Nonsurgical Management of Gingival Enlargement in an Elderly patient with Cardiac disease: Case Report

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Abstract
In older individuals, antihypertensive drugs in the calcium channel blocker group are widely used. Amlodipine is a calcium channel blocker used in the treatment of hypertension and angina. Although amlodipine is considered as a safe drug, very rarely it may induce gingival overgrowth also. Here we present a case of amlodipine-induced gingival overgrowth in a 64-year-old male patient who had undergone cardiac surgery and was on medication. Complete resolution of the enlarged gingival mass was achieved without any invasive procedure.The treatment included Phase-1 therapy followed by supportive periodontal therapy resulting in good and acceptable clinical outcome.

Keywords: Cardiovascular ailment, anti-hypertensive treatment, drug induced gingival enlargement, nonsurgical therapy.

INTRODUCTION

A common trait of gingival disease is gingival enlargement or gingival overgrowth which characterized by an increased gingival size. Gingival overgrowth is predominantly an inflammatory reaction to the presence of plaque, which is modified by the presence of systemic disease or use of medicines. Sometimes genetic conditions though rare can cause overgrowth of gingiva without the presence of plaque. Use of immunosuppresing drugs, calcium channel blockers and some anti-convulsants can cause gingival enlargement and create problems in esthetics, mastication, eruption of teeth and speech in susceptible patients.[1]

In medical practice, use of calcium channel blockers is widespread in the management of CVS disorders. Gingival overgrowth is a recognized adverse effect associated with many these drugs. Dihydropyridines belonging to this large group of drugs are frequently implicated in the cause of enlargement.[2] of which amlodipine, used mainly for treatment of hypertension and angina, was the first of this class reported in 1994 by Seymour et al to be causative for enlargement.[3] This is a case report of a patient presenting with amlodipine-induced gingival overgrowth who had undergone cardiac surgery and was on medication. The treatment was carried out in the outpatient department with Phase-1 therapy followed by supportive periodontal therapy.

Case report:

A 64-year-old male patient reported to a Department of Periodontology in Uttar Pradesh with a chief complaint of swelling and redness with bleeding from the upper and lower front teeth regions since 3 months. No significant findings extra orally were noted. On thorough clinical intraoral examination, generalized gingival enlargement of marginal and interdental gingiva extending up to attached gingiva with diffuse erythematous bead like nodular growth was observed in the maxillary and right mandibular anterior teeth with poor oral hygiene maintenance (Figure 1). Gingiva in the affected area was fiery red and also bled on probing. Ortho-pantomogram revealed no significant changes except for a moderate generalized bone loss. The medical history revealed that the patient was hypertensive and had a cardiovascular disease for which he had undergone surgery 5 years back and was medicated with 5mg amlodipine since. The patient did not report any deleterious habits or drug allergies and his vital signs were normal.

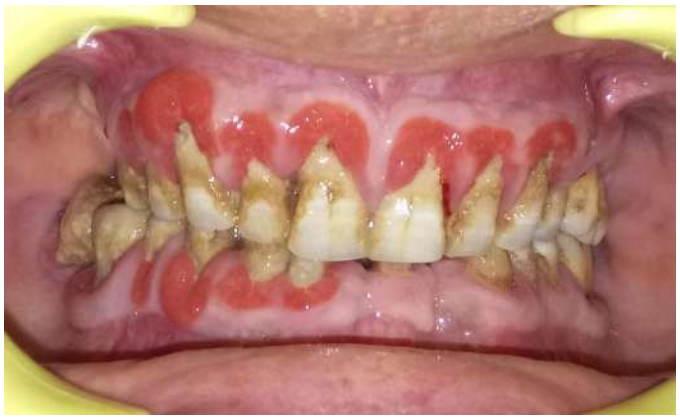

Figure 1: Pre-operative view showing gingival enlargement

Correlating medical history, the clinical presentation of gingival enlargement and a history of prolonged usage of

amlodipine, the case was diagnosed as combined gingival enlargement. In the preliminary phase, oral prophylaxis under local anesthesia was performed, followed by reinforced oral hygiene instructions and was prescribed with a twice daily mouthwash of 0.2% chlorohexidine. Follow up recall was after 21 days, 1 and 3 months. After 1 month, there was definite resolution of the gingival enlargement and marked improvement in the color and texture of the gingiva (Figure 2). Further scaling and root planing was done with reinforcement of oral hygiene instructions. After 3 months, complete resolution of enlargement was noted with an appreciable reduction of inflammation (Figure 3). Patient was satisfied and comfortable with the final outcome.

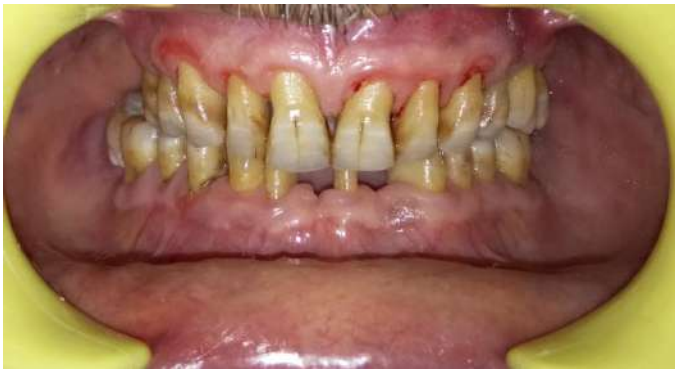

Figure 2: Post-operative view showing response to non-surgical periodontal therapy after 1 month.

