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Management Of Amlodipine Associated Drug Induced Gingival Enlargement With Non-Surgical Periodontal Therapy - A Case Report

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Abstract:
Background: *Drug-induced gingival enlargement (DIGE), also known as gingival hypertrophy, is a well-documented adversity of certain class of drugs, including anticonvulsants, immunosuppressants, and calcium channel blockers. It manifests as abnormal gingival overgrowth, often leading to functional impairment and esthetic concerns.*
Case Report: *A 58-year-old female presented in the Outpatient Department of Periodontics, with a chief complaint of generalized swelling of her gingiva for past 1 year associated with episodes of pus discharge in upper left front tooth region. The medical history revealed long-term polypharmacy for myasthenia gravis, systemic hypertension, and diabetes mellitus.*
Intervention: *The patient underwent a comprehensive evaluation to identify the causative drug. A non-surgical, two-phased periodontal therapy was implemented, including meticulous oral hygiene reinforcement and professional debridement. Coordination with the patient's physician was undertaken to explore alternative medications to amlodipine.*
Results: *Following periodontal therapy, a significant reduction in gingival inflammation and enlargement was achieved, with marked improvement in function and oral hygiene. Consideration of alternative antihypertensive therapy was also discussed to minimize recurrence risk.*
Conclusion: *This case underscores the importance of timely recognition of DIGE, especially in patients on multiple systemic medications. An interdisciplinary approach, combining periodontal therapy with medical consultation for drug substitution, is essential for effective management and prevention of recurrence.*
Key words: *Amlodipine, Gingival hypertrophy, Drug induced gingival enlargement,*

INTRODUCTION:

Drug-induced gingival enlargement (DIGE) is a well-recognized adverse effect associated with specific pharmacological agents, notably anticonvulsants, immunosuppressants, and calcium channel blockers (CCBs).[1,2] Among CCBs, nifedipine is most frequently implicated, while amlodipine, a third-generation dihydropyridine derivative, is comparatively less associated, with reported prevalence rates ranging between 1.7% and 3.4%.[3,5] Despite this relatively low incidence, amlodipine-induced gingival enlargement (AIGE) has gained clinical significance due to its increasing recognition in both clinical studies and case reports.

The pathogenesis of AIGE remains multifactorial and incompletely understood. Proposed mechanisms include altered calcium influx within gingival fibroblasts, leading to abnormal collagen metabolism, as well as genetic predisposition, particularly multidrug resistance 1 (MDR1) gene polymorphisms, which may modify inflammatory responses to drugs.[6,7] Other risk factors such as plaque-induced inflammation, oral hygiene status, drug dosage, and duration of therapy further modulate the severity of the condition.[4,8]

Clinically, AIGE presents as firm, lobulated, or nodular enlargement of the gingiva, predominantly affecting the anterior interdental papillae. However, atypical presentations, including palatal involvement and edentulous ridge overgrowth, have been documented. [9] This condition may compromise aesthetics, speech, mastication, and oral hygiene, while also accelerating periodontal disease progression.[10]

Given these challenges, management requires a multidisciplinary approach. Substitution of the causative drug (where medically feasible), strict plaque control, and in severe cases, surgical excision of the overgrown tissue forms the basis of therapy.[5]

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Case report:

A 58-year-old female presented in the Outpatient Department of Periodontics, with a chief complaint of generalized swelling of her gingiva for past 1 year associated with episodes of pus discharge in upper left front tooth region. The swelling was reported to have a slow onset and a gradual progress to attain current size. Episodes of daily bleeding during brushing without association with pain was reported. Patient was a known case of Myasthenia gravis (MG), diabetes mellites (DM), hypertension (HTN) for past 10 years. She was on Azathioprine, Pyridostigmine, prednisolone, gabapentin and calcium/ Vitamin D supplements as apart of treatment for MG. Also, she was on amlodipine (5 mg twice daily dose) and metformin (500mg three doses/ day) for HTN and DM respectively.

General examination showed that the patient is moderately built had conjunctival pallor and was otherwise normal. Intraoral examination showed a well-defined swelling in left upper tooth region in relation to 22 and 23 teeth involving marginal, attached gingiva and interdental papilla, measured about 3 x 3 cm which extended antero-posteriorly from the mesial aspect of #22 to distal aspect #**24 [FIG 1**]. The consistency of the swelling was firm and was tender to palpation with evidence of pus discharge in the same region. A similar non-tender swelling was noted in the right lower region involving marginal, attached and interdental papilla in relation to the #45, #44, #43 teeth. The other dental findings were generalised attrition and mobility present in multiple teeth (#18, #16, #21, #22 and #23 teeth). Generalised bleeding on probing (BOP) was present with spontaneous bleeding in #22-#24 areas. There were pseudo pockets (4 mm- 5 mm deep in relation to #22, #23 respectively). Partial clinical crown coverage was seen by gingival tissue in this area. A provisional diagnosis of drug induced gingival enlargement (DIGE) was made keeping periodontal abscess, squamous cell carcinoma, pyogenic granuloma (with respect to #23, #24) and combined drug induced enlargement (CDIE) as the differential diagnoses. The routine blood examination showed lower levels of haemoglobin [(Hb)=7.8 gm/dl] and otherwise normal remarkable blood counts (i.e Total leukocyte count =8900/mm^3 with differential picture having 70% neutrophils, 21 %lymphocytes, 4.4% eosinophils, 3.7% monocytes and 4% basophils). The glycated haemoglobin was consistent with existing DM (HbA1c =6.6) and platelet count was 436 10^3/ul which within the normal limits. Based on the clinical findings, a working diagnosis was made drug as induced gingival enlargement (DIGE) probably associated with Amlodipine.

The treatment plan consisted of identification of causal drug, Scaling root planning (SRP) protocol, alternative medical therapy for identified causal drug (under medical expert guidance) and antibiotics as needed. The timeline of these treatments is given **in Table 1. The** patient was on drugs for MG, HTN, and DM of which amlodipine being a causative of DIGE is well established in medical/ dental literature. However, we identified the causal after being classified as "possible adverse reaction" (score of 7) on the Naranjo drug reaction scale for amlodipine was done as per previous report.[5] The scores for other drugs ranged between 2-4 which patient was on for MG and DM.

The patient had complete reduction in the gum swelling after shifting to enalapril (ACE inhibitor) form amlodipine **[FIG 2,3].** The dental treatment was given in 2 phases evaluating for alteration in swelling of gingiva, BOP, and BP. A temporary surge in BP was managed by re-use of amlodipine before substitution with Furosemide (loop diuretic). The BP was normalised, and patient was satisfied with the overall treatment for DIGE. Currently the patient is in the 6th month of follow-up.

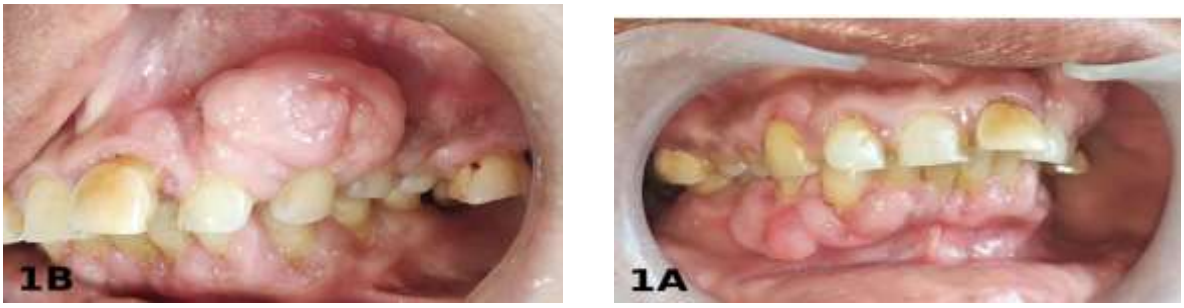

FIGURE 1: A & B initial lesion at time of presentation (whole arch)

Date and day of visit	Patient summary	Interventions
Day 0	• Well defined swelling 3 x 3 cm firm in consistency, irregular border,	• Advised Chlorhexidine mouth wash 0.2% for a period of 2 weeks.

	tender on palpation with pus discharge noted. (figure 1)	• Advised medical fitness fand, request for alterative to identified causal drug
Day 3		• Oral enalapril was the substitution to Amlodipine under medical expert's opinion.
Day 9		• Patient was deemed fit for dental therapy (SRP) • Was kept on iron supplements and multivitamins by medical team.
Day 10		• Phase I therapy started (scaling done)
Day 11	• Reduction in swelling/ inflammation • Bleeding on probing (BOP) was reduced • blood pressure (BP) spiked to 170/102 mmHg.	• SRP done with systemic antibiotics • Patient was re-started on Amlodipine due to high blood pressure (BP) as per medical advice.
Day 12	• BP was 172/99 mm Hg	• SRP repeated, oral hygiene instructions (OHI) reinforced and was advised to continue with chlorhexidine mouth rinse.
1 month follow-up	• BP was 164/96 mm Hg	• Patient was shifted to Furosemide 20mg once daily as per medical advice
3 months follow-up	• BP was 142/76 mm Hg	• Phase I repeated, • Swelling size has reduced
5 month follow-up	• No spikes in BP	• Gingival swelling reduced considerably
6 months follow-up	• BP was 130/90 mm Hg	• Swelling completely reduced (See Figure 3)

