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A Rare Case of Severe Amlodipine-Induced Gingival Overgrowth: A Case Report

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A Rare Case of Severe Amlodipine-Induced Gingival Overgrowth: A Case Report

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Abstract

Gingival overgrowth, or gingival hyperplasia is the hypertrophy of periodontium caused by local systemic diseases or medications. These medications can be broadly categorized into three main groups including immunosuppressants, anticonvulsants, and calcium channel blockers (CCBs). Amlodipine-induced gingival overgrowth (AIGO) is a less documented phenomenon. This is a unique case of 68-year-old African American woman who developed severe AIGH after taking amlodipine for three years for essential hypertension. The gingival overgrowth happened over a period of two weeks and was so extreme that the patient was unable to approximate her lips or tolerate any oral intake. Labs, images, and biopsies were performed to rule out any other cause of the gingival overgrowth and were negative. AIGO was suspected, and the patient's amlodipine was stopped. At the three-month follow-up, full resolution was noted.

Keywords

Gingival hyperplasia, amlodipine, calcium channel blocker, gingival overgrowth, gingival, hyperplasia, antihypertensive

Conflict of Interest Statement

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Cover Page Footnote

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Background

Gingival overgrowth, or gingival hyperplasia, is the hypertrophy of the periodontium, which can be caused by medications or local systemic diseases. The most common medication classes with documented gingival overgrowth are anticonvulsants (e.g., phenytoin) and immunosuppressants (e.g., cyclosporine). Less frequently, cases of gingival hyperplasia have been found with calcium channel blocker (CCB) use (nifedipine, amlodipine). It is estimated that 50% of adults treated with phenytoin, 30% with cyclosporin, and 20% with nifedipine experience gingival enlargement.[1] The prevalence of amlodipine-induced gingival hypertrophy is between 1.7% and 3.3%.[2] We present a case of amlodipine-induced gingival hyperplasia.

Case Presentation

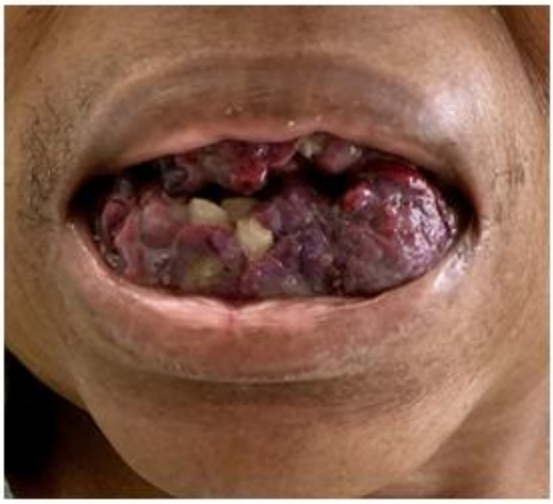
A 68-year-old African American female presented to the Emergency Department (ED) with symptoms of profuse gingival bleeding while brushing and significant gingival swelling with pain (Figure 1, A). The patient reported that she had initially noticed discomfort for one month and visited her dentist 2 weeks prior to ED presentation with mild symptoms and pain. The dentist diagnosed her at that time with periodontitis and treated her with antibiotics. Her symptoms continued to worsen over the next 2 weeks, and the dentist instructed the patient to go to the ED. The patient reported no significant medical history other than hypertension, gastroesophageal reflux disease (GERD), and arthritis, which she has taken lisinopril 5 mg, amlodipine 10 mg, and pantoprazole 20 mg for the past three years with no recent changes. The patient's CBC, CMP, blood cultures, TSH, RF, ANA, and HIV were negative or within normal limits. The ESR and CRP were elevated and thought to be secondary to the acute inflammation. The computerized tomography (CT) of neck and soft tissue showed severe odontogenic disease with extensive thickening of the mucosa around the teeth consistent with interval overgrowth without evidence of abscess or infection based on patient's history (Figure 1, B), though no information from the dentist was available at the time of ED evaluation.

An Otorhinolaryngology (ENT) consultant examined the patient and preliminarily diagnosed her with Kaposi's sarcoma based on clinical presentation with violaceous discoloration (Figure 1, B). Patient was prescribed dexamethasone 5 mg daily. ENT performed an excisional biopsy for confirmation of Kaposi's sarcoma; however, the biopsy showed squamous mucosa with features consistent with gingival overgrowth with subjacent hematoma, negative HHV-8 immunoperoxidase stain, and no features of dysplasia or malignancy (Figure 2, A&B). Patient's history was reviewed again, and the decision was made to discontinue amlodipine as a potential cause of her gingival hyperplasia. She received seven days of steroids with close monitoring, resulting in significantly reduced gingival swelling and pain.

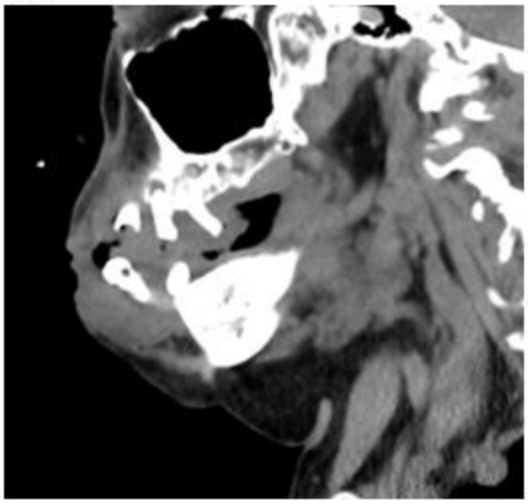
Three months later, the patient reported to the resident out-patient clinic for follow-up. At that time, her gingival overgrowth had fully resolved (Figure 1, C). Notably, several of her teeth had been extracted by her dentist due to poor oral hygiene and unrelated to the hyperplasia.

Figure 1. Initial presentation with computed tomography scan and after three months of discontinued amlodipine.

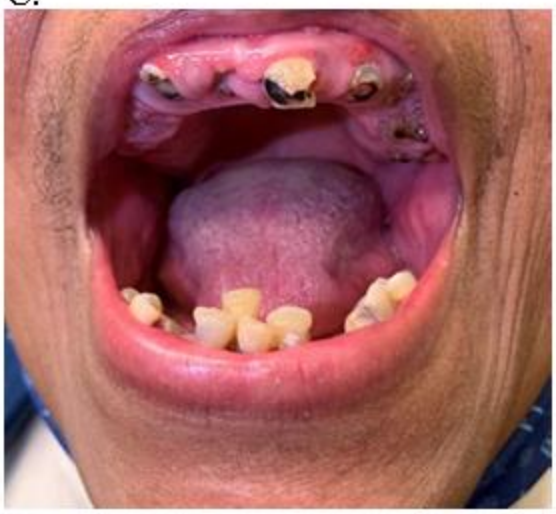
A.



B.



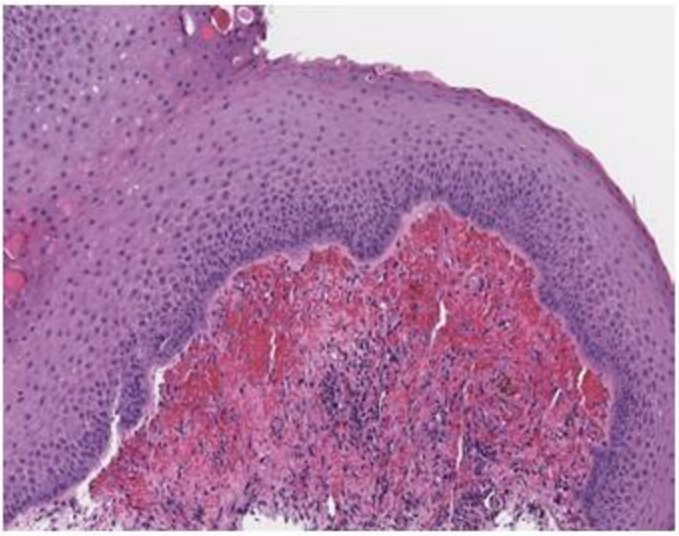
C.



