Scholars Journal of Applied Medical Sciences (SJAMS) **ISSN 2320-6691 (Online)**
Sch. J. App. Med. Sci., 2016; 4(3F):1029-1031 **ISSN 2347-954X (Print)**

(An International Publisher for Academic and Scientific Resources)
www.saspublisher.com

Case Report

Amlodipine induced gingival enlargement managed by a combined approach

Dr Khan Rafya N[1], Dr. Muzafar Ahmad Bhat[2], Dr. Suhail Majid Jan[3], Dr Mudasir Ahmad Bhat[4]
[1,2]PG 3rd year, Dept. Of Periodontics, Govt. Dental College, Srinagar
[3]Professor and head of Dept. Of Periodontics, Govt. Dental College, Srinagar
[4]PG Student , SKIMS medical college, Bemina, Srinagar

***Corresponding author**
Muzafar Ahmad Bhat
Email: muzafarbhat.nit@gmail.com

Abstract: Gingival overgrowth represents an over-exuberant response to a variety of local and systemic conditions. Certain anticonvulsants, immuno-suppressive drugs and a number of calcium channel blockers have been shown to produce similar gingival overgrowths in certain susceptible patients. Amlodipine, a third generation calcium channel blocker, used mostly as an anti-hypertensive drug has been shown to promote gingival overgrowth, although reported in very limited cases. A rare case of amlodipine-induced gingival overgrowth has been reported herein in a 55-year-old male patient. The treatment aspect included Phase-1 therapy, substitution of the drug, the surgical excision and the maintenance and supportive therapy resulting in excellent clinical outcome.
Keywords: Calcium channel blocker, Amlodipine, Surgical excision

INTRODUCTION:

Gingival overgrowth is one of the most important clinical features of gingival pathology. It has multifactorial etiologies and has been frequently associated with inflammatory changes in the gingiva. Other factors related to this condition are hereditary (familial), malignancies, hormonal, conditional, and those resulting from adverse effects associated with systemic administration of certain drugs like phenytoin, cyclosporine and calcium channel blocking agents [1]. Amlodipine, a dihydropyridine derivative, is a third generation of calcium channel blockers, which is shown to have longer action and weaker side effect compared to the first generation like nifedipine [2]. The prevalence of gingival overgrowth in patients taking amlodipine was reported to be 3.3%, [3] which is lower than the rate in patients taking nifedipine, 47.8% [4]. During the past few years, amlodipine has been used with increasing frequency and also has been reported to promote gingival overgrowth [5].

CASE HISTORY:

A 55-year-old male patient came to the Department of Periodontics, Govt. Dental College and Hospital, Srinagar with the chief complaint of enlarged gums in both upper and lower jaws. The extensive gingival swelling was associated with a foul odour, bleeding and fetid discharge from gums. The enlargement started with small bead-like nodular enlargement of the gums that gradually progressed to the present size. Medical history revealed that the patient was taking treatment for hypertension and was on Amlodipine 10 mg, once a day for the past 2 years. There was no history of intake of any other drugs. The patient gave a history of progressive gingival enlargement since the commencement of amlodipine therapy. However, his past dental history was non-contributory. His personal history revealed that he used to clean his teeth once daily with brush and paste, which he discontinued recently because of the coverage of teeth with the enlarged gums. His general physical examination revealed that the patient was moderately built and his vital signs were within the normal range. There were no significant extra-oral findings. On intra-oral examination, a generalized and firm overgrowth of the gingiva was found throughout the maxilla and mandible particularly at the buccal side. Marginal and interdental gingival enlargement was well-appreciated covering almost cervical one-third of maxillary and mandibular anterior teeth. Gingiva was pink in colour with erythematous area at some places and lobulated surface. Margins of the gingiva were rolled out with the loss of normal gingival scalloping.[figure 1. Pre-operative view]

