CASE REPORT

Amlodipine-induced Enlargement on Hard Palate and Its Management: A Case Report

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Abstract

Hypertension and angina are widely managed with calcium channel blockers in medical practice. Calcium channel blockers are now known to cause gingival overgrowth as a side effect. Plaque retention sites have the potential to impact the oral bacterial load and negatively affect the quality of life. However, relatively few cases of gingival overgrowth have been reported with amlodipine, a third-generation calcium channel blocker. It is believed that a good oral hygiene regimen is the best way to control gingival overgrowth. The most preferred method of treatment in severe cases is surgical excision, which is followed by careful oral hygiene practices. An amlodipine-treated hypertensive patient with gingival overgrowth is the subject of this case report.

Keywords: Amlodipine, Calcium channel blocker, Drug-induced gingival enlargement, Management.

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Introduction

Gingival overgrowth is one of the key clinical features of gingival pathology. It arises from a variety of causes and is often accompanied by gingival inflammation. In addition to genetics (family), malignancies, and adverse reactions to certain drugs, hereditary factors also play a role in this condition.[1] Phenytoin, cyclosporine, and calcium channel blockers are all associated with this condition. Compared to the first generation of calcium channel blockers, such as nifedipine, amlodipine is a dihydropyridine derivative that has longer action and fewer side effects.[1] The prevalence of gingival overgrowth in patients taking amlodipine was reported to be 3.3%,[2] which is lower than the rate in patients taking nifedipine, that is, 47.8%.[3] There have been reports that amlodipine has promoted gingival overgrowth over the past few years.

Case Description

A 55-year-old female patient came to the department of periodontology, Kanti Devi Dental College and Hospital, Mathura, Uttar Pradesh, India with the chief complaint of growth in her hard palate since the past 2–3 months (Figs 1 to 3).

The patient gives a history of growth on the left side of her hard palate since the past 2–3 months. She also gives a history of a burning sensation but no associated pain and sensitivity to hot or cold food. There was no pus or blood discharge from the lesion. It started as a small lesion but then started to grow and reached diffused size after 3 months. The patient's past medical history revealed she had been on Amlodipine 10 mg, once daily, for the past 4 years. In spite of this, her past dental history was not relevant.

Her personal history does not reveal any adverse habits. She used to clean her teeth once daily with a brush and paste. Her general physical examination revealed that the patient was moderately built and her vital signs were within the normal range. There were no significant extraoral findings.

On intra-oral examination there is irregular growth present on the hard palate extending from the anterior rugae area to the posterior palatal seal region.

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The growth/lesion is reddish pink, corrugated with irregular borders, and undermined appearance predominantly at the mid-palatal region and left rugae area. The uvula is conical in shape.

On palpation, the growth was non-tender, with no discharge present. It is inflamed, compressible, and with a rise in temperature

