CASE REPORT

GINGIVAL HYPERPLASIA ASSOCIATED WITH AMLODIPINE: CASE REPORT

TRATAMENTO DA HIPERPLASIA GENGIVAL ASSOCIADA AO USO DE ANLODIPINO: RELATO DE CASO

Giovanna Tofani Baer Beraldo[1], Luiz Eduardo Monteiro Dias da Rocha[1]

ABSTRACT

Plaque-induced gingival hyperplasia modified by medication has been associated with certain pharmacological agents, including amlodipine, a dihydropyridine-class antihypertensive. This condition is characterized by excessive gingival overgrowth, which compromises both oral hygiene and aesthetics. The objective of this study was to report the integrated therapeutic approach in the management of severe gingival hyperplasia associated with amlodipine use, following the change of medication by the cardiologist in a male patient, specifying the diagnosis, treatment plan, and clinical outcome. Following the change in medication, the patient exhibited a reduction in gingival hyperplasia, supported by dental biofilm control and supragingival and subgingival scaling. After reevaluation, gingivectomy and gingivoplasty were performed, though to a lesser extent. The patient was advised on the importance of routine oral hygiene for maintaining periodontal health. The case underscores the importance of integrated medical and dental management in effectively reducing gingival hyperplasia and minimizing the extent of surgical intervention. The therapeutic approach—comprising basic periodontal therapy, oral hygiene instruction, and periodontal surgery—resulted in significant clinical improvement, meeting both aesthetic and functional patient needs.

Keywords: Gingival hyperplasia; Amlodipine; Gingivoplasty; Dental biofilm; Calcium channel blockers; Arterial hypertension.

RESUMO

A hiperplasia gengival induzida pela placa e modificada por medicamentos pode estar associada a certos fármacos, entre eles o anlodipino, um anti-hipertensivo da classe das di-hidropiridinas. Essa condição se manifesta pelo crescimento gengival excessivo, que compromete a higienização e estética do paciente. O objetivo deste estudo foi relatar a conduta terapêutica integrada ao manejo da hiperplasia gengival severa associada ao uso de anlodipino, após a mudança de medicamento pelo cardiologista, em um paciente do sexo masculino, especificando diagnóstico, plano de tratamento e evolução clínica. O paciente apresentou redução do crescimento gengival após a alteração da medicação pelo cardiologista, controle do biofilme dental, raspagem supragengival e subgengival. Após a reavaliação do tratamento inicial, realizou-se gengivectomia e gengivoplastia, mas em menor extensão. O paciente foi alertado quanto à importância da higiene oral rotineira para manutenção da saúde periodontal. Conclui-se que o manejo eficaz envolvendo a integração médica e odontológica foi essencial na condução do caso clínico, reduzindo a hiperplasia e a extensão da necessidade de correção cirúrgica. A abordagem terapêutica, incluindo terapia periodontal básica, instruções de higiene oral e cirurgia periodontal, resultou em melhora clínica significativa, atendendo às demandas estéticas e funcionais do paciente.

Palavras-chave: Hiperplasia gengival; Anlodipino; Gengivoplastia; Biofilme dentário; Bloqueadores dos canais de cálcio; Hipertensão arterial.

[1] Department of Integrated Clinical Procedures, Periodontics, School of Dentistry, Universidade do Estado do Rio de Janeiro, Rio de Janeiro, RJ, Brazil.

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INTRODUCTION

Dental biofilm-induced gingivitis constitutes a highly prevalent disease worldwide (1). According to Murakami *et al*. (2), plaque-induced gingival diseases present with clinical signs and symptoms of inflammation restricted to the gingival margin and the attached gingiva as the main characteristics, without extending beyond the mucogingival junction. Inflammation can be minimized by effective biofilm removal, since the abundant presence of microorganisms in bacterial plaque favors the onset and worsening of the disease process (3).

Factors such as hormonal alterations, use of specific medications (e.g., anticonvulsants, antihypertensives, and immunosuppressants), hyposalivation, systemic diseases, smoking, age, and genetic predisposition can influence this condition (2).

Drug-induced gingival hyperplasia is a common side effect associated with medications used to treat a variety of systemic conditions (3). Notable among these medications is nifedipine, considered the most often related to gingival hyperplasia (4). Amlodipine, an agent widely used to manage systemic arterial hypertension and angina pectoris, can also induce gingival growth. Studies indicate that 1.7 to 3.3% of patients who use amlodipine may develop gingival hyperplasia, characterized by excessive tissue growth on the dental crown which hinders proper oral hygiene and dental biofilm control (4).

Seymour *et al*. (5) reported the first documented case of gingival hyperplasia associated with amlodipine use in 1994. Several risk factors, including age, gender, genetic predisposition, and local oral hygiene conditions, can influence the severity of the clinical condition, the effective therapeutic management of which requires a multifaceted approach, considering both aesthetic and functional aspects, to restore periodontal health (6-8). When associated with amlodipine, biofilm-induced gingivitis can present symptoms such as gingival bleeding, edema, masticatory functional impairment, aesthetic impairment and, in advanced cases, bone loss and tooth mobility (9).

Also known as amlodipine besylate, the medication is an antihypertensive drug from the class of calcium channel blockers, more specifically dihydropyridine, widely used to treat hypertension, arrhythmias and angina pectoris (10). Lafzi *et al*. (4) reported that amlodipine dosage can influence the occurrence of gingival hyperplasia, and that patients taking 10 mg/day are more susceptible to this condition than those taking 5 mg/day. In these cases, patients report a gradual and painless enlargement of the gingiva, usually starting in the papilla within three weeks of starting treatment (4).

Drug-induced gingival hyperplasia is associated with two main mechanisms: the inflammatory and non-inflammatory pathways (11). The non-inflammatory mechanism occurs due to abnormal activity of the enzyme collagenase, associated with increased folic acid levels, blockage of the aldosterone hormone production axis by the renal cortex, and increased levels of adrenocorticotropic hormone (ACTH). In turn, the inflammatory mechanism relates to the deposition of the drug in the gingival fluid, which, in the presence of dental plaque, promotes an increase in inflammatory mediators, resulting in hyperplasia (10).

Treatment involves replacing the antihypertensive medication, under medical guidance, and biofilm control by a dentist. The latter includes supragingival and subgingival instrumentation, and detailed oral hygiene instructions for patients. In cases in which these measures do not lead to a satisfactory clinical response, periodontal surgery may be indicated. However, failure in effective biofilm control and drug replacement pose a significant risk of gingival growth recurrence, compromising treatment stability (12).

Given this scenario, this study reports on an integrated therapeutic approach to managing severe gingival hyperplasia associated with amlodipine use after a medication change prescribed by the cardiologist, specifying diagnosis, treatment plan and clinical evolution.

CASE REPORT

A 76-year-old Black male patient presented to the Periodontics Specialization Clinic of the Universidade do Estado do Rio de Janeiro, referred by the Stomatology Service of the Piquet Carneiro Polyclinic. The chief complaints were gingival bleeding, tooth mobility and increased gingival volume.

During anamnesis, the patient reported having arterial hypertension and taking the following medications on a daily basis: losartan 50 mg, amlodipine 10 mg/day and indapamide 1.5 mg. He denied other systemic diseases and any history of allergies. He reported being a smoker, consuming an average of three cigarettes per day, and consuming alcoholic beverages socially. Blood pressure was measured at 120 x 90 mmHg.

Clinical examination revealed complete edentulism in the maxillary arch and, in the mandibular arch, teeth 34, 33, 32, 31, 41, 42, 43 and 44 accompanied by severe gingival hyperplasia in the region (Figure 1).