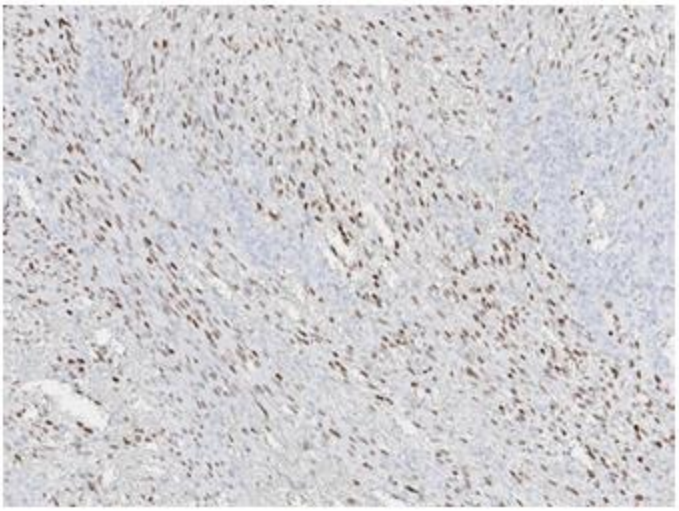
A: Initial presentation with gingival hyperplasia.
B: CT neck and soft tissue showing odontogenic disease with extensive thickening of the mucosa around the teeth consistent with interval hyperplasia.
C: Three months after discontinuation of amlodipine.

Figure 2. Histology.

A.



B.



A: Hematoxylin eosin stain showing squamous mucosa with mild acanthosis and subjacent hematoma formation consistent with gingival hyperplasia.

B: Negative HHV-8 immunoperoxidase stain, without features of dysplasia or malignancy.

Discussion

The differential diagnosis for gingival overgrowth/hyperplasia includes Kaposi sarcoma, acute myelogenous leukemia (most commonly M4 and M5 subtypes), Wegener's granulomatosis, sarcoidosis, tuberculosis, medication-induced, plaque-induced, and hereditary gingival fibromatosis. While medications are the most common cause of gingival overgrowth, it is important to take a detailed history and to evaluate systemic diseases as a cause. Gingival overgrowth induced by CCBs can present between two to fourteen months after treatment initiation, but in this case, the adverse reaction did not occur until three years after the introduction of the medication.[3] The pathogenesis of CCB-induced gingival overgrowth is unclear, but there are several proposed theories. According to experimental data, CCBs inhibit both the dihydropyridine and nondihydropyridine binding sites.[4] If inhibiting dihydropyridine binding sites contribute to gingival hyperplasia, this could possibly explain why more cases have been reported with nifedipine, which is a dihydropyridine calcium antagonist. Furthermore, the gingiva is composed of epithelial and connective tissues, where the epithelial tissue follows a strongly regulated scheme at the cellular level to function as a barrier.[5] Within the complex regulation, influx of calcium contributes to the anti-inflammatory process.[5] The combination of poor oral hygiene and CCB may contribute to increased risk of gingival hyperplasia. More studies are required to investigate the pathophysiology of gingival overgrowth due to CCBs.

Physicians and dentists should be aware of the etiologic medications that can lead to gingival overgrowth and be able to identify early changes in periodontal tissues; early treatment, such as stopping and substituting caustic medications, can potentially prevent permanent periodontal damage and tooth loss. CCB-induced gingival hyperplasia should be considered, despite low incidences, as CCBs are commonly prescribed and immunohistochemical studies can aid in its diagnosis. Once diagnosed, interdisciplinary work between medical and dental health care professionals are critical in minimizing damages and optimizing outcomes in those patients.

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