Case Report

Negligence of gingival overgrowth leading to loss of entire dentition

Monali Shah, Vandana Shah,[1] Megha Ellu Poojari, Nishanta Bora[2]

Departments of Periodontology and [1]Oral and Maxillofacial Pathology, K.M. Shah Dental College and Hospital, Sumandeep Vidyapeeth, Vadodara, Gujarat, India, [2]MSc Public Health Student, University of East London, London, UK

The work belongs to the Department of Periodontology, K.M. Shah Dental College and Hospital, Vadodara, Gujarat, India

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Address for correspondence:
Dr. Monali Shah, Room No: 7, Department of Periodontology, K.M. Shah Dental College and Hospital, Waghodia Road, Piparia, Waghodia Taluka, Vadodara - 391 760, Gujarat, India.
E-mail: smonali2011@gmail.com

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Abstract:

Drug-induced gingival enlargement often occurs due to patient's lack of awareness about the side effects of prescribed medications. This case report details an unusual instance of massive drug-induced gingival overgrowth in a 50-year-old female, successfully managed through a multidisciplinary approach, including surgical intervention and prosthetic rehabilitation. The surgical treatment involved multiple extractions and the excision of excessive tissue. Both arches healed completely after surgery, and the patient underwent prosthetic rehabilitation, with no signs of recurrence. Effective management of such cases relies on patient counseling and appropriate drug substitution. Increasing awareness about the side effects of certain medications and the connection between systemic and oral health is crucial to prevent such cases of gingival enlargement.

Key words:

Antihypertensive, drug-influenced gingival enlargement, fibrous enlargement, generalized periodontitis Stage IV Grade C

INTRODUCTION

The prevalence of drug-induced gingival enlargement, commonly linked to antihypertensive medications, has risen significantly (25%–75%) in recent years, coinciding with the growing incidence of hypertension among younger populations.[1] Frequently prescribed antihypertensives associated with gingival overgrowth include calcium channel blockers such as amlodipine, nifedipine, benidipine, diltiazem, felodipine, manidipine, nitrendipine, nicardipine, nisoldipine, nimodipine, verapamil, along with other drugs such as atenolol, propranolol, and metoprolol.[2] These medications inhibit aldosterone synthesis by blocking calcium flux, triggering increased adrenocorticotropic hormone (ACTH) secretion from the pituitary, which in turn stimulates fibroblast and collagen proliferation via dihydrotestosterone receptors.[3] However, not all individuals experience this side effect, as responses vary, with some being "responders" and others "nonresponders" to the drug's pharmacological impact.[4]

Several other factors contribute to drug-induced gingival hyperplasia, including age, gender, genetic predisposition, poor nutrition, and inadequate oral hygiene, which can amplify the drug's effects on gingival tissue. This condition is often accompanied by pain, halitosis, plaque accumulation, tender gums, aesthetic concerns, and difficulties with speech and mastication, ultimately impairing an individual's overall health and quality of life.[2] The severity and extent of gingival involvement vary, with severe cases affecting both the maxillary and mandibular arches.

Gingival enlargement can lead to the destruction of underlying structures, tooth mobility, and eventual tooth loss. In such cases, extensive surgery is often required to restore the gingiva, and multiple teeth may need extraction if supporting structures cannot be salvaged. The loss of teeth, particularly in younger patients, can result in anxiety, psychological distress, and financial strain due to the need for full-mouth rehabilitation. This situation often arises from a lack of knowledge and awareness about the side effects and complications associated with antihypertensive drugs. We present a rare case of severe drug-induced gingival hyperplasia involving both jaws, leading to complete tooth loss in a 50-year-old female. The report highlights pre- and postoperative care, follow-up, and management, with particular emphasis on patient education and awareness regarding the side effects of antihypertensive medications.

