EFFECTIVENESS OF NON-SURGICAL PERIODONTAL THERAPY ON AMLODIPINE INDUCED GINGIVAL ENLARGEMENT: A CASE REPORT

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Abstract

Gingival enlargement, [sometimes abbreviated to GO (gingival overgrowth)] is an increase in the size of the gingiva. It is a common feature of gingival disease. Gingival enlargement is a well known side-effect of drugs like anticonvulsants, calcium channel blockers and immunosuppressant. A case of amlodipine induced gingival enlargement was reported and after drug substitution when the patient was treated non-surgically (scaling and root planing), the enlargement subsides to a normal state which suggested the effectiveness of non-surgical periodontal therapy in the treatment of drug induced gingival enlargement.

Keywords: Anticonvulsants, Immunosuppressants, Calcium channel blockers, gingival enlargement

Introduction

Drug induced gingival enlargement was first reported in 1939 associated with chronic use of antiepileptic drug phenytoin. Now, other drugs have been clearly identified as causing this lesion, immunosuppressants like cyclosporin and calcium channel blockers like nifedipine. Clinically and histologically, gingival enlargement induced by different drugs, are virtually indistinguishable. "Drug induced gingival enlargement", also sometimes abbreviated to "DIGO". Gingival enlargement may also be associated with the administration of three different classes of drugs, all producing a similar response:

- anticonvulsants (such as Phenytoin, phenobarbital, lamotrigine, valproate, vigabatrin, ethosuximide, topiramate and primidone).
- calcium channel blockers, such as nifedipine, amlodipine, and verapamil. The dihydropyridine derivative isradipine can replace nifedipine and does not induce gingival overgrowth.
- cyclosporine, an immunosuppressant.

Of all cases of DIGO, about 50% are attributed to phenytoin, 30% to cyclosporins and the remaining 10-20% to calcium channel blockers. Clinically and histologically, gingival enlargement induced by different 2 drugs, are virtually indistinguishable. Calcium channel blockers are the drugs developed for the treatment of cardiovascular conditions like hypertension, angina pectoris, coronary artery spasms, and cardiac arrhythmias. They inhibit calcium ion influx across the cell membrane of heart and smooth muscle cells, blocking intracellular mobilization of calcium. This induces direct dilation of the coronary arteries and arterioles, improving oxygen supply to the heart muscle, also reduces hypertension by dilating the peripheral 3 vasculature. These drugs are dihydropyridine derivatives (nifedipine, felodipine, amlodipine); benzothiazine derivatives (diltiazem). Many reports had discussed patients taking nifedipine induced GO. Recently, Lafzi et al. had reported rapidly developed gingival hyperplasia in patient received 10 mg per day of amlodipine within two months of onset. The clinical features of GO usually presented as enlarged interdental papillae and resulting in a lobulated or nodular morphology (Hallmon and Rossmann). Histologically, in nifedipine-induced gingival overgrowth it was described as thickening of the spinous cell layer, slight to moderate hyperkeratosis, fibroblastic proliferation and fibrosis of lamina propria. In this case report, we treated severe GO in patient taking amlodipine for treatment of hypertension. The management consists of oral hygiene procedures and drug substitution.

The most probable mechanisms of drug induced gingival enlargement are described in fig 1.
CASE REPORT

A 38 year-old female patient reported to the department of periodontology at DJ Dental College, Modinagar, U.P, with the chief complaint of swollen and bleeding gums since the last 5 months. The past medical history was remarkable for diagnosed hypertension for which the patient was taking amlodipine (5mg/ day) and metoprolol (100mg/day, in divided dose orally) from past 9 months. After taking the drug for about 3 months, the patient had noticed small, painless enlargement of gingiva that gradually increased. Within two months the enlargement attained a massive size and patient found difficulty in mastication, speech, maintenance of oral hygiene and had aesthetic concerns (Fig 2). The patient complained of foul odour from oral cavity and bleeding gums on brushing.

The oral examination of the patient revealed a generalized, and firm gingival enlargement throughout the maxillary and mandibular arch, particularly severe in maxillary anterior region. The diffuse enlargement was pale pink, firm and resilient, with irregular contours covering the portions of crowns. At some sites the marginal gingiva in maxilla was bluish red, soft and shiny, with bleeding on provocation. Gingival clefts and suppuration was also present. Periodontal pockets were present in all the upper teeth and lower anterior teeth along with subgingival plaque and calculus that caused secondary inflammation at some sites.

LEGENDS

Fig. 1: Schematic diagram to illustrate the potential multifactorial features and interactions involved in the pathogenesis of Drug- induced gingival overgrowth

Fig. 2: Pre operative - Showing Amlodipine induced gingival enlargement

Fig. 3: Post operative - After 1 month of Non surgical periodontal therapy and drug

MANAGEMENT

The first line management of gingival overgrowth is improved oral hygiene, ensuring that the irritative plaque is removed from around the necks of the teeth and gums. Situations in which the chronic inflammatory gingival enlargement include significant fibrotic components that do not respond to and undergo shrinkage when exposed to scaling and root planing are treated with surgical removal of the excess tissue, most often with a procedure known as gingivectomy.

In DIGO, improved oral hygiene and plaque control is very important to help reduce any inflammatory component that may be contributing to the overgrowth. Reversing and preventing gingival enlargement caused by drugs is as easy as ceasing drug therapy or substituting to another drug. However, this is not always an option; in such a
situation, alternative drug therapy may be employed, if possible, to avoid this deleterious side effect.

In the present case, the drug Amlodipine was substituted by dihydropyridine derivative Radipidine for some uses of calcium channel blocking which does not induce gingival overgrowth and patient was undergone non-surgical periodontal therapy i.e. scaling and root planing. After a month of drug substitution and non-surgical periodontal therapy, when patient was recalled, then a significant and noticeable amount of reduction in gingival enlargement was observed. This showed the effectiveness of non-surgical periodontal therapy and the importance of substitution of drug (Fig 3).

DISCUSSION

Drug induced gingival enlargement is a serious concern for both the patient and the clinician. Calcium channel blockers are considered potential etiologic agents for drug induced gingival enlargement. The incidence of nifedipine induced gingival enlargement is about 20% of the patients taking this drug. The other drugs of same class dihydropyridine derivatives like amlodipine, felodipine, nicardipine also cause gingival enlargement.

Taib et al[4] stated that the interaction between the drug and the gingival tissues could be enhanced by gingival inflammation caused by poor oral hygiene. It has been shown by Hallmon and Rossman[6] that there was significant reduction of nifedipine-induced GO by thorough scaling and root planing and scrupulous plaque control. Discontinuation of the related drug has also been shown to reduce the GO, however the growth will recur when the drug was readministered[2]. Isradipine, a companion dihydropyridine calcium channel blocker has shown regression about 60% of the GO previously induced by nifedipine (Hallmon and Rossman[6]). However, Taib et al[4] reported good results of drug induced gingival enlargement after surgical therapy (gingivectomy) done by laser.

The present case report showed that substitution/replacement of drug with good nonsurgical periodontal therapy (scaling and root planing) alone is very effective to treat the massive drug induced gingival enlargement (Fig 3).

REFERENCES

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