AMLODIPINE INDUCED GINGIVAL HYPERPLASIA

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ABSTRACT

Amlodipine is a calcium channel blocker. Because of its long acting effect it is being widely used as antihypertensive drug. Gingival hyperplasia is one of its side effects. The histological and clinical evidences were consistent with amlodipine-induced gingival hyperplasia. The aim of publishing this report is to draw the attention of dentists towards this adverse effect.

Key words: Amlodipine, Drug induced Gingival hyperplasia

INTRODUCTION

Drugs associated with gingival enlargement can be broadly divided into three categories: anticonvulsants, calcium channel blockers and immunosuppressants. Although pharmacologic effect of each of these drugs is different and directed towards various primary target tissues, all of them seem to act similarly on secondary target tissue, i.e, the gingival connective tissue, causing common clinical histo-pathological findings. Many studies show high prevalence of nifedipine (short acting beta blocker) induced gingival hyperplasia, but amlodipine has rarely been reported as the potential etiologic cause of gingival hyperplasia.

CASE REPORT

A 41 year old male was referred to the Department of Orthodontic, Istanbul University. The patient complained of extensive gingival enlargement along with foul odor, bleeding and fetid discharge from gums. He also complained of continuous mild pain and a feeling of heaviness in both upper and lower jaws. On general examination, the patient was medium built and was suffering from hypertension for the last one year and was under amlodipine therapy. ECG and blood investigations were performed and were found within normal limits. Lips were incompetent, hence patient had a mouth breathing problem. Intraoral examination revealed that all the teeth were present except left mandibular 2nd and 3rd molars. Left mandibular 1st molar and right mandibular 2nd and 3rd molars were carious. Cervical abrasion was present on all the teeth. A generalized nodular enlargement of the marginal and interdental gingiva on both facial and lingual aspects was noticed which was bright red in colour and fibrotic in consistency (Figure 1). At isolated places, particularly in anterior region, inflammatory changes were seen. Probing depth of gingival sulcus ranged from 3mm to 6mm. Anterior teeth were in labioversion and lower anterior showed diastamas. Prior to local management, the patient was thoroughly assessed by a physician and a suitable premedication therapy was instituted. Under local anaesthesia, the enlargement was removed segment wise by a modified flap surgical procedure. Restoration of carious teeth and replace-
ment of missing teeth was done. There were no postop-
erative complications and the healing was uneventful. The patient was followed up for a period of one year regularly. A marginal inflammatory recurrence, however, was noticed which was easily managed by routine therapeutic procedures.

DISCUSSION

Calcium channel blockers especially dihydropy-
ridine group produce gingival hyperplasia. Approximately 10% of patients taking nifedipine develop clinically significant gingival hyperplasia. It has potential cosmetic implications and provides new niches for the growth of microorganisms and is of serious concern for both the patients and the clinician. Calcium channel blockers are one of the most commonly used drugs for the management of cardiovascular disorders and are known for causing gingival enlargement as side effects. Although the incidence of nifedipine-induced gingival hyperplasia is about 10%, very few reports of amlodipine-related gingival hyperplasia exist in the literature. In the present case the lack of inflammatory component parallels the absence of vertical gingival growth and consequently the lack of periodontal pockets or pseudopockets. We hypothesize that the formation of pocket/pseudopocket assembly is a phenomenon that is associated with gingival inflammation. Among the old and relatively new pharmacologic agents involved in gingival enlargement, overall, phenytoin still has the highest prevalence rate (approximately 48%), with calcium channel blockers and cyclosporine associated enlargements about half less. Current studies on the pathogenetic mechanism of drug associated enlargement are focusing on the direct and indirect effects of these drugs on gingival fibroblast metabolism. Treatment is generally targeted on drug substitution and effective control of local inflammatory factors such as plaque and calculus. When these measures fail to cause resolution of the enlargement, surgical intervention is required. These treatment modalities, although effective, do not necessarily prevent recurrence of the lesions. Newer molecular approaches are needed to clearly establish the pathogenesis of gingival enlargements and to provide novel information for future preventative and therapeutic modalities.

DIAGNOSTIC & TREATMENT

The diagnosis of drug-induced gingival enlargement is based on the clinical appearance of the gingivae and specially the medical history.

The best approach for controlling drug induced gingival enlargements is by maintaining good oral hygiene and preventing plaque formation and regular professional care.

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REFERENCES