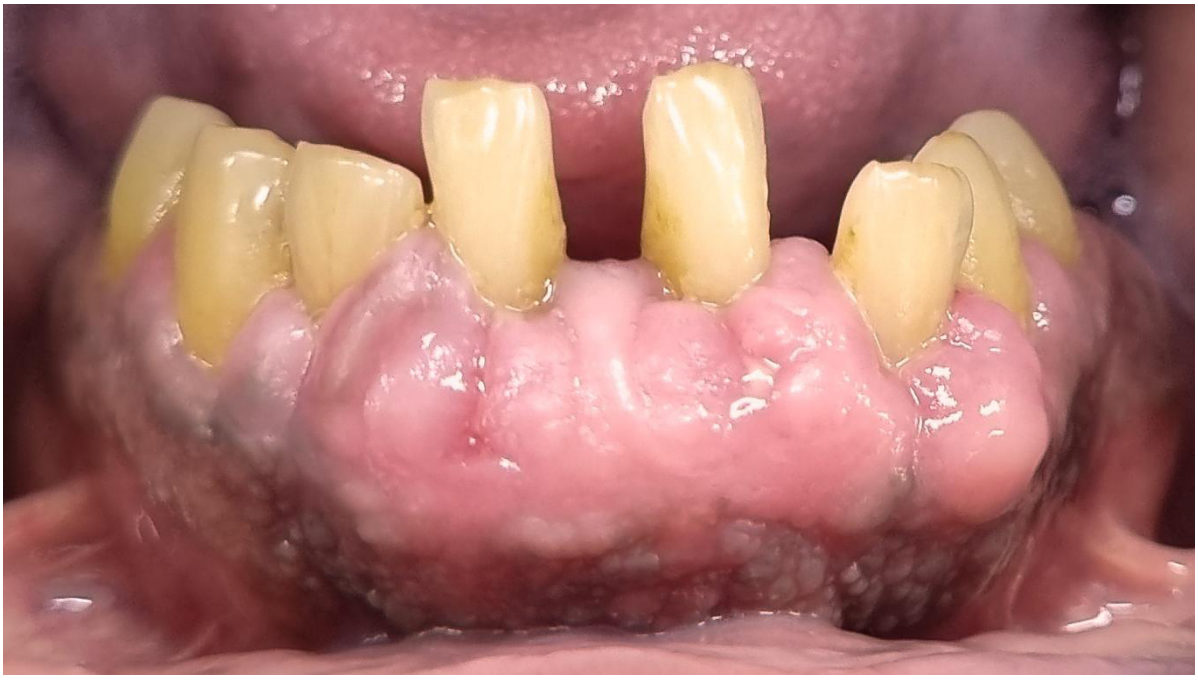

Figure 1 - Initial photograph of the lower arch of the reported clinical case, showing gingival hyperplasia.

Periodontal examination revealed pseudopockets in teeth 33, 32, 43, and 44, and periodontal pockets in teeth 31, 41, and 42, with the greatest depth recorded being 15 mm in tooth 31. We observed bleeding on probing in all evaluated sites, grade 2 mobility in teeth 31 and 41, and supragingival biofilm on all surfaces, especially in the cervical region close to the gingival margin (Figure 2).