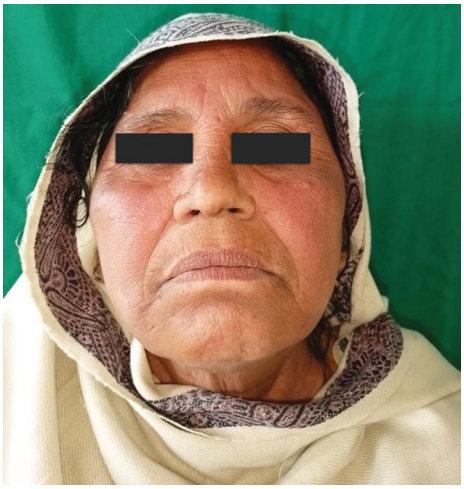

Fig. 1: Clinical picture of the patient

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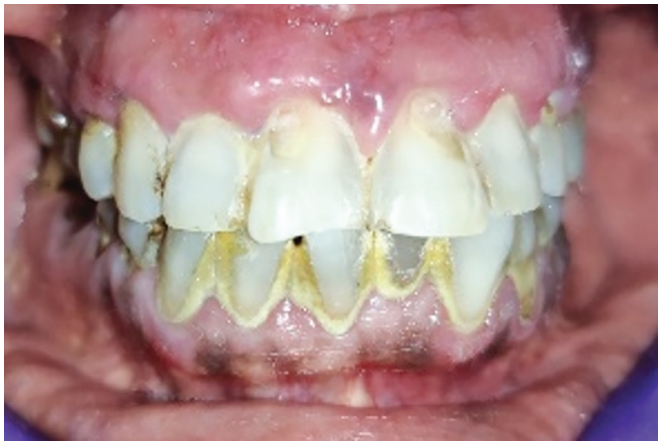

Fig. 2: Clinical picture of patient on first visit

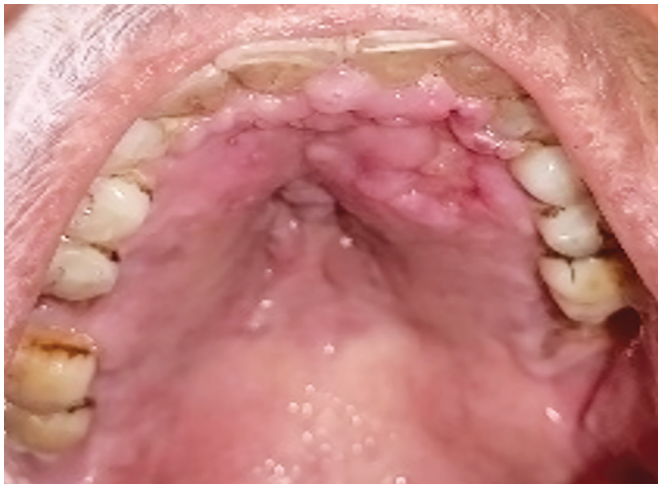

Fig. 3: Clinical picture of the upper teeth and palate of the patient

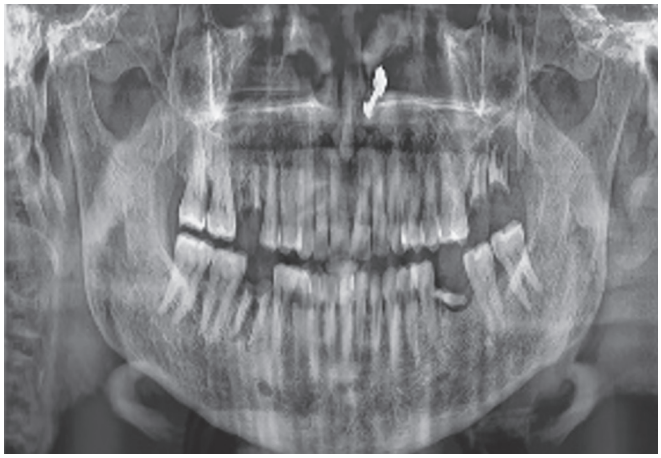

Fig. 4: Radiographic image

than the surrounding structures. The oro–pharyngeal region is also mildly inflamed. All parameters of the hemogram were within normal limits for the patient. There was generalized bone loss and incomplete dentition on orthopantogram (Fig. 4). In the stroma, there were areas of calcification and infiltrated inflammatory cells according to the histopathological report (Fig. 5). On the

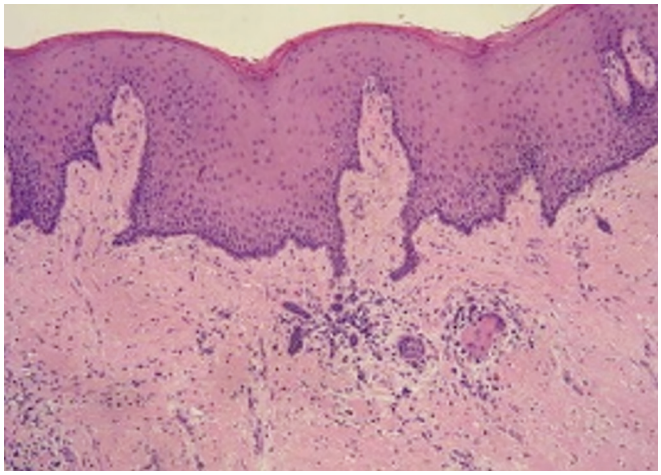

Fig. 5: Histopathological report revealed a mixture of the dense and loose fibrous components with inflammatory cell infiltrate

