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Amlodipine-Induced Junctional Bradycardia in an Elderly Patient with Resistant Hypertension: A Case Report

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Abstract

Amlodipine, a dihydropyridine calcium channel blocker, is widely prescribed for hypertension due to its vasodilatory effects and minimal influence on heart rate. However, rare cases of bradycardia have been reported, particularly in elderly patients with multiple comorbidities. We describe the case of an 84-year-old male with chronic resistant hypertension and stage III chronic kidney disease who developed asymptomatic junctional bradycardia while on amlodipine 10 mg daily as part of a multidrug antihypertensive regimen. Laboratory workup excluded electrolyte abnormalities, thyroid dysfunction, and myocardial infarction. Amlodipine was discontinued, resulting in resolution of bradycardia 48 hours later. This case highlights the potential for amlodipine to cause clinically significant bradycardia even at therapeutic doses, particularly in elderly patients with polypharmacy and impaired renal function. Early recognition may prevent unnecessary invasive interventions.

Keywords

Amlodipine, Bradycardia, Adverse Drug Reaction, Junctional Rhythm, Calcium Channel Blocker

1. Introduction

Amlodipine is a widely prescribed dihydropyridine calcium channel blocker (CCB) primarily used for the management of hypertension and angina pectoris. Its therapeutic efficacy stems from its ability to inhibit L-type calcium channels, leading to vasodilation of peripheral arteries and a subsequent reduction in blood pressure

[1]. Unlike non-dihydropyridine CCBs such as verapamil and diltiazem, which exert significant effects on cardiac conduction by acting on the sinoatrial (SA) and atrioventricular (AV) nodes, amlodipine generally exhibits minimal influence on cardiac electrophysiology due to its vascular selectivity [1].

Although bