

## Treatment and Management of Calcium Channel Blocker-Induced Gingival Enlargement

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### Abstract

This article introduces the management of severe Calcium channel blocker-induced gingival enlargement. Patient with severe gingival enlargement was treated with combination of non-surgical and surgical treatments. Clinical examination including probing depths, clinical attachment levels, bleeding on probing (BOP) and radiographic bone levels were recorded at baseline and 12-months postoperatively. At the end of the 12-months follow-up period, there was found to be significant reduction in probing depths, reduction in BOP, and re-alignment of the dentition due to resolution of severe gingival inflammation and osseous recontouring.

**Keywords:** *Gingival Enlargement; Calcium Channel Blocker; Amlodipine; Nifedipine*

### Introduction

Three main classes of drugs are known to cause gingival enlargement: 1) Immunosuppressant (Cyclosporine); 2) calcium channel blockers (CCBs) and 3) anticonvulsants (Phenytoin). Cyclosporine is most likely to cause gingival enlargement among immunosuppressant. Gingival enlargement (GE) is a clinical manifestation often found in patients using either medications. GE is generally plaque induced and is exaggerated with CCBs due to increased extracellular ground substance as well as increased numbers of fibroblasts [1]. GE prevalence varies among different CCBs including 75% for nifedipine; 31.4% for amlodipine and 25% for combined amlodipine and metoprolol [2-4].

Gingival enlargement can occur even in patients with good oral hygiene, but microbial plaque is the main factor causing gingival enlargement in so-called "drugs-induced gingival enlargement". Inclusion of medical consultation with patient's physician is an important step in management of these cases. Exclusion or changing of the suspected medication is possible depending on the medical status of the patient. Regression and gingival inflammation reduction may be seen if the drug can be discontinued or changed after couple of months.

However, non-surgical therapy, including control of local factors, plaque and calculus with scaling and root planning and good oral hygiene are recommended regardless of the patient being able to safely discontinue or change the drug or is still experiencing gingival enlargement. Surgical intervention is needed if patient does not respond to non-surgical therapy alone.

This case report describes a patient with a history of taking nifedipine, recently changed to amlodipine, presenting with severe generalized gingival enlargement superimposed on severe periodontitis and miss-aligned dentition, malposition and flaring, specially of anterior teeth due to severe inflammation. The objective of this report is to present a clinical case of drug-induced gingival enlargement attributed to calcium channel blocker, outline clinical features, non-surgical and surgical management.

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Methods

Clinical presentations

Patient

A 51-year-old African American male, ASA II, was referred to the Graduate Periodontics Clinic at the Detroit Mercy School of Dentistry for management of drug-induced gingival enlargement. Medically, the patient was a non-smoker and diagnosed with hypertension (HTN) controlled with amlodipine with no known drug allergies. Clinical examination revealed generalized erythematous gingival enlargement (Figure 1) with probing depth ranging between 4 - 11 mm and moderate to severe bleeding upon probing, anterior teeth splaying and malposition. Radiographically (Figure 2), patient had bone loss of 15 - 30% with moderate to heavy subgingival calculus.



Figure 1: 1a-1c: Clinical appearance at initial presentation showing generalized erythematous gingival enlargement.

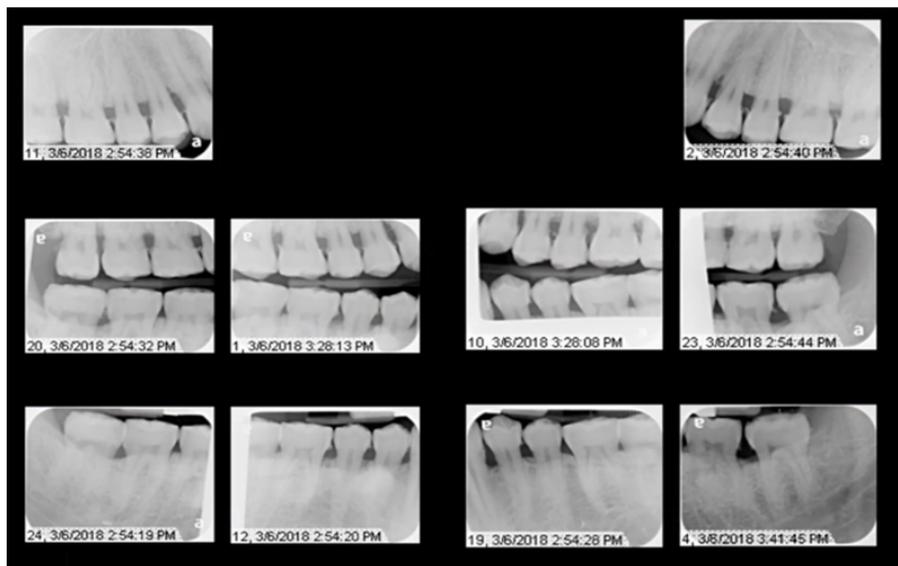
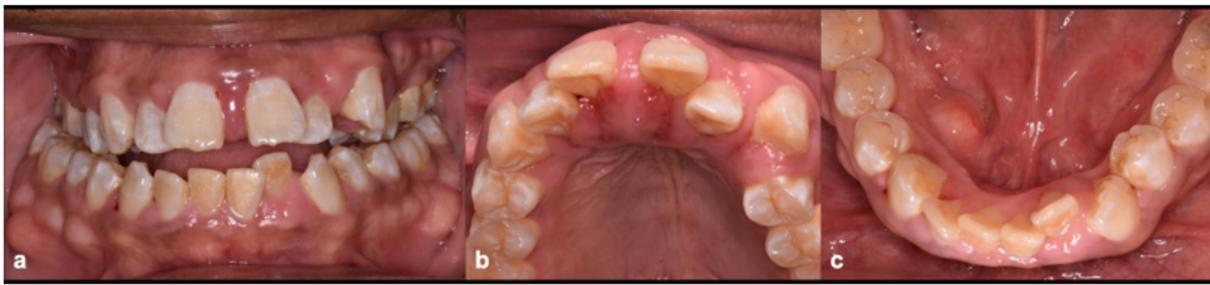