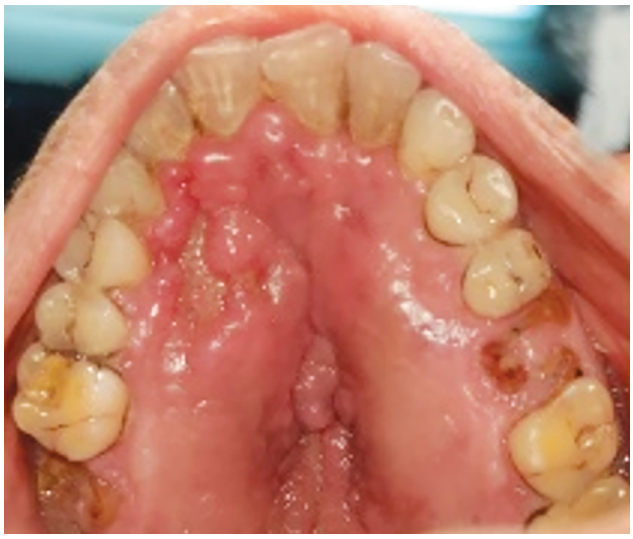

Fig. 6: Regression in the size of gingival enlargement on the third month of drug dosage reduction

basis of the patient's history, clinical features, radiographic and histopathological investigation a diagnosis of amlodipine-induced gingival overgrowth (AIGO) was made.

Initially, the patient was treated non-surgically. Scaling and root planing were performed as part of phase-1 therapies. In order to determine whether it was appropriate to substitute the drug or withdraw it, the patient's physician was consulted. The physician reduced the drug dosage with tablets, telmisartan (40 mg) and amlodipine (5 mg). The use of chlorhexidine oral rinses was recommended for maintaining good oral hygiene.

Despite reducing the drug dosages and maintaining good oral hygiene for 3 months, there was a slow response. A diminution in gingival enlargement was seen from the third to the sixth month (Figs 6 and 7). To restore the normal shape and contour of the gingiva, gingivectomy/gingivoplasty with electrocautery was planned to remove gingival hyperplastic tissue (Fig. 8). An esthetically pleasing gingival contour was achieved postoperatively after the enlarged gingival tissue was successfully eliminated (Fig. 9).

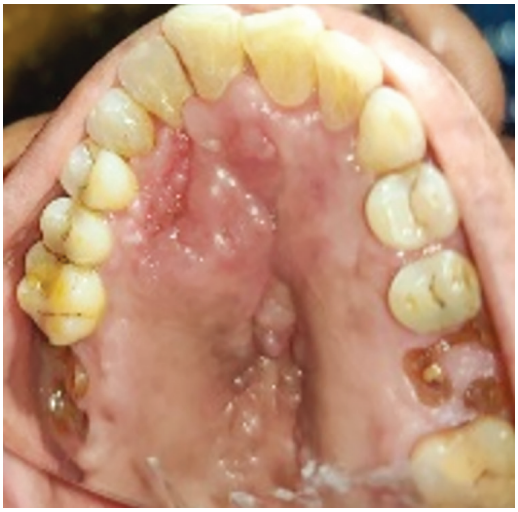

Fig. 7: Regression in the size of gingival enlargement after sixth month of drug dosage reduction