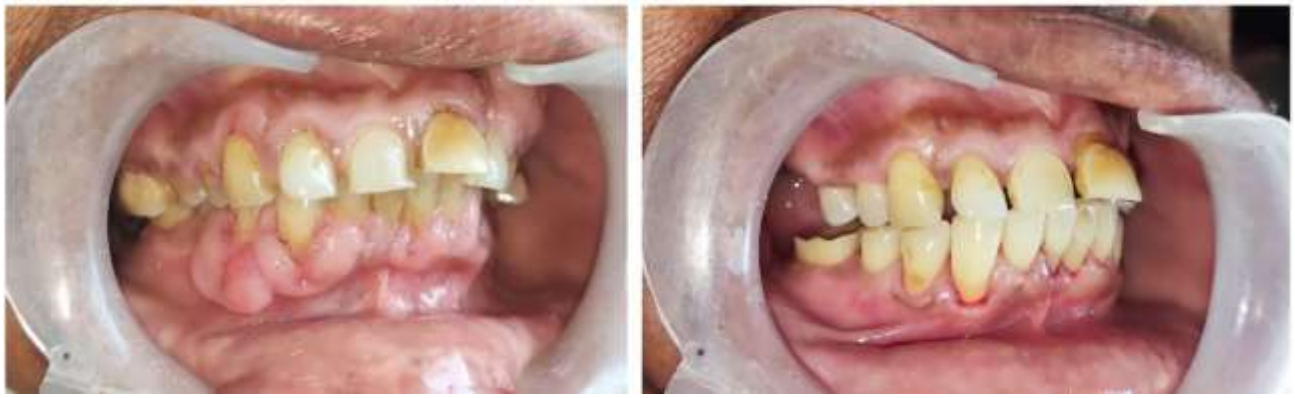

FIGURE 2: Pre and post-operative 6 months view in lower teeth region

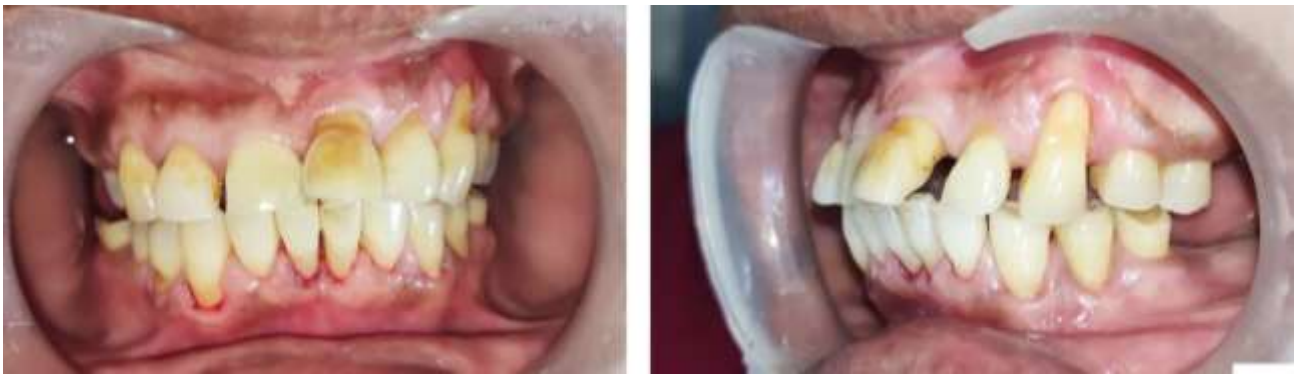

FIGURE 3: 6- months follow up image showing complete regression of DIGE

DISCUSSION:

The current literature indicates that while the prevalence of AIGE is lower than nifedipine-induced enlargement, the condition presents significant diagnostic and therapeutic challenges. Tejnani et al. reported a prevalence rate

of 3.4% in a rural Indian population, with gingival overgrowth strongly correlated with plaque scores and drug dosage.[4] This finding underscores the synergistic role of systemic drug action and local inflammatory factors in pathogenesis.

Atypical cases highlight diagnostic dilemmas. Narwal et al. described a unique case where amlodipine-induced hyperplasia masked an underlying salivary gland malignancy, leading to delayed diagnosis.[1] Similarly, Mathur et al. reported persistent gingival overgrowth in an edentulous patient on amlodipine, suggesting that certain fibroblast subpopulations may sustain drug-induced changes independent of teeth.[9]

Genetic predisposition has been increasingly implicated. Naik et al. reported a case linking MDR1 C3435T polymorphism with AIGE, reinforcing the role of pharmacogenetic susceptibility in disease expression.[6] This may explain why some patients on identical doses of amlodipine develop gingival enlargement while others do not, pointing toward the potential for future personalized risk assessment.

From a management perspective, conservative strategies such as drug substitution, scaling, and strict oral hygiene maintenance have variable outcomes. In most cases, surgical intervention gingivectomy or flap procedures is required for definitive resolution.[11] Nevertheless, recurrence is common if the offending drug is continued and plaque control is inadequate.

In summary, AIGE is a multifactorial condition where drug action, plaque-induced inflammation, and host genetic susceptibility interact. Clinicians must maintain vigilance in patients undergoing long-term amlodipine therapy. Early diagnosis, interdisciplinary collaboration, and individualized treatment protocols are crucial to prevent functional and aesthetic impairment, and to ensure comprehensive management of this drug-related adverse event.[11]

Conflicting Interest (If present, give more details): Nil
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