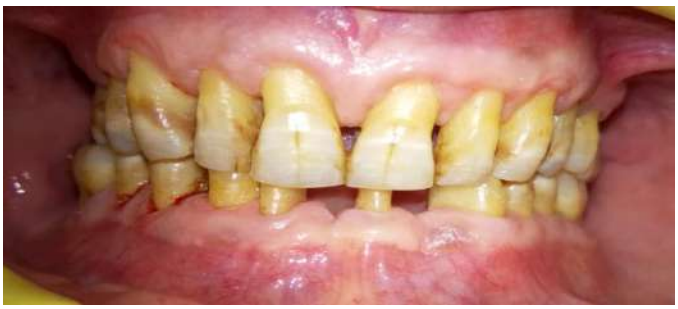

Figure 3: Post-operative view showing improved response to therapy after 3 months

Discussion:

Lederman in 1984 first described gingival overgrowth associated with calcium channel blockers in patients treated with nifedipine. The prevalence of gingival overgrowth due to the use of calcium channel blockers such as diltiazem, verapamil, amlodipine is reported as 74%, 21%, 3.3%, respectively.[4] Amlodipine is a 3rd generation dihydropyridine calcium channel blocker which is structurally similar to nifedipine and is of frequent use in the conservative management of hypertension and angina. The first reported gingival overgrowth due amlodipine was by Ellis et al. in 1993,[5]. Since then, very few reports regarding the prevalence of AAGE have appeared in studies as compared to other calcium channel blockers including nifedipine.[6] Clinical manifestations of AAGE usually happen within 1 to 3 months after start of treatment with a therapeutic dose of 10 mg/day and normally starts as interdental papilla enlargement mainly in the anterior segment of labial surface. Seymour et al[2] reviewed the pathophysiology of drug-induced gingival overgrowth and considered it to follow a multi-factorial model, with involvement of several factors, including the interaction between the drug and its metabolites with gingival fibroblasts.

The pathogenesis of the enlargement is undetermined, and the treatment for the same is limited to oral prophylaxis, reinforcement and maintenance of an adequate level of oral hygiene and surgical removal of the overgrown tissue.[7]Although, both inflammatory and non-inflammatory pathways are considered as the underlying mechanism behind drug-induced gingival enlargement, the non-inflammatory mechanisms are proposed to include defects in the collagenase activity due to reduced uptake of folic acid; increase in adrenocorticotropic feedback due to blockage of aldosterone synthesis in the adrenal cortex and enhancement of keratinocyte growth factor. An alternate hypothesis suggests that the inflammation may a direct result of the toxicity of the concentrated drug in GCF with or without the presence of bacteria. This inflammation could enhance several cytokines and signalling molecules i.e. transforming growth factor-β1.[8]

Various studies done on amlodipine suggest that effect of amlodipine on gingival hyperplasia is not appreciable at 5 mg/ day dose even when taken for more than 6 months but studies do indicate a dose of 10mg/day can be a cause of appreciable hyperplasia.[9] The present case was a unique presentation as hyperplasia occurred at a low dose of amlodipine i.e.5 mg/day and appeared after prolonged usage of 5 years. The mechanism by which these drugs act on the gingiva to induce enlargement is poorly understood but it has been suggested that phenytoin and calcium channel blockers may interfere with the intracellular Ca^{2+} uptake hence stimulating the gingival fibroblasts and causing increased collagen production. The percentage of patients developing enlargement is low even though receiving the same drug and dosage indicating that patient susceptibility to the drug may also be a probable reason.[8]

Conclusion:

In present case, no invasive surgery was performed considering the age and medical condition of the patient and was subjected to phase 1 and supportive periodontal therapy. Patient followed up after 1 month showed marked resolution of the gingival enlargement along with the inflammatory component of gingiva. Three months follow up showed a clinically appreciable healthy gingiva indicating a successful treatment outcome. Overall the patient was very happy and satisfied with the final outcome.

References:

1. Sam G, Sebastian SC. Nonsurgical Management of Nifedipine Induced Gingival Overgrowth. Case Reports Dent 2014; 2014:741402.
2. Seymour RA, Thomason JA, Ellis JS. The pathogenesis of drug-induced gingival overgrowth. J Clin Periodontol 1996; 23:165-75.
3. Seymour RA"Calcium channel blockers and gingival enlargements." Br Dent J 1991; 170:376-379.
4. Gopal S, Joseph R, Santhosh VC, Kumar VV, Joseph S, Shete AR. Prevalence of gingival overgrowth induced by antihypertensive drugs: A hospital-based study. J Indian Soc Periodontol 2015;19.
5. Ellis JS, Seymour RA, Thomason JM, Monkman SC, Idle JR. Gingival sequestration of amlodipine and amlodipine-induced gingival overgrowth. Lancet 1993; 341(8852):11023.
6. Jorgensen MG. Prevalence of amlodipine-related gingival hyperplasia. J Periodontol 1997;68:676-8.
7. Saleem M, Deepa D. Management of amlodipineinduced gingival enlargement by diode laser. J Curr Res Sci Med 2016;2:49-52.
8. Raghavendra MH, Shreehari AK, Srivastava D. Non-Surgical Management of a Case of Amlodipine-Induced Gingival Enlargement. J Evolution of Med Dent Sciences 2014;3:9685-9689.
9. Triveni MG, Rudrakshi C, Mehta DS. Amlodipine-induced gingival overgrowth. J Indian Soc Periodontol 2009;13:160-3.

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