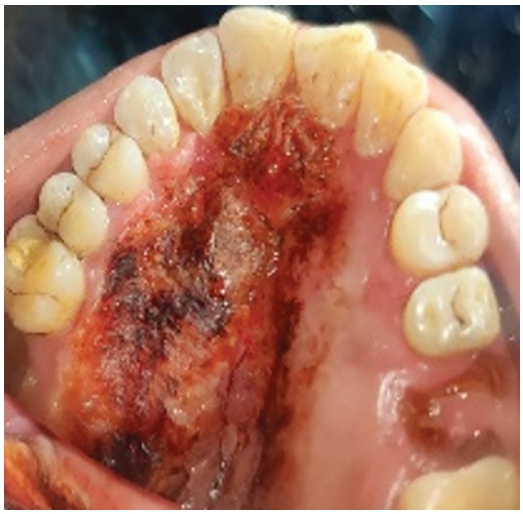

Fig. 8: Surgical excision of gingival hyperplastic tissue by the use of electrocautery

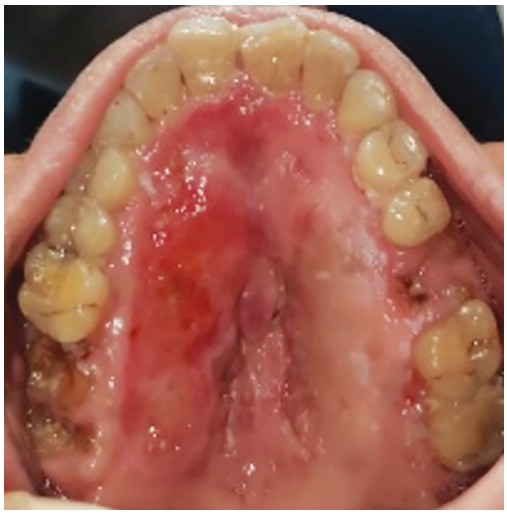

Fig. 9: Postoperative clinical picture after 1 week of surgical excision

Discussion

Abnormal growth of gingival tissue caused by the use of systemic medications is called drug-induced gingival overgrowth (DIGO). In reality, it is not a true term since neither the epithelium nor the cells of the connective tissue are hyperplastic or hypertrophic. Collagen, which makes up most of the extracellular matrix, is responsible for the increase in gingival size. Therefore, it is designated as DIGO.

It has been reported that nifedipine and cyclosporine can cause gingival enlargement as a side effect, in addition to anticonvulsants and immunosuppressants. Other reports indicate that erythromycin and trimethoprim sulphamethoxazole can lead to gingival overgrowth. In addition to phenytoin, some other anticonvulsants have been reported to cause gingival enlargement, including vigabatrin, sodium valproate, primidone, and phenobarbital. Among the calcium channel blockers, gingival enlargement is most commonly associated with nifedipine and also with amlodipine, etc.[4] During the aforementioned period, our patient did not use any of the above medications concurrently. Among the factors contributing to DIGO is poor oral hygiene.[5] Cross-sectional studies have supported the hypothesis that bacterial plaque is a contributory factor or a consequence of gingival overgrowth, but there is no clear evidence.[5] There is still a lack of understanding of the mechanism behind gingival enlargement. It has already been suggested that there are two main pathways, one inflammatory and one non-inflammatory.[6–7] These proposed non-inflammatory mechanisms involve an increase in adrenocorticotropic hormone and keratinocyte growth factor. In addition, decreased folic acid uptake leads to defective collagenase activity, aldosterone synthesis is blocked in the adrenal cortex, and adrenocorticoids are overproduced. As a result of the direct toxic effects of drugs, a bacterial plaque or crevicular gingival fluid may cause inflammation. Several cytokine factors could be upregulated as a result of this inflammation, including transforming growth factor-beta 1 (TGF-β1).[6,8]