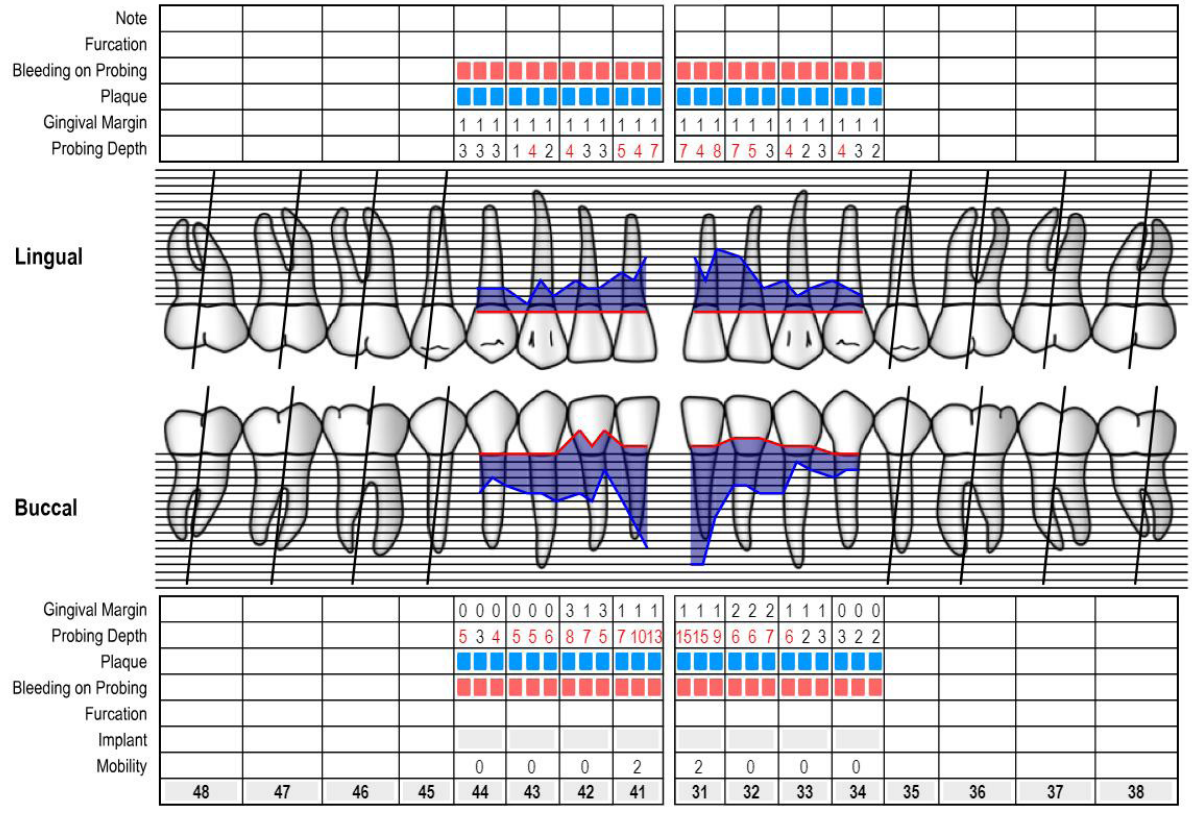

Figure 2 - Digital periodontal chart recorded in the patient's initial examination.
Source: The author (2024). Available at: https://www.periodontalchart-online.com/pt/.

Diagnosis and elaboration of the treatment plan required the following complementary tests: panoramic radiography and complete periapical radiography. Laboratory tests included complete blood count, prothrombin time (PT), activated partial thromboplastin time (APTT), C-reactive protein (CRP), and glycated hemoglobin (HbA1c), to evaluate systemic conditions that could interfere with periodontal and surgical treatment.

Radiographic analysis revealed mild horizontal bone loss in teeth 34, 33, 32, 43 and 44, and severe vertical bone loss in teeth 31, 41 and 42. Laboratory tests showed an increase in CRP (12 mg/L) and HbA1c (6.2%) levels.

Diagnosis of generalized stage IV periodontitis, grade B was established based on the 2018 Classification of Periodontal Diseases and Conditions. The established treatment plan included: disclosing dental plaque, oral hygiene instruction and reinforcement of brushing technique at all appointments; a single session of supragingival instrumentation and prophylaxis on all teeth; and subgingival instrumentation on teeth 32, 31, 41 and 42.

Additionally, the patient was referred to the surgery clinic for removal of the root remnants corresponding to teeth 35 and 45 and extraction of teeth 31 and 41. A referral to a cardiologist was also requested to assess the possibility of replacing the antihypertensive drug amlodipine.

After supragingival and subgingival instrumentation, the patient was scheduled for reassessment within 60 days. At the follow-up appointment, after non-surgical periodontal therapy, he reported that the antihypertensive medication (amlodipine) had been replaced with methyldopa 250 mg, as prescribed by the cardiologist. Modified Bass brushing technique was introduced to improve microbial control along the gingival margin. As a result, a significant improvement in the clinical picture of gingival hyperplasia was observed at this follow-up appointment (Figure 3). However, considering the aesthetic demands and the need for feasibility for prosthetic rehabilitation, we decided to plan a gingival plastic surgery in the region of teeth 33 to 43.

The patient returned after extraction of teeth 31 and 41. A reddish papule of approximately 3 mm, with an irregular surface and sessile base, was identified near the mucogingival junction, located on the vestibular attached gingiva of the area corresponding to tooth 32 (Figure 3). Although the lesion presented clinical characteristics compatible with fibrous hyperplasia, an intraoperative biopsy was indicated during gingival plastic surgery for histopathological confirmation.

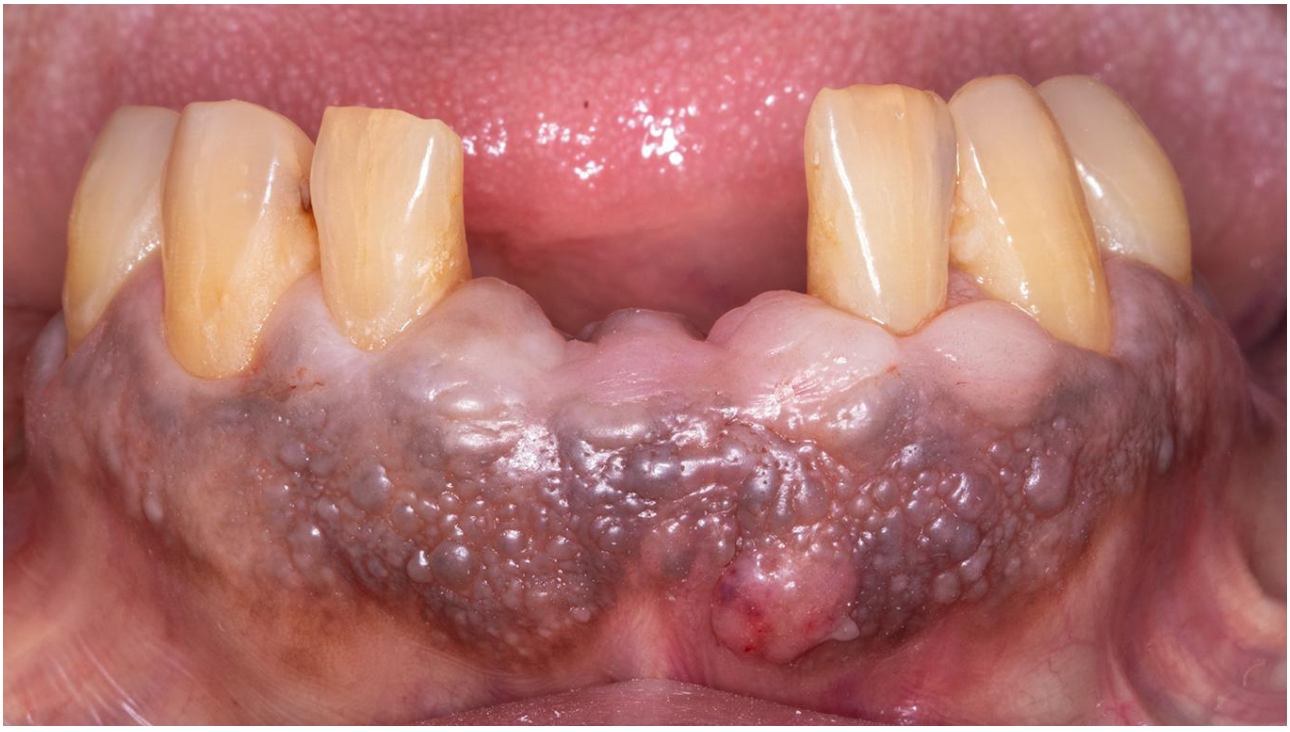

Figure 3 - Initial preoperative photograph, after changing hypertensive medication and non-surgical periodontal treatment: frontal view. A sessile-based papule is noted in the gingival area, inserted mesially to tooth 32, near the mucogingival junction.

New laboratory tests were requested prior to surgery, which showed a reduction in CRP levels from 12 to 6 mg/L. Gingival plastic surgery was performed in the region of teeth 34 to 44, using the gingivectomy and gingivoplasty technique, associated with excisional biopsy of the hyperplastic lesion located in the vestibular attached gingiva of the area corresponding to tooth 31 (Figure 4). The biopsy was sent to the oral pathology laboratory at the Universidade do Estado do Rio de Janeiro for histopathological analysis.