Figure 2: Full mouth series showing generalized bone loss of 15 - 30% with moderate to severe subgingival calculus.

### Case management

The clinical examination revealed generalized deep probing depths of 5 - 11 mm with bleeding on probing (BOP). Radiographically, generalized bone loss less than 30% with generalized heavy subgingival calculus. Medical consultation provided prior to periodontal treatment for a possible change of hypertensive medication. Non-surgical therapy consisting of oral hygiene instructions and scaling and root planning were the primary methods of treating the inflammatory component of gingival enlargement. Patient was instructed to follow strict home care oral hygiene. Periodontal re-evaluation was completed after 4 - 6 weeks and significant resolution of gingival inflammation was noted, but non-surgical therapy alone was not enough to sufficiently reduce deep pockets. There was a difficulty to access wisdom teeth for proper oral hygiene care at home, and professional instrumentation (Figure 3). Although dramatic reduction of gingival inflammation and probing depths were achieved, it was decided at the re-evaluation appointment that patient required surgical therapy as well. Patient's physician has also switched patient's CCB to Amlodipine which has lower incidence of gingival enlargement as compared to Nifedipine. Periodontal maintenance was completed prior to surgical phase which also gave us an opportunity to re-assess patient's homecare and oral hygiene (Figure 4).



**Figure 3:** 3a-3c: Periodontal re-evaluation was completed after 4 - 6 weeks and significant resolution of gingival inflammation noted.

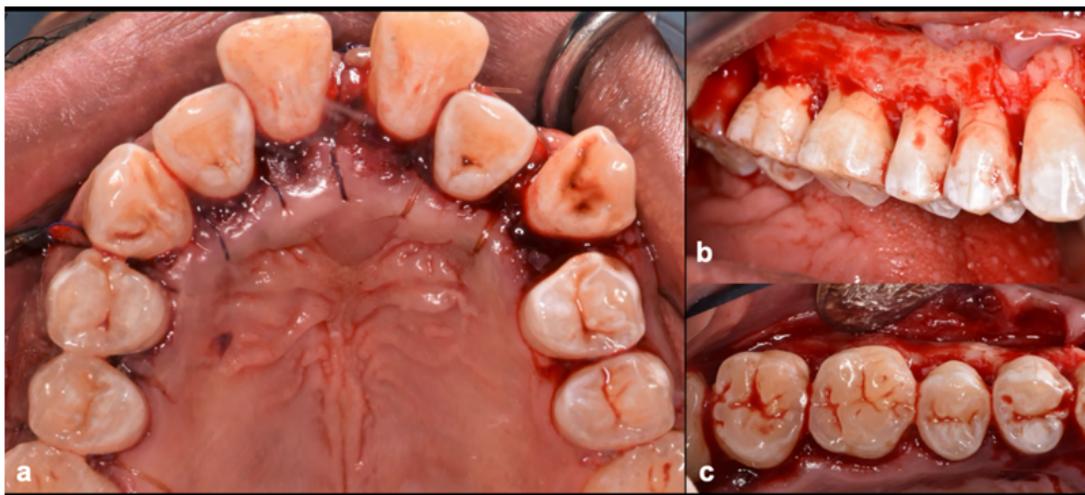


**Figure 4:** 4a-4c: Periodontal maintenance completed prior to surgical phase to also re-assess patient's homecare and oral hygiene.