For understanding the pathogenetic mechanism of drug-associated enlargement, researchers study the effects of these drugs on gingival fibroblast metabolism directly and indirectly. Since only a subset of patients treated with this medication will experience gingival overgrowth, there is a theory that their fibroblasts have abnormal susceptibility to the medication's adverse effects. The overgrown gingiva of these patients produces an increased amount of protein synthesis, mainly collagen. Additionally, differential proportions of fibroblast subsets in every individual may be involved in susceptibility or resistance to pharmacologically induced gingival enlargement.[9] Plaque and calculus are generally controlled via drug substitution and effective drug substitution.[10] An enlargement that is not resolved by these measures is recommended to be surgically corrected. In spite of these effective treatment options, lesions may recur after treatment.[9] It is important to carefully evaluate whether surgery is needed and when it should be performed. Surgery is normally performed for cosmetic/esthetic needs before any functional consequences are present.[1,10] Surgical intervention has been required in most cases of amlodipine gingival overgrowth.[11] An approach that reduces gingival enlargement using carbon dioxide lasers has provided rapid postoperative hemostasis, a potentially useful technique. Surgical treatment for immunosuppressed patients should be discussed with their physician prior to surgery.[12] Soft tissue lasers are readily absorbed by water and are therefore excellent for use in soft tissues that contain a large amount of water.

Lasers are therefore superior to scalpels due to their hemostatic and bactericidal effects, as well as the fact that their relatively dry fields improve visibility when surgery is performed.[13] In conclusion, an amlodipine side effect that can occur even at low doses or for a short period of time is gingival overgrowth.

References

1. Ellis JS, Seymour RA, Thomason JM, Monkman SC, Idle JR. Gingival sequestration of amlodipine and amlodipine-induced gingival overgrowth. Lancet 1993;341(8852):1102–1103. DOI: 10.1016/0140-6736(93)92470-e.
2. Jorgensen MG. Prevalence of amlodipine-related gingival hyperplasia. J Periodontol 1997;68:676–678. DOI: 10.1902/jop.1997.68.7.676.
3. Nery EB, Edson RG, Lee KK, Pruthi VK, Watson J. Prevalence of nifedipine-induced gingival hyperplasia. J Periodontol 1995;66(7): 572–578. DOI: 10.1902/jop.1995.66.7.572.
4. Sucu M, Yuce M, Davatoglu V. Amlodipine induced massive gingival hypertrophy. Can Fam Physician 2011;57(4):436–437. PMID: 21490356.
5. Hegde R, Kale R, Jain AS. Cyclosporine and amlodipine induced severe gingival overgrowth: Etiopathogenesis and management of a case with electrocautery and carbon dioxide (CO_2) laser. J Oral Health Comm Dent 2012;6(1): 34–42. DOI: 10.5005/johcd-6-1-34.
6. Srivastava AK, Kundu D, Bandyopadhyay P, Pal AK. Management of amlodipine induced gingival enlargement: A series of three cases. J Indian Soc Perodontol 2010;14:279–281. DOI: 10.4103/0972-124X.76931.
7. Routray SN, Mishra TK, Pattnaik UK, Satapathy C, Mishra CK, Behera M. Amlodipine induced gingival hyperplasia. J Assoc Physicians India 2003;51:818–819. PMID: 14651149.
8. Sharma S, Sharma A. Amlodipine induced gingival enlargement: A clinical report. Compend Contin Educ Dent 2012;33(5):e78–e78. PMID: 23268589.
9. Grover V, Kapoor A, Marya CM. Amlodipine induced gingival hyperplasia. J Oral Health Comm Dent 2007;1:19–22. DOI: 10.7727/wimj.2014.089.
10. Triveni MG, Rudrakshi C, Mehta DS. Amlodipine induced gingival overgrowth. J Indian Soc Periodontol 2009;13(3):160–163. DOI: 10.4103/0972-124X.60231.
11. Lombardi T, Fiore DG, Belser U, Di FR. Felodipine induced gingival hyperplasia: A clinical and histologic study. J Oral Pathol Med 1991;20:89–92. DOI: 10.1111/j.1600-0714.1991.tb00896.x.
12. Marshall RI, Bartold PM. A clinical review of drug induced gingival overgrowth. Oral Surg Oral Med Oral Pathol 1993;76(4):543–548. DOI: 10.1016/0030-4220(93)90027-2.
13. American Academy of Periodontology (AAP). Lasers in periodontics. J Periodontol 2002;73(1):1231–1239. DOI: 10.1902/jop.2002.73.10.1231.