CASE REPORT

Amlodipine-induced gingival hyperplasia

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ABSTRACT

Drug-induced gingival hyperplasia is the increased growth of gingival epithelium and submucosa which may cause bleeding and loss of teeth. Certain anticonvulsant, immunosuppressive, and antihypertensive drugs are the underlying cause in most cases. Among them, cases due to amlodipine are very rare. Here, we discuss the topic along with a case of gingival overgrowth induced by amlodipine. A 51-year-old female presented with gingival overgrowth. She was on amlodipine because of primary hypertension. She had gingival hyperplasia increasingly growing in the last few months. Her antihypertensive regimen was changed with cessation of amlodipine. This study suggests that amlodipine may lead to gingival hyperplasia.

KEY WORDS: Gingival Hyperplasia; Calcium Channel Blockers; Amlodipine

INTRODUCTION

Drug causing gingival hyperplasia is in the form of enlargement along the gingival epithelium and submucosal connective tissue. Gingival enlargement is apparent in gingiva below lips and in maxillary labial mucosa. If not treated, it can result in gingival bleeding and loss of teeth.¹ Drug-associated gingival overgrowth basically arises from three groups of drugs: Anticonvulsants, calcium channel blockers (CCB), and immunosuppressive drugs. Gingival hyperplasia has estimated to occur in 25% of cyclosporine and phenytoin users, in 5–15% of nifedipine users and in 5–10% of non-dihydropyridine CCB users.²³

Amlodipine-induced gingival overgrowth is rarer and estimated to have a frequency of 3.3%.⁴⁵ Since CCB and immunosuppressive drugs are commonly used in the practice of nephrology and internal medicine, we wanted to discuss the topic on a rare case in which we detected amlodipine induced gingival hyperplasia.

CASE REPORT

A 51-year-old female had been using amlodipine tablets (10 mg/day) for 8–9 months because of hypertension. She did not have regular blood pressure monitoring. No medical history, she never used tobacco or alcohol. Her familiar history not significant. After 8–9 months of taking tab amlodipine she noticed gingival overgrowth [Figure 1]. She did not see a physician regularly and continued to use her medications. During the last month, gingival overgrowth appeared and increased. Gingival bleeding was not developed. However, the patient did not have teeth loss. In physical examination, her cooperation was full and following measurements were recorded: Body mass index 27 kg/m², blood pressure 170/80 mmHg, and pulse: 78/min., number of breath 16/min. She had gingival hyperplasia and dental plaques in oral examination. No feature was detected in pulmonary and abdominal examinations. Laboratory tests were as follows: Serum creatinine 1 mg/dl; Na 140 mEq/l; K 3.7 mEq/l;
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uric acid 6.2 mg/dl; C reactive protein 4.9 mg/l; glucose 110 mg/dl; Ca 9.9 mg/dl; P 3.2 mg/dl; albumin 4.6 g/dl; alanine aminotranspherase 18 U/l; low-density lipoprotein 101 mg/dl; triglyceride 147 mg/dl; thyroid stimulating hormone 0.83 uIu/ml; hemoglobin 12.8 g/dl; leukocytes 8900/mm3; and platelets 239,700/mm3. Moreover, no feature was observed in urine microscopy. EKG was in accordance with normal sinus rhythm and heart rate was 78/min.

The case was considered to be amlodipine induced gingival hyperplasia. After consultation with the department of dentistry, amlodipine administration was discontinued and new antihypertensive regimen was arranged as telmisartan 40 mg/day. Dental recovery was observed in follow-up visits [Figure 2]. Blood pressure was within targeted range. The patient gave her informed consent.

DISCUSSION

Drug causing gingival hyperplasia was first defined in cases receiving antiepileptic treatment with phenytoin and phenobarbital and in cases receiving immunosuppressive treatment with cyclosporine. In addition to phenytoin and phenobarbital, valproic acid, carbamazepine, and vigabatrin are antiepileptics occasionally reported to cause gingival hyperplasia. Phenytoin causing gingival hyperplasia is especially seen in patients with dental plaques and gingivitis. Cyclosporine related cases are also observed in patients with an inflammatory basis who are taking higher doses and disappear when the drug is discontinued. Drug causing gingival hyperplasia could also be associated with antihypertensive drugs. This side effect have been seen with dihydropyridines, occasionally with amlodipine. It has been reported that use of nifedipine together with cyclosporine augments cyclosporine related gingival hypertrophy. Gingival hyperplasia may arise with non-dihydropyridines (diltiazem and verapamil).

Etiology of drug-induced gingival hyperplasia has been considered to be multifactorial. The potential risk factors are poor oral hygiene, periodontal disease, periodontal pocket size, gingival inflammation, presence of dental plaques, and dose and duration of cyclosporine use. Apart from these poor oral hygiene and presence of dental plaques are important which result in gingival inflammation and accumulation of drugs such as phenytoin and cyclosporine and of some metals such as nickel in dental plaques. Keratinocytes and fibroblasts in gingival mucosa were found to be prone to the accumulation of drugs such as phenytoin and nifedipine. It was reported, phenytoin causing gingival overgrowth is more common in young age groups and CCB related gingival hyperplasia is more common in males. Effects of demographic features of the cases and the dose of the drug on the pathogenesis are not clear. It is understood that in the presence of gingival inflammation, use of drugs lead to proliferation in sensitive epithelium and fibroblasts. In addition to inflammation and bacterial plaques, non-inflammatory processes were also proposed to contribute to pathogenesis of gingival overgrowth. Among them are impaired collagenase activity related to folate deficiency and increased keratinocyte growth factor which could arise as a result of decreased aldosterone synthesis other systemic diseases such as Leukemia, Vitamin C deficiency, Wegener’s Granulomatosis, Crohn’s disease, Sarcoidosis, plasma cell gingivitis, and tuberculous gingival enlargement may also cause Gingival enlargement. Since drug related gingival hyperplasia is an entity that could only be demonstrated through case series, its prevalence was not precisely determined. As mentioned above amlodipine may be defined as notably rarer cause for gingival hyperplasia among other CCBs. Periodontists refer to oral hygiene for the occurrence of this severe side effect. As long as dental care is maintained, complaints are eliminated even when calcium antagonist use is continued.

CONCLUSION

Pathogenesis of drug related gingival hyperplasia is still not clear. However, it is known to occur in gingival inflammation background. Stop using the drug, oral hygiene, and
surgical removal of extra tissues are some of the treatment options.\[^{13,15}\] In our case, after changing amlodipine with other group antihypertensives and achieving proper oral hygiene, intraoral lesions declined noticeably.

If the diagnosis had been further delayed, surgical intervention (gingivectomy) would have been necessary. Current hypertension treatment guidelines recommend CCB to be a first option in combinations.\[^{16}\] It is important to keep in mind that these drugs used in everyday practice could cause gingival hypertrophy and inform our patients about this fact that oral hygiene and dental care could eliminate serious complications.

**REFERENCES**


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