculus deposits especially subgingivally. The mean plaque index recorded was 2.2 and mean gingival index 1.9. On probing bleeding was found and 4 -8mm of mainly pseudo periodontal pocket was present. There was no mobility in any of the teeth.

Blood investigations: A complete hemogram revealed blood counts within normal limits.

Diagnosis: After thorough examination and investigations, a clinical diagnosis of drug induced gingival enlargement was made.

Treatment: Patient was referred to the concerned physician for substitution of drug. Patient reported back after one week and the drug was replaced with Atenolol 50 mg/day. Thorough oral prophylaxis was done and 0.2% chlorhexidene was prescribed. Patient was educated for the side effects of the drug and role of plaque in modifying its response and motivated for keeping meticulous oral hygiene. Patient was recalled after three months for review and plan for any further surgical treatment if required. Patient reported back after three months with considerable regression of gingival enlargement (Fig. 2a). On six months follow up visit almost complete reduction in gingival enlargement was seen with no requirement for any further surgical treatment (Fig. 2b).

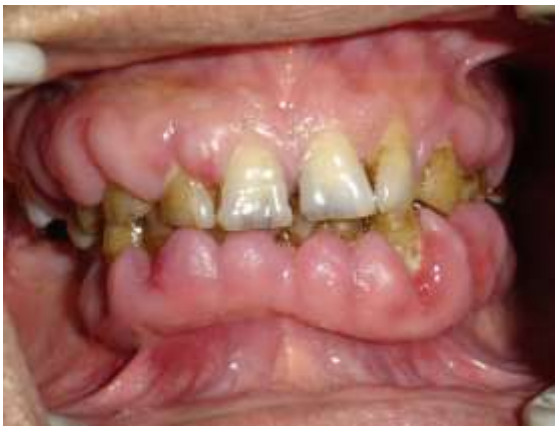

Fig. 1 a: Preoperative

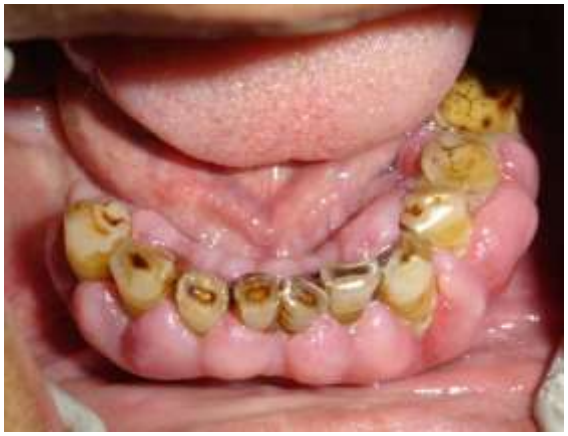

Fig. 1 b: Preoperative

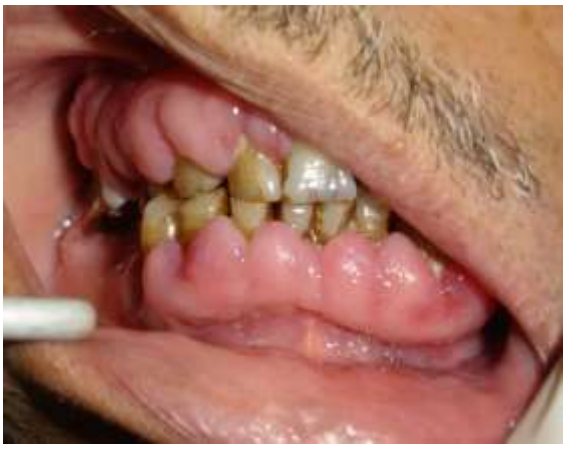

Fig. 1 c: Preoperative

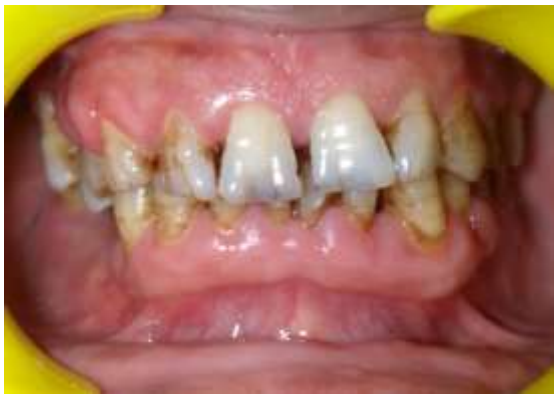

Fig. 2 a: Post-operative 3 months

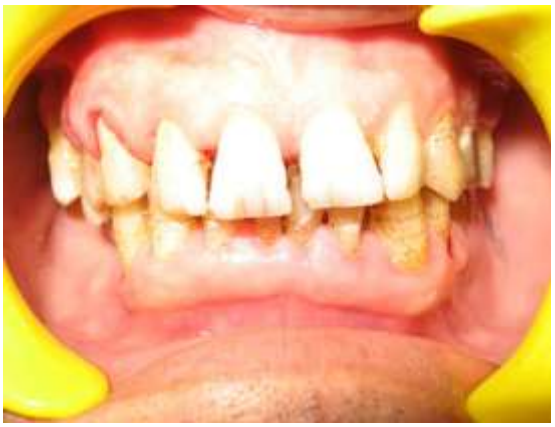

Fig. 2 b: Post-operative 6 months

Discussion

Gingival enlargement is a concern to patient both in terms of esthetics and functions. It affects the quality of life as well as creates the plaque retentive area in oral cavity which may further enhance the disease progression.

The underlying mechanism behind drug induced gingival enlargement involves inflammatory and non-inflammatory pathways. The anticipated non-inflammatory mechanisms include defective collagenase activity due to decrease uptake of folic

acid, blockage of aldosterone synthesis in adrenal cortex and consequent feedback increases in ACTH level and up regulation of keratinocyte growth factor.

During inflammatory pathway, the inflammation may develop as a result of direct toxic effects of concentrated drug in gingival fluid and bacterial plaque. This inflammation could lead to the up regulation of several cytokine factors such as TGF- β1. Individual sensitivity or host response to a drug metabolic pathway might also be a trigger. Several factors may affect the relationship between the drugs and individual gingival tissues.[8] Those factors include age, genetic predisposition, pharmacokinetic variables, alteration in gingival connective tissue, homeostasis, histopathology, ultra structural factors, inflammatory changes and drug action on growth factors. The interaction between the drug and the gingival tissues could be enhanced by gingival inflammation caused by poor oral hygiene.[9]

In our reported case, drug replacement followed by thorough scaling and root planing resulted in considerable improvement and management of gingival enlargement. This goes in accordance with the finding of Hancock R.[10]

Conclusion

A conservative approach of drug substitution and thorough scaling and root planing may regress the unwanted side effect of gingival enlargement without requiring a surgical approach. Replacement of drug and maintenance of oral hygiene must be given priority and considered as the first and the most important step in treatment approach to drug induced gingival enlargement.

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