

CASE REPORT



Drug-induced gingival enlargement in a hypertensive patient with chronic smoking behavior – A case report

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Abstract

Drug-induced gingival enlargement is often observed as a side effect of some pharmacologic agents which might have been prescribed for non-dental reasons, for which the gingival tissue is not the intended target. These medications mainly include antihypertensives, anticonvulsants, and immunosuppressants class of drugs. Here, we describe a case of amlodipine-induced massive gingival enlargement in a 40-year-old male patient with chronic smoking habit. The patient was advised regarding the gingival enlargement and its probable cause. His family physician was consulted, who substituted an angiotensin receptor blocker for the amlodipine. He was further counseled for tobacco cessation, instructed on good oral hygiene techniques, and advised for a surgical periodontal treatment.

Introduction

Seymour *et al.*, in 1994, were the first to report gingival enlargement associated with amlodipine, a long-acting calcium channel blocker, used as an anti-hypertensive and anti-anginal drug. They studied various factors which impacted the relationship between drugs and gingival tissues. These include age, genetic predisposition, alteration in gingival connective tissue homeostasis, ultra-structural factors, inflammatory changes, and the action of drug on growth factors.^[1] Here, we present a case of amlodipine-induced massive gingival enlargement in a patient with chronic smoking habit.

Clinical Presentation

A 40-year-old male patient had reported to the department of oral medicine and radiology of the dental college and hospital with the chief complaint of swollen gums and missing teeth in the upper and lower jaws for the past 1 year. His medical history revealed that he was hypertensive and was prescribed 10 mg amlodipine daily for the past 2 years. Drug history also revealed that he was on medications for anxiety approximately 2 years ago which included

a 4-month course of alprazolam 0.5 mg daily. He was a chronic smoker (smoking for past 15 years about 5–6 cigarettes/day) and had quit the habit 6 months ago on his physician's advice.

On general examination, the patient was moderately built and nourished with no significant extraoral changes [Figure 1]. Intraoral examination of the soft tissues on inspection revealed diffuse generalized bulbous gingival enlargement of both the upper and the lower jaws involving marginal gingiva, attached gingiva and the interdental papilla [Figure 2]. The surface of gingival enlargement appeared lobulated and erythematous with loss of scalloping. The presence of deep periodontal pockets in relation to almost all the teeth was observed with very minimal bleeding on probing. On palpation, inspeitory findings were confirmed. The gingiva was soft and fibrous in consistency, non-tender without any suppuration. Hard tissue examination revealed spacing between displaced maxillary and mandibular anterior teeth. Teeth were mobile with thick deposits of calculus and tobacco stains on their buccal/facial and lingual/palatal surfaces. Smoker's melanosis of the palate and the lips were also observed. Based on the patient's medical history and clinical features, a clinical diagnosis of amlodipine-induced gingival enlargement was made.

Panoramic radiography revealed the missing posterior teeth. There was generalized bone loss till apical one-third of the teeth and poorly defined (lost) lamina dura. Ill-defined radiolucencies were observed in the periapical areas in relation to the left mandibular first pre-molar, right mandibular canine, and first molar suggesting perio-endo lesion [Figure 3]. A radiographic diagnosis of chronic generalized periodontitis was made.

Case Management

The patient was informed about the gingival enlargement and the effects of the medication that he had been taking for 2 years. Patient's physician was consulted regarding drug substitution, who consequently substituted an angiotensin receptor blocker for the amlodipine and also advised diet control along with physical exercise. The patient was subjected to planned sessions



Figure 1: Extra oral pictures showing facial profile (frontal and lateral)

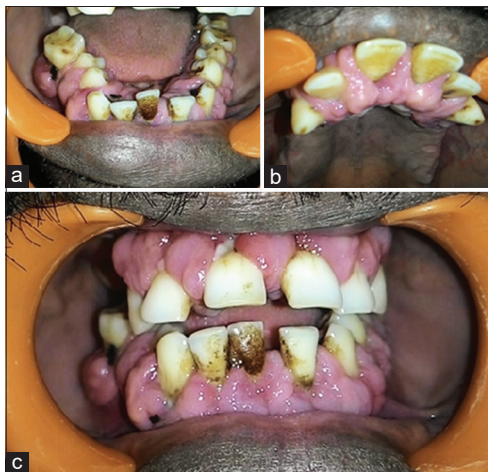


Figure 2: Intraoral picture showing diffuse generalized bulbous gingival enlargement of both the upper and the lower jaws involving marginal gingiva, attached gingiva, and the interdental papilla. (a) Mandibular arch; (b) maxillary arch; and (c) both the arches

of deep periodontal scaling and root planning. This was followed by counseling session to motivate the patient to sustain tobacco cessation and instructions to maintain a good oral hygiene with the use of chlorhexidine oral rinses. Importance of mental well-being was also emphasized on. Before being referred for prosthesis treatment for replacing the missing teeth, he was also advised on surgical periodontal treatment for esthetic reasons and to solve the functional problems associated with the mild gingival enlargement which still persisted. However, the patient was not willing to undergo any surgical intervention.

Discussion

Literature suggests that more than 20 prescription medications are closely associated with gingival hypertrophy, with antihypertensives, anticonvulsants, and immunosuppressants topping the list.^[2] The prevalence of amlodipine-induced gingival hypertrophy is between 1.7% and 3.3% and seen more commonly in men than women in the ratio of 3.3:1.^[2,3]

Our patient was on anti-anxiety drug of alprazolam for a period of 4 months. However, alprazolam is not known to cause gingival enlargement as a side effect.^[4] The main risk factor which is responsible for the expression of drug-induced gingival overgrowth is poor oral hygiene.^[5] Thus, we concluded that the gingival enlargement was mainly amlodipine-induced and was further exacerbated by poor oral hygiene status of the patient. Poor oral hygiene status results in secondary inflammatory changes which add to the size of the lesion caused by the drug thereby obliterating the lobulated surface demarcations, causing bluish red discoloration and increased bleeding tendency.^[6] All these changes were observed in our patient except for the increased bleeding tendency. Bleeding on probing of drug-induced gingival enlargements has been elicited in many case studies.^[5,7,8] Minimal bleeding on probing in our patient could possibly be due to the chronic smoking behavior of the patient. Evidence suggests that prolonged exposure to smoking exerts a diminishing effect on bleeding

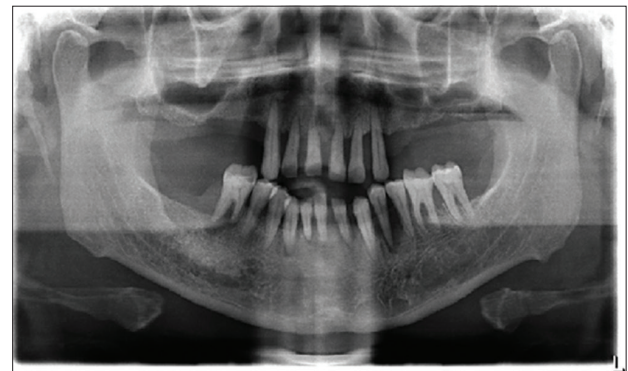


Figure 3: Orthopantomograph showing generalized bone loss till apical one-third of the teeth and with poorly defined (lost) lamina dura

on probing and this could be attributed to the vasoconstrictive nature of nicotine.^[9]

Management of drug-induced gingival hypertrophy includes withdrawal or substitution of the drug under physician's supervision. This has to be supplemented with a meticulous plaque control treatment. Excision of excessive gingival tissue, that is, gingivectomy is generally reserved for severe cases where oral functions are compromised or may be performed to restore esthetics.^[7] Evidence suggests that the gingival lesions might take 1–8 weeks for complete resolution, post-substitution of the offending drug.^[10] Unfortunately, not all patients respond to this mode of treatment as observed in this case where mild gingival enlargement persisted even after drug substitution and effective plaque control techniques.

The significance of this study is that it reports a case of drug-induced gingival enlargement in a patient who exhibited chronic smoking behavior and was also subjected to multiple drug therapies. Hence, the foremost step in the treatment plan was to identify the causative drug for gingival enlargement; in this case, it being amlodipine and not alprazolam. This case elicited almost all the typical clinical features of drug-induced gingival enlargement except for the minimal bleeding on probing which could be attributed to the prolonged exposure to tobacco smoking. Counseling of the patient was crucial in this case considering his mental status, chronic smoking behavior, poor oral hygiene status, and his medical drug history.

Limitations of the study include loss of eventual follow-up and reporting of the progress of the gingival condition due to lack of patient compliance.

Conclusion

An oral physician might be the first point of contact in such cases of drug-induced gingival enlargements. The patient should be informed about this condition, motivated to maintain good oral hygiene and a prompt coordination with the patient's physician for drug substitution would be helpful to timely manage such cases.

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