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Case Note

Non-surgical approach in the management of amlodipine induced gingival enlargement

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Abstract

Drug-induced gingival enlargement is a well documented unwanted side effect within the literature. It has been associated with the use of three different types of pharmaceutical agents, including phenytoin, cyclosporine and calcium channel blocking agents. Amlodipine belongs to the dihydropyridine-derived calcium blocking agents that may cause the side effect of drug-induced gingival enlargement. Treatment options include meticulous plaque control, and in severe cases, gingivectomy. Gingival enlargement can be prevented with meticulous plaque control or avoidance of the offending medication.

This paper presents a rare case of amlodipine-induced gingival overgrowth in a 40-year-old male patient. The treatment aspect included Phase-1 therapy, substitution of the drug and the maintenance and supportive therapy resulting in excellent clinical outcome.

Key words: Amlodipine, Gingival enlargement, Non surgical

Introduction

An increasing number of medications are associated with gingival enlargement. Drugs associated with gingival enlargement can be broadly divided into three categories: anti-convulsants, calcium channel blockers and immune-suppressants[1]. Although the pharmacologic effect of each of these drugs is different and directed toward various primary target tissues, all of them seem to act similarly on a secondary target tissue i.e., the gingival connective tissue, causing common clinical and histopathological findings.

Calcium channel blockers are widely used in medical practice for the management of Hypertension and in the prophylaxis of Angina. Gingival overgrowth is now a recognized unwanted effect associated with many of calcium channel blockers. Of this large group of drugs, the dihydropyridines are the agents most frequently implicated[2].

The incidence of gingival overgrowth varies substantially depending on the drug category involved. Phenytoin is associated with the greatest incidence[3]. 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 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[1]. Earlier studies found prevalence of gingival overgrowth related to chronic medication with calcium channel blockers is low, with nifedipine causing the most significant gingival changes as compared to amlodipine. The prevalence with verapamil, diltiazem, felodipine, or amlodipine was significantly smaller[4].

Case report

A 40-year old hypertensive male patient presented to Department of Periodontology, Manipal College Of Dental Sciences, Mangalore with the chief complaint of enlarged gums in the upper and lower front tooth region for three months. His medical history revealed that the patient was on medication (Amlodipine 10 mg, once daily) for the past two years. There was no history of intake of any other drugs.

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On intraoral examination, marginal and interdental gingival enlargement was well appreciated covering almost coronal one-third of mandibular anterior teeth. Gingiva was red in colour with erythematous area and lobulated surface. Margins of the gingiva were rolled out with loss of normal gingival scalloping (Fig 1). On palpation, gingiva was firm and resilient in consistency. Hypertrophied areas were painless. Poor oral hygiene status of the patient was assessed from the presence of local irritating factors contributing to the inflammatory component of the gingival enlargement.The probing of gingival sulcus revealed presence of pseudo-pockets and elicited the bleeding. On the basis of the patient's history and clinical features, a clinical diagnosis of amlodipine induced gingival overgrowth was made.

Patient was subjected to phase I therapy including the planned sessions of scaling and root planing. Azithromycin was administered as an adjunct to non surgical periodontal therapy. Patient's physician was consulted regarding drug substitution or withdrawal of the drug. The physician substituted the drug with enalapril (5mg BD). Patient was instructed to maintain good oral hygiene with the use of chlorhexidine oral rinses.

A dramatic response was noticed after three weeks of drug substitution and maintenance of regular oral hygiene. There was regression in the size of gingival enlargement with minimal of fibrotic component left at the end of 6 weeks (Fig 2) The surgical therapy for remaining minimal amount of gingival enlargement was deferred because patient was not willing to go for surgery.

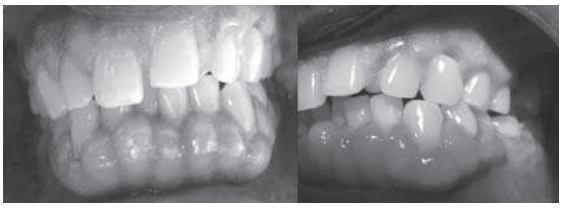

Fig 1: Before phase I therapy

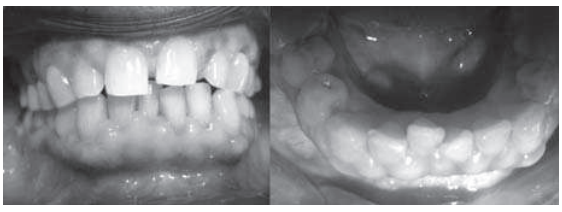

Fig 2: 6 weeks after phase I (non-surgical) therapy

Discussion

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. Usually, a three-month interval for periodontal maintenance therapy has been recommended in drug induced gingival enlargement.

The treatment options for drug-induced gingival enlargement should be based on the medication being used and the clinical presentation of the individual case. First, consideration should be given to the possibility of discontinuing or substituting the drug. Either of those scenarios should be examined in consultation with the patient's physician. Simple discontinuation of the drug is usually not a practical solution. However, its replacement with another medication is the right decision. 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, substitute drug, that is, Enalapril (ACE inhibitor) along with phase-1 therapy and antibiotic therapy resulted in clinically significant improvement in six weeks time.

The role of antibiotic therapy along with phase I therapy is to enhance the effects of mechanical debridement procedures by reducing the recurrence rate of periodontal infection. In the present case, we administered Azithromycin as an adjunct to non surgical periodontal therapy. Azithromycin is the first of a subclass of macrolides called azalides. It has a long half-life and good tissue penetration. Following a dose of 500 mg once a day for three days significant tissue levels will persist in most tissues for a week to ten days. It is preferentially taken up by phagocytes and so its level in infected tissues will be much higher than in similar non infected sites[5].

Various case reports have described the efficacy and safety of azithromycin for the partial or complete remission of drug induced gingival enlargement. The exact mechanism by which azithromycin causing remission of drug induced gingival enlargement is not clear but the recent study suggested that it acts by blocking the drug induced cell proliferation and collagen synthesis and activating MMP-2 in gingival fibroblasts which results in increased collagen degradation leading to remission of enlargement[6].

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