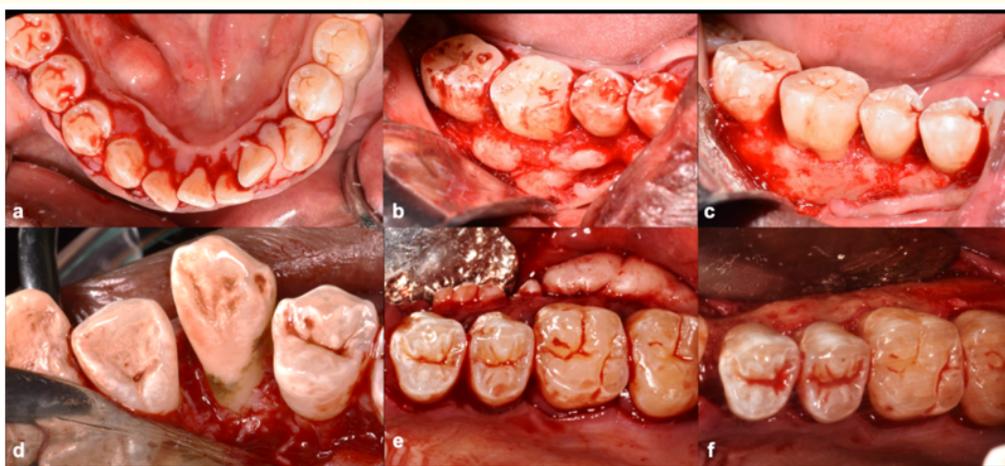
Surgical intervention included extraction of 3<sup>rd</sup> molars, osseous pocket reduction and gingival recontouring surgery for all 4 quadrants under moderate conscious IV sedation (Figure 5). Per our surgical protocol, 800 mg Ibuprofen was given to the patient prior to the procedure, patient rinsed with 15 mL of 0.12% Chlorhexidine Gluconate for one minute and local anesthesia was administered. Combination of inverse bevel and sulcular incisions were made with full thickness flap reflection, mechanical debridement using hand instrumentation

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and ultrasonic scaler. Osteoplasty and osteoectomy was completed, and buccal and lingual flaps were apically positioned and sutured using vertical mattress technique (Figure 6). The palatal flaps were repositioned and primary closure obtained using resorbable Chromic gut sutures. All wisdom teeth were removed in the Oral Surgery Department at Detroit Mercy School of Dentistry. Post-surgically, the patient received prescriptions for Ibuprofen 800 mg and 0.12% Chlorhexidine. Post-surgical instructions were given and the patient resumed gentle brushing two weeks post-operatively. The patient received periodontal maintenance every 3-months and was followed up to 12-months (Figure 7).



**Figure 5:** 5a-5c: Osseous pocket reduction and gingival re-contouring surgery. a) Vertical mattress suture used to secure an apically positioned flap. b-c) Full thickness flap with osteoplasty and osteoectomy to create positive architecture and eliminate buccal bony ledges and exostoses.



**Figure 6:** Combination of inverse bevel and sulcular incisions were made with full thickness flap reflection (6a), mechanical debridement using hand instrumentation and Cavitron, osteoplasty and osteoectomy completed (b-c). Subgingival calculus was noted on lingual of #11 upon flap reflection (6d). After debridement, degranulation and osseous recontouring, flaps were re-positioned (palatal) and apically positioned (buccal and lingual) and secured using vertical mattresses sutures (6b-c-e&f).



**Figure 7:** The patients received periodontal maintenance every 3-months and were followed up to 12-months.

### Clinical outcomes

There was an overall reduction in PD from baseline to 12-months after surgery. Generalized probing depths decreased to 3 - 4 mm with improvement in gingival contours and significant re-alignment of the dentition due to resolution of severe inflammation. Patient's motivation, compliance with periodontal maintenance and strict home care oral hygiene helped with further improvement post-surgery. Complete resolution of the gingival enlargement was noted.

### Discussion

The pathogenesis of CCB-induced GE is poorly understood and is considered a multifactorial condition. Management of drug-induced gingival enlargement can be complicated by the difficulties in adequate plaque control with unfavorable contour of the enlarged gingival tissues, and high incidence of recurrence [5]. Medical consultation and replacing a drug by patient's physician can be very beneficial, but underlying medical condition limits this option.

The risk of severe gingival enlargement has been reported to be higher in patients treated with CCBs in combination with other HTN medication and CCBs. Newer generation of CCBs showed less severe gingival enlargement [6]. The mechanism and etiology of CCB induced gingival enlargement are still not completely understood, but it has been reported that the presence of local factors, dental plaque and inflammation might be a significant risk factor [7]. Many surgical approaches are beneficial, however osseous resective modality had optimal periodontal pocket reduction with severe cases [8]

### Conclusion

Both non-surgical and surgical therapy can be used successfully to treat patients with drug-induced gingival enlargement when non-surgical therapy and improved plaque control are insufficient. Although dramatic results can be obtained using non-surgical treatment, sometimes patients require surgical intervention [9]. Surgical approach to gingival enlargement include gingivectomy, open flap debridement and/or osseous surgery depending on the level of inflammation and presence of bony defects and exostosis. Patients with this condition must be on a strict periodontal maintenance and good oral hygiene, especially if medical consultation does not allow the change of hypertensive medications. The life quality of these patients may be raised by frequent recall appointments and maintaining periodontal health.

### Conflict of Interest and Source of Funding Statement

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