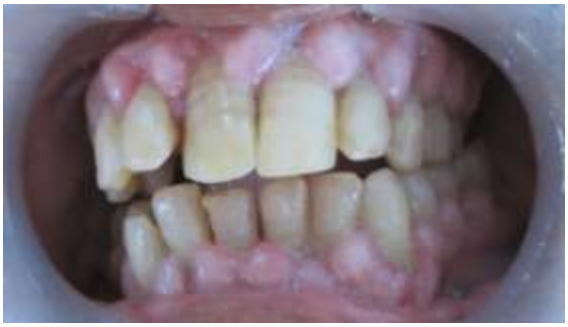

Fig 1: pre-operative view

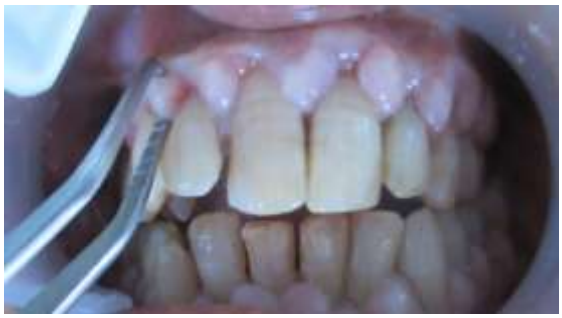

Fig 2: Bleeding points induced by pocket marker

Hypertrophied areas were painless. On palpation, gingiva was firm and resilient in consistency. Poor oral hygiene status of the patient was assessed from the presence of local irritating factors contributing to the mild inflammatory component of the gingival enlargement. The probing of gingival sulcus revealed presence of pseudo-pockets and elicited the bleeding.[figure 2. Bleeding points induced by pocket marker]. On the basis of the patient's history and clinical features, a clinical diagnosis of amlodipine-induced gingival overgrowth combined with inflammatory enlargement was made. Patient was subjected to complete haemogram and all the parameters were found to be within normal range. Orthopantomogram revealed complete set of dentition with generalized moderate bone loss.

The treatment of the patient was started with nonsurgical approach. Patient was subjected to Phase-1 therapy including the planned sessions of scaling and root planning. Patient's physician was consulted regarding drug substitution or withdrawal of the drug. The physician substituted the drug with tab. Norma date 100 mg (Labetalol). Patient was instructed to maintain good oral hygiene with the use of chlorhexidine oral rinses. A little response was noticed after 4 weeks of drug substitution and maintenance of regular oral hygiene. Although there was regression in the size of gingival enlargement but the fibrotic component was still left . Finally, surgical excision of gingival hyperplastic tissue was planned employing the techniques of gingivectomy/gingivoplasty to restore the normal shape and contour of the gingiva [Figure 3. Gingivectomy done by Kirkland knife].

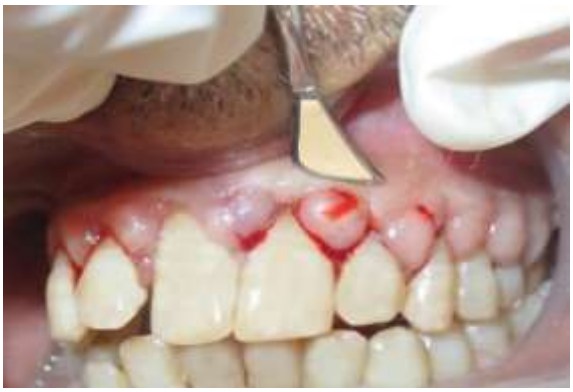

Fig 3: Gingivectomy done by Kirkland knife

Postoperatively, there was successful elimination of enlarged gingival tissue [figure 4. Immediate post operative view] and restoration of a physiological gingival contour giving the patient an esthetically pleasing appearance [figure 5. 3 month Post -operative view]

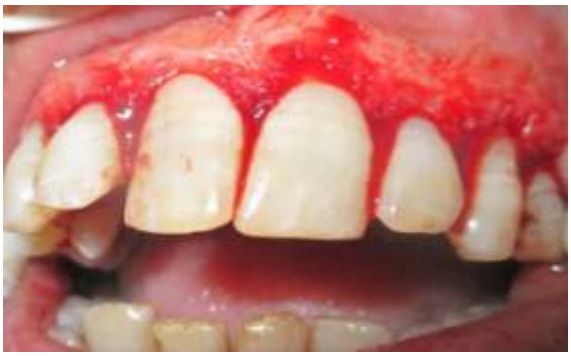

Fig 4: Immediate post operative view

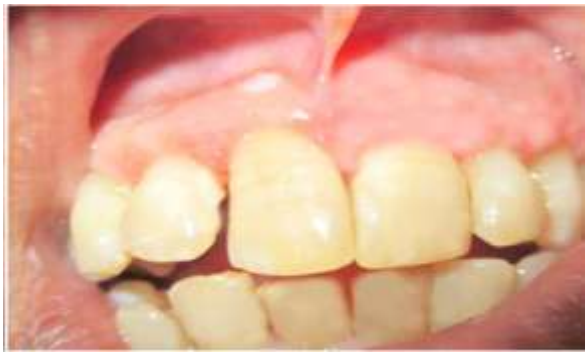

Fig 5: 3 month Post -operative view]

DISCUSSION:

Gingival hyperplasia with its potential cosmetic implications and also providing new niches for the growth of microorganisms is a serious concern for both patients and clinician. Calcium channel blockers are considered as potential etiologic agents of drug-induced gingival hyperplasia. Although the incidence of nifedipine-induced gingival hyperplasia is high, very few reports of amlodipine-related gingival

hyperplasia exist in the available literature [6, 7, 8]. Lafzi *et al.;* (2006) had reported rapidly developing gingival hyperplasia in patient receiving 10 mg/day of amlodipine within 2 month of onset [9]. The prevalence of amlodipine-induced gingival overgrowth was reported to be 3.3% (Jogersen, 1997). The underlying mechanism of gingival enlargement still remains to be fully understood. However, two main inflammatory and non-inflammatory pathways have already been suggested. The proposed non-inflammatory mechanisms include defective collagenase activity due to decreased uptake of folic acid, [10] blockage of aldosterone synthesis in adrenal cortex and consequent feedback increase in ACTH level [11] and upregulation of keratinocyte growth factor [12]. Alternatively, inflammation may develop as a result of direct toxic effects of concentrated drug in crevicular gingival fluid and/or bacterial plaques [13]. This inflammation could lead to the upregulation of several cytokine factors such as TGF-β1 [14].

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. In this present case, the local environmental factors such as poor plaque control at the initial presentation may act as risk factors that had contributed to worsen the existing gingival enlargement and therefore complicate the oral hygiene procedures. There was some reduction of the overgrowth after the initial Phase I therapy was advocated. Discontinuation of the related drug has been shown to reduce the gingival overgrowth. However, the growth recurs when the drug is re-administered [15].

In this present case, gingival overgrowth was satisfactorily treated through initial periodontal therapy including oral hygiene instruction and motivation, and drug substitution. However, due to incomplete resolution of the enlargement external bevel gingivectomy was carried out. Patient was recalled every month until first 3 months and then at the end of 6 months. The postoperative results were found to be extremely satisfactory both aesthetically and functional

REFERENCES:

1. Bhatia V, Mittal A, Parida AK, Talwar R, Kaul U; Amlodipine induced gingival hyperplasia: A rare entity. Int J Cardiol 2007; 122:23-4.
2. Ellis JS, Seymour RA, Thomason JM, Monkman SC, Idle JR; Gingival sequestration of amlodipine and amlodipine-induced gingival overgrowth. Lancet 1993; 341:1102-3.
3. Jorgensen MG; Prevalence of amlodipine-related gingival hyperplasia. J Periodontol 1997;68:676-8
4. Nery EB, Edson RG, Lee KK, Pruthi VK, Watson J; Prevalence of nifedipine-induced gingival hyperplasia. J Periodontol 1995; 66:572-8.
5. Seymour RA, Ellis JS, Thomason JM, Monkman S, Idle JR; Amlodipine-induced gingival overgrowth. J ClinPeriodontol1994; 21:2813.
6. Grover V, Kapoor AA, Marya CM; Amlodipine induced gingival hyperplasia. J Oral Health Community Dent 2007; 1:19-22.
7. Banthia R, Jain P, Banthia P, Belludi S, Jain AK; Amlodipine- induced gingival overgrowth: A case report. J Mich Dent Assoc 2012; 94:48-51.
8. Srivastava AK, Kundu D, Bandyopadhyay P, Pal AK; Management of amlodipine-induced gingival enlargement: Series of three cases. J Indian Soc Periodontol 2010;14:279-81
9. Lafzi A, Farahani RM, Shoja MA; Amlodipine induced gingival hyperplasia. Med Oral Patol Oral Cir Bucal 2006; 11:E480-2.
10. Brown RS, Sein P, Corio R, Bottomley WK; Nitrendipine-induced gingival hyperplasia. First case report. Oral Surg Oral Med Oral Pathol 1990; 70:593-6.
11. Nyska A, Shemesh M, Tal H, Dayan D; Gingival hyperplasia induced by calcium-channel blockers: Mode of action. Med Hypotheses 1994; 43:115-8.
12. Das SJ, Olsen I; Keratinocyte growth factor is upregulated by hyperplasia-inducing drug nifedipine. Cytokine 2000; 12:1566-9.
13. Van der Vleuten CJ, Trijbels-Smeulders MA, van de Kerkhof PC; Telang ectasia and gingival hyperplasia as side-effects of amlodipine (Norvasc) in a 3-year-old girl. Acta Derm Venereol 1999; 79:323-4.
14. Border WA, Noble NA; transforming growth factor beta in tissue fibrosis. N Engl J Med 1994; 331:1286-92.
15. Lederman D, Lumerman H, Reuben S, Freedman PD; Gingival hyperplasia associated with nifedipine therapy. Report of a case. Oral Surg Oral Med Oral Pathol 1984; 57:620-2.