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CASE REPORT

A 50-year-old female presented with a chief complaint of swollen gums in both the upper and lower jaws, along with difficulty chewing and tooth loosening for the past 2 months. Her history revealed that the enlargement began as small, painless, bead-like swellings 3 months earlier, which progressively grew into large tissue folds, partially covering the crowns of her teeth. The patient reported being hypertensive and having hyperthyroidism, for which she had been on medication for 3 years. Her medications included tablet telmikind-AM (telmisartan IP: 40 mg and amlodipine IP: 5 mg), tablet thyroxin 12.5 mg, and tablet revelol-AM (metoprolol IP: 50 mg and amlodipine IP: 5 mg). The dental examination showed significant tooth loss and loosening of the remaining teeth, attributed to the gingival enlargement. No relevant family history was noted.

Intraoral examination revealed extensive gingival overgrowth, covering more than two-thirds of the teeth in both the upper and lower jaws. The enlargement was painless, pale pink, and exhibited a bulbous, bead-like appearance in the interdental papilla, extending to the facial and lingual surfaces, with pseudo-clefts present. In certain areas, reddish lesions disrupted the lobulated surface demarcation [Figure 1]. Generalized bleeding was noted upon probing, and the gingival tissue appeared friable. There was widespread grade III tooth mobility, with a few teeth already lost due to this mobility. Pathological migration affected all teeth, halitosis was pronounced, and deep combined pockets were observed. The Oral Hygiene Index indicated a mean score of 2.4, reflecting fair oral hygiene, while the Gingival Index averaged 3, indicating severe gingival inflammation.

An orthopantomograph (OPG) and cone-beam computed tomography were recommended for a comprehensive periodontal evaluation. The OPG revealed the presence of 18 teeth, with impacted tooth numbers 28, 38, and 48. Tooth 28 showed distoangular impaction, while 38 and 48 exhibited mesioangular impaction. Generalized horizontal bone loss extending to the apical third of the teeth was evident, along with severe periodontal bone loss. Cortical borders remained intact, but mucosal thickening was noted in the floor of the maxillary sinus on both sides. Maxillary sinus pneumatization extended to the crest in the regions of tooth numbers 17, 16, 26, and 27. There was a discontinuity in the sinus floor extending to the crest near tooth numbers 17 and 26–27 on the palatal side [Figure 2]. Based on clinical and radiographic findings, a tentative diagnosis of generalized periodontitis Stage IV Grade C, with drug-induced gingival enlargement (Degree III) affecting both the maxilla and mandible, was made.

The treatment plan was structured as follows: Phase I involved nonsurgical interventions, including scaling and root planing, reinforcement of oral hygiene practices, and prescribing 0.2% chlorhexidine gluconate rinses. The patient was also referred to a physician for drug substitution. The physician replaced the existing medications with tablet telmikind (telmisartan IP: 40 mg) and tablet metoprolol-XL (metoprolol succinate IP: 50 mg). The patient was recalled and followed up for 3 weeks, after which a reevaluation of their oral condition was performed. No significant improvement in the patient's oral status was observed.

Phase II involved surgical intervention. Before the procedure, hematological investigations were conducted. Periodontal flap surgery was performed on both the maxilla and mandible under local anesthesia, and the remaining teeth were extracted due to poor prognosis. The enlarged gingival tissue was excised, and the flap was sutured using 3-0 silk with a simple interrupted suture technique [Figures 3 and 4]. A periodontal dressing (Coe-Pak, GC America Inc., Alsip) was applied to protect the wound and facilitate healing. The excised tissue was sent for histopathological analysis.

Histopathological examination revealed hyperplastic stratified squamous epithelium with dense connective tissue proliferation. The connective tissue exhibited collagen bundle fibrosis and a dense inflammatory infiltrate consisting of lymphocytes, a few mast cells, and predominantly plasma cells [Figures 5 and 6]. A diagnosis of fibrous enlargement was established.

A follow-up oral examination conducted 4 weeks later revealed fully healed gingiva in both the maxilla and mandible, with no signs of recurrence. Subsequently, Phase III of treatment, focused on prosthetic rehabilitation, was initiated. Considering the patient's financial situation and best interests, a complete denture was fabricated and delivered. The patient remains under follow-up care with no signs of recurrence to date.