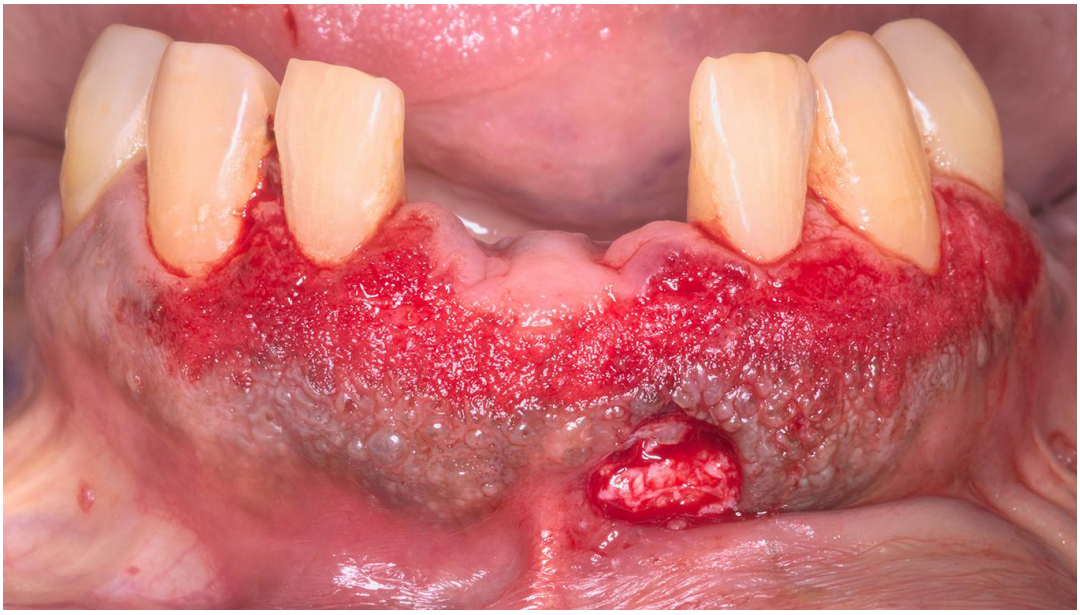

Figure 4 - Intraoperative photograph: gingivoplasty and excisional biopsy of the lesion located in the mesial region of tooth 32.

The patient was followed up at post-surgical check-ups at seven and 41 days after the procedure (Figure 5). Biopsy diagnosis confirmed the suspicion of inflammatory fibrous hyperplasia, and the clinical picture showed considerable aesthetic improvement, allowing the patient to be referred to the prosthodontics clinic for subsequent rehabilitation. During follow-up appointments, oral hygiene instruction was reinforced, emphasizing to the patient that his condition depends on daily home control of dental plaque, since a lack of cooperation can lead to a recurrence of the inflammatory and hyperplastic condition.

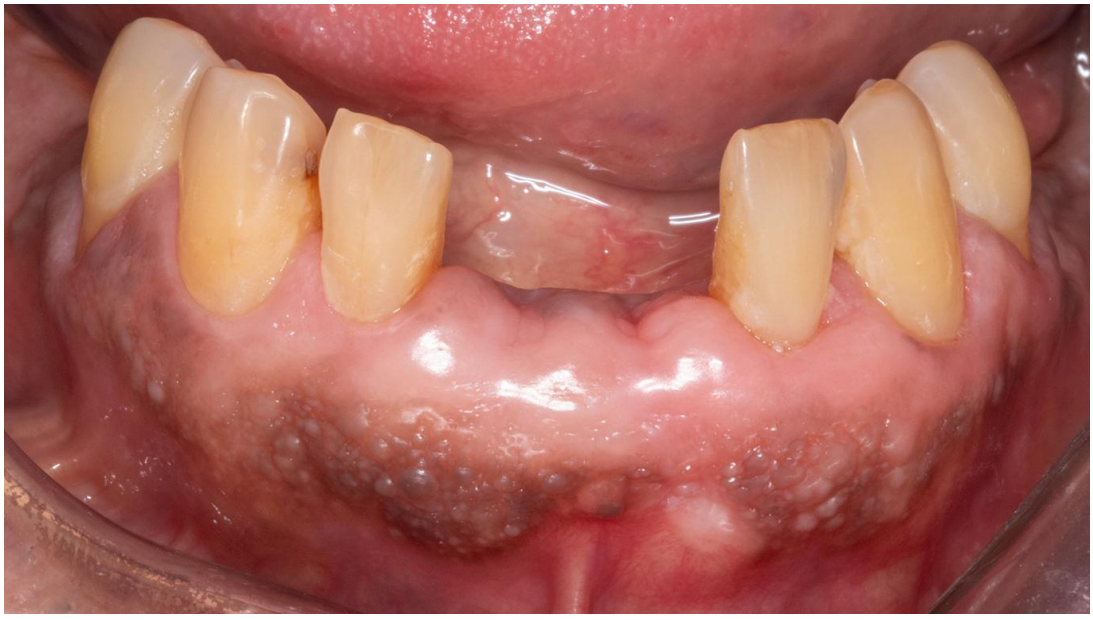

Figure 5 - Postoperative photograph at 41 days: frontal view. The keratinized mucosa appears pinkish, healthy, and in the final stage of maturation.

