Drug-induced gingival hyperplasia can be a serious concern for both patients and clinicians. Drug-induced gingival hypertrophy is a well-documented side effect of some pharmacologic agents, including, but not limited to, calcium channel blockers (CCBs), phenytoin, and cyclosporine. Amlodipine, a long-acting CCB, is a commonly used hypertension drug. Here we describe a case of amlodipine-induced massive gingival hyperplasia.

Case description
A 14-year-old boy was admitted for gingival swelling. He had received a renal transplant for focal segmental glomerulosclerosis in 2005. He also had a history of hypertension, which he had been treating with oral amlodipine (10 mg twice daily) for the past 3 years. His mother said that he had been suffering from increasing gum swelling for 2 years. There was no history of any other prescription drug use during this time. Examination of the oral cavity revealed substantial diffuse gingival hypertrophy of both the upper and lower gums. The hypertrophic gingiva were painless, and there was no sign of inflammation or ulceration. He brushed his teeth regularly and his oral hygiene was normal. The rest of the physical examination findings and laboratory test results were normal. After excluding other potential causes, we considered the diagnosis of amlodipine-induced massive gingival hypertrophy. We substituted an angiotensin receptor blocker for the amlodipine, and within 6 months the gingival hypertrophy had regressed completely.

Discussion
Amlodipine is a second-generation dihydropyridine CCB that can cause gingival hypertrophy. The prevalence of amlodipine-induced gingival hypertrophy has been shown to be between 1.7% and 3.3%. The incidence of gingival hypertrophy with nifedipine therapy has been reported to be as high as 20%, and a 2002 study reported that the prevalence with the use of CCBs might be as high as 38%. Gingival hypertrophy is 3.3 times more common in men than in women. The most common form is bacterial plaque–induced gingival disease, which presents as gingivitis. Use of phenytoin, cyclosporine, and CCBs, as well as vitamin C deficiency, can also cause the condition, as can hormonal shifts during

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pregnancy. The reason for this adverse event is not absolutely known, but mechanisms involving inflammatory and noninflammatory pathways have been suggested. For example, individual sensitivity to a drug’s metabolic pathway might be a trigger. Untreated gingival hypertrophy might lead to bleeding, infection, abscess, ulceration, cosmetic deficiency, and functional difficulty (eg, chewing, talking). Treatment of drug-induced gingival hypertrophy includes cessation of the drug and decreasing other risk factors with meticulous mechanical and chemical plaque control. Replacing the affecting drug with another agent is also recommended when possible. Gingivectomy (excision of excessive gingival tissue) should be reserved for severe cases that affect oral hygiene or functionality, or can be performed for cosmetic reasons.

Conclusion

Our patient’s amlodipine-induced massive gingival hyperplasia completely regressed after the amlodipine was replaced by an angiotensin receptor blocker. As amlodipine is a commonly prescribed hypertension drug in family practice, every physician should be aware of this usually overlooked but potentially harmful side effect, particularly if adverse oral symptoms arise during drug use.

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