DISCUSSION

This report presents a case of drug-induced generalized gingival enlargement that resulted in the complete loss of dentition. The clinical, radiographic, and histological findings, along with their management, are discussed. Drug-induced gingival enlargement can manifest as discrete, localized, or generalized overgrowth, as seen in this case, depending on the extent of involvement. It is more prevalent in children and females, with the overgrowth typically confined to the attached gingiva, which becomes soft, friable, and prone to bleeding. Systemic medications that may contribute to gingival enlargement include anticonvulsants, immunosuppressants, and calcium channel blockers. The 2017 classification introduced by the World Workshop categorizes drug-induced gingival enlargement as gingivitis induced by dental biofilm.[2] In cases of drug-induced gingival conditions, plaque bacteria, in conjunction with the medication, play a significant role in eliciting a gingival response. There are very few documented instances in the literature where drug-induced gingival enlargement has resulted in complete tooth loss. This case report details the clinical, radiographic, and histological findings, as well as the management of extensive drug-induced gingival enlargement.

In this case, the family history was noncontributory, and the patient's past dental history did not align with the cause of the condition. The patient has been taking tablet amlodipine (10 mg) for hypertension for the past 3 years. Amlodipine is a third-generation dihydropyridine calcium antagonist, structurally similar to nifedipine, and pharmacodynamically comparable. It is prescribed for the

management of hypertension and angina pectoris and is known to induce gingival hypertrophy. According to Jorgensen, the prevalence of amlodipine-induced gingival enlargement ranges from 1.7% to 3.3%.[5] The incidence of gingival enlargement caused by amlodipine is lower than that associated with nifedipine.[5] Typically, drug-induced gingival enlargement occurs within the first 3 months of administration;[6] however, in this case, the patient had been on the medication for 3 years, with enlargement observed only in the last 2 months.

Although the mechanisms by which various drugs induce gingival enlargement differ, the clinical manifestations and changes are consistent. The enlargement typically begins as a nodular overgrowth of the interdental papilla, which then expands to encompass the facial and lingual gingiva as well as the crowns of the teeth, ultimately forming extensive growths with pseudo-clefts.[7] Histologically, this condition is characterized by hyperplastic para-keratinized stratified squamous epithelium with acanthosis in the spinous layer, while the underlying connective tissue appears densely fibrous, marked by an increased number of fibroblasts. The inflammatory cell infiltrate primarily consists of lymphocytes and plasma cells.[8] Any gingival enlargement is associated with an increase in the connective tissue component, with fibroblasts being the key target cells involved.[9] Furthermore, gingival enlargement is described as "multi-factorial," influenced by various predisposing factors such as poor oral hygiene, gingival inflammation, genetic predisposition, and hypersensitivity, all of which contribute to the enlargement. This suggests that gingival enlargement results from an interaction among gingival fibroblasts, plaque, inflammatory mediators, and drug metabolites.[4]

The pathogenesis of amlodipine-induced gingival enlargement is not yet fully understood, though both inflammatory and noninflammatory mechanisms have been proposed. It has been suggested that a decrease in collagenase activity results from reduced folic acid uptake, the inhibition of aldosterone synthesis in the adrenal cortex, and a subsequent increase in ACTH levels, along with the upregulation of keratinocyte growth factor. Alternatively, inflammation may arise from the direct toxic effects of concentrated drug levels in crevicular gingival fluid and/or bacterial plaque, which could lead to the upregulation of various cytokines, such as transforming growth factor-beta 1.[10] There may also be an abnormal susceptibility of fibroblasts to the administered drugs, as each individual possesses a unique subset of fibroblasts, potentially leading to varying responses to the same medication. This variation can explain why a particular drug may cause gingival enlargement in some patients but not in others.[10]