DISCUSSION

Plaque-induced and drug-modified gingivitis is influenced by several factors, including the class of drugs used, the dose administered, and treatment duration. Modifying factors such as gender, age, genetic predisposition and, crucially, the presence of bacterial plaque, are determinants of clinical severity (1,13). The patient in this report was receiving the highest recommended dose of amlodipine, 10 mg, until the attending physician changed his medication to methyldopa 250 mg. According to Bakshi *et al.* (12), men have a 3.3-fold higher risk of developing gingival hyperplasia compared with women. Additionally, continued use of 5 mg of amlodipine for a prolonged period is associated with the onset of gingival hyperplasia. According to Lafzi *et al*. (4), patients who use 10 mg/day of amlodipine tend to present this condition more quickly, usually about three months after starting treatment.

Amlodipine, a calcium channel blocker of the dihydropyridine class, can concentrate in gingival fluid (14). This mechanism is associated with the inflammatory pathway involved in gingival hyperplasia induced by bacterial plaque and exacerbated by the medication. In the reported case, the change in medication performed by the cardiologist, combined with the removal of supragingival and subgingival biofilm and rigorous control of bacterial plaque, resulted in a significant improvement in the host's inflammatory response, with a consequent reduction in gingival hyperplasia.

Studies by Pavlic *et al*. (15), Andrade *et al.* (14) and Bakshi *et al*. (12) described gingival hyperplasia as a benign and painless condition, characterized by thickening of the keratinized attached gingiva and interdental papillae, frequently associated with edema, although atypical presentations may occur (12,14-16). Histopathological confirmation of this condition can be obtained through biopsy, showing the presence of fibrous connective tissue with predominantly lymphocytic inflammatory infiltrate without malignant characteristics (11). *In vitro* studies corroborate these findings, showing that amlodipine can directly influence human gingival fibroblasts (17). More recent studies continue to explore the molecular mechanisms involved, seeking new perspectives for clinical management (18).

According to Murakami *et al*. (2), treating gingival hyperplasia begins with basic periodontal therapy. The literature shows that, in many cases, the combination of drug replacement with rigorous non-surgical plaque control can lead to complete resolution of the condition, avoiding the need for surgical intervention (19). This approach combined, whenever possible with medication substitution by the attending physician, represents the first line of treatment. However, as reported by Goiris *et al.* (10), Lafzi *et al.* (4) and Madi *et al*. (3), procedures such as gingivectomy and gingivoplasty are necessary to meet aesthetic and functional demands (4,3,10). More recent surgical approaches, such as the use of high-power laser, have also proven effective as a therapeutic option (20). In the reported case, the patient showed a significant improvement in gingival hyperplasia after changing medication and controlling dental biofilm. Thus, the need for surgical correction remained, albeit to a lesser extent.

Nonetheless, recurrence may occur if plaque control is neglected. In the reported case, surgery was performed to improve the aesthetic conditions of the affected region and facilitate the hygiene of the affected area by removing excess gingival tissue. The patient was informed about the importance of controlling supragingival plaque to maintain periodontal health and prevent recurrence of gingival hyperplasia.

CONCLUSIONS

Effective management of gingival hyperplasia associated with amlodipine use required an integrated approach between the medical and dental teams, underscoring the importance of controlling bacterial plaque and replacing antihypertensive medication, under medical guidance. Proper diagnosis combined with a therapeutic approach including basic periodontal therapy, oral hygiene instructions, and periodontal surgery (gingivectomy and gingivoplasty), resulted in significant clinical improvement, meeting the aesthetic and functional demands of the patient. Thus, the relevance of this case lies in the evidence provided regarding the need for interdisciplinarity in the differential diagnosis of drug-induced oral diseases, reinforcing that drug substitution must be accompanied by strict biofilm control for treatment success.

Corresponding author:
Giovanna Tofani Baer Beraldo. Boulevard. 28 de setembro, 157, Vila Isabel – Rio de Janeiro, RJ, Brasil. CEP: 20551-030.
E-mail: giovannatbb@gmail.com.

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