Treatment for amlodipine-induced gingival enlargement involved Phase I nonsurgical therapy, which included scaling and root planing, reinforcement of oral hygiene practices, and chlorhexidine gluconate rinses. The patient was then referred to a physician for medication substitution. Upon reevaluation after 3 weeks, there was no regression of the gingival enlargement, prompting the initiation of Phase II therapy, which included full mouth gingivectomy and the extraction of teeth with hopeless prognosis. Following curettage in the affected areas, the tissue was approximated using 3-0 silk sutures [Figures 3 and 4]. Sutures were removed at 2 weeks postoperative and uneventful healing was noted [Figures 7 and 8]. The patient has since been placed on a regimen of frequent recalls and follow-ups [Figures 9 and 10]. After complete healing of the lesions, Phase III prosthetic rehabilitation was undertaken. Full-mouth rehabilitation is necessary to restore functionality in such cases, with options including implant-supported prostheses, implant-supported overdentures, and complete dentures. Considering the patient's needs and financial situation, a complete denture was fabricated and delivered. A 2-year follow-up with the complete denture indicated full functionality and no signs of recurrence [Figure 11].

This case report highlights that massive drug-induced gingival enlargement was caused by amlodipine. Successful treatment requires identifying and eliminating the etiological factors, alongside reinforcing oral hygiene and surgically excising the affected tissue. Due to the complete loss of dentition resulting from the poor prognosis of the teeth, a multidisciplinary approach was essential for effective management. The patient's quality of life significantly improved following treatment. Further studies and reports are necessary to enhance understanding and management of similar cases.

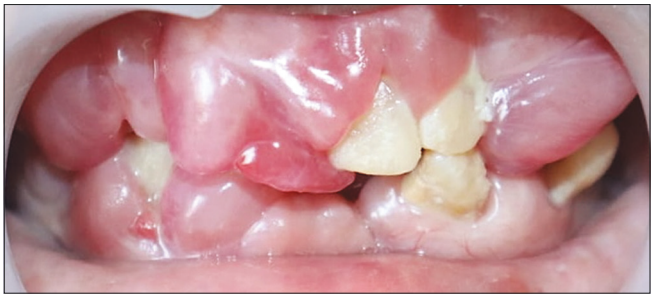

Figure 1: Intraoral front view of maxilla and mandible showing excessive overgrowth of gingiva

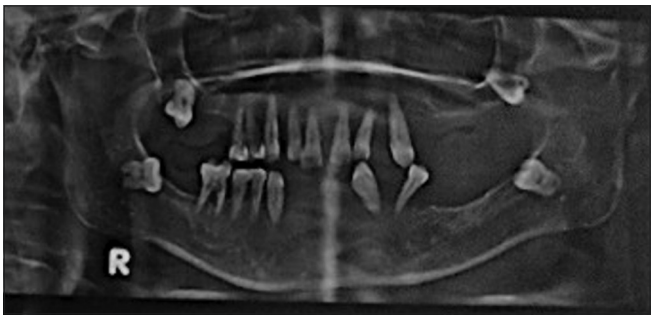

Figure 2: Orthopantomagram showing tooth with hopeless prognosis

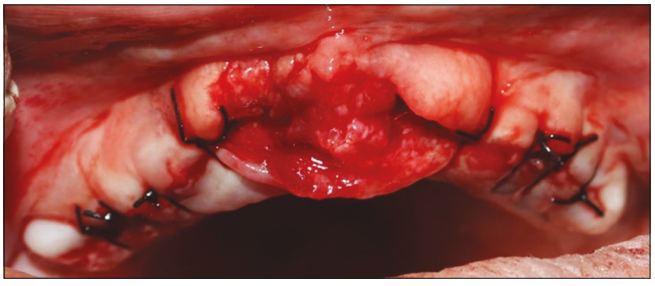

Figure 3: Intraoral occlusal view of maxilla after full mouth extraction and surgical excision of excessive gingival tissue

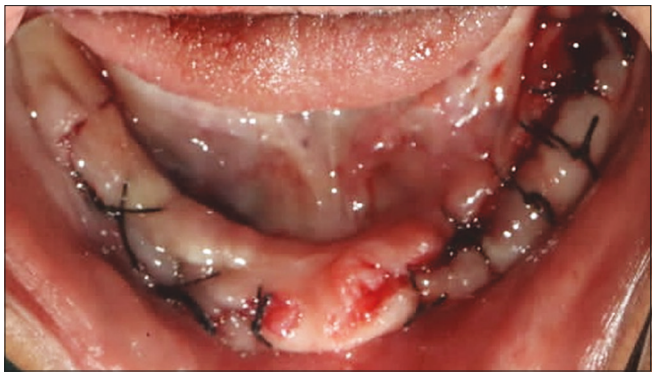

Figure 4: Intraoral occlusal view of mandible after full mouth extraction and surgical excision of excessive gingival tissue

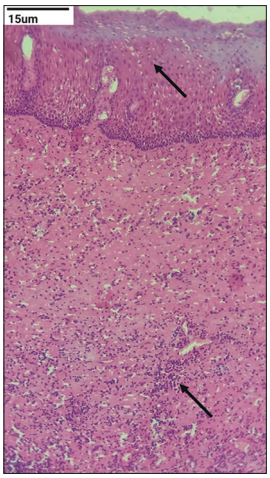

Figure 5: Histologic section at 15 μm showing hyperplastic stratified squamous epithelium (top black arrow) and dense connective tissue proliferation (lower black arrow)

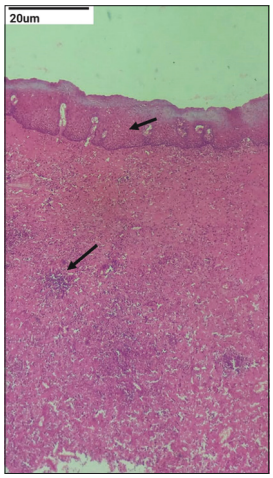

Figure 6: Histologic section at 20 μm showing hyperplastic stratified squamous epithelium (top black arrow) with fibrosis of collagen bundle (lower black arrow) and dense inflammatory cell infiltrate

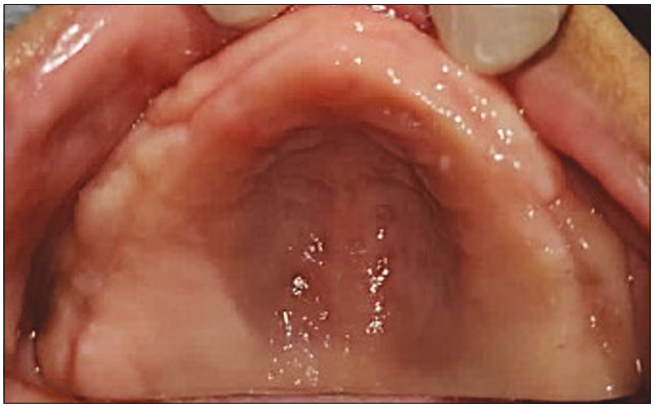

Figure 7: Intraoral occlusal view of maxilla at 1-month follow-up

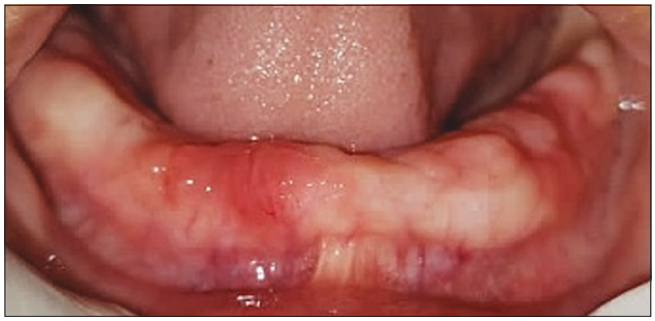

Figure 8: Intraoral occlusal view of mandible at 1-month follow-up

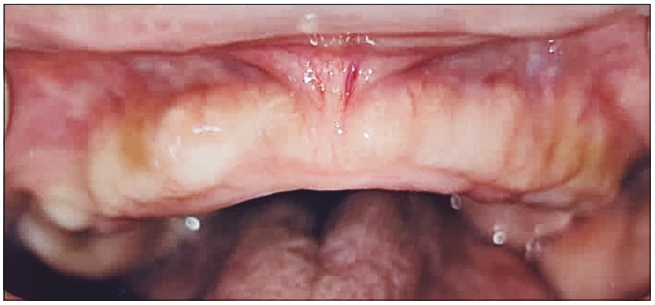

Figure 9: Intraoral frontal view of maxilla at 1-year follow-up

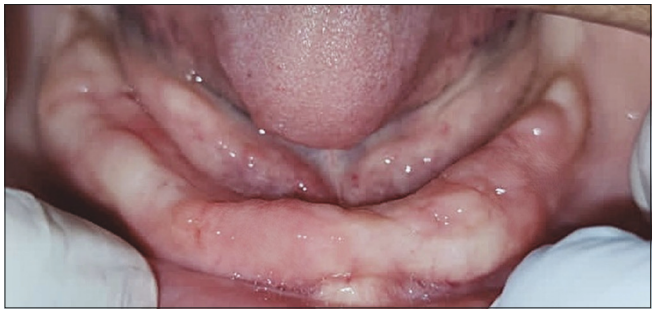

Figure 10: Intraoral occlusal view of mandible at 1-year follow-up

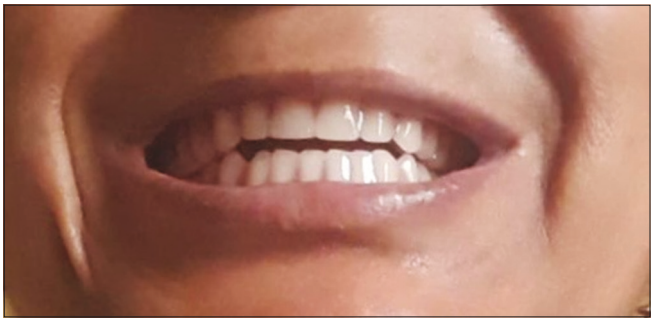

Figure 11: Prosthetic rehabilitation using complete denture

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. 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;19:308-11.
2. Bharti V, Bansal C. Drug-induced gingival overgrowth: The nemesis of gingiva unravelled. J Indian Soc Periodontol 2013;17:182-7.
3. Tonsekar P, Tonsekar V. Calcium-channel-blocker-influenced gingival enlargement: A conundrum demystified. Oral 2021;1:236-49.
4. Seymour RA, Thomason JM, Ellis JS. The pathogenesis of drug-induced gingival overgrowth. J Clin Periodontol 1996;23:165-75.
5. Jorgensen MG. Prevalence of amlodipine-related gingival hyperplasia. J Periodontol 1997;68:676-8.
6. Raghavendra MH, Shreehari AK, Srivastava D. Non-surgical management of a case of amlodipine-induced gingival enlargement. J Evol Med Dent Sci 2014;3:9658-89.
7. Bhatnagar S. Treatment of gingival enlargement. In: Gingival Disease-A Professional Approach for Treatment and Prevention. London, UK: IntechOpen; 2019.
8. Droździk A, Droździk M. Drug-induced gingival overgrowth-molecular aspects of drug actions. Int J Mol Sci 2023;24:5448.
9. Ramírez-Rámiz A, Brunet-LLobet L, Lahor-Soler E, Miranda-Rius J. On the cellular and molecular mechanisms of drug-induced gingival overgrowth. Open Dent J 2017;11:420-35.
10. Joshi S, Bansal S. A rare case report of amlodipine-induced gingival enlargement and review of its pathogenesis. Case Rep Dent 2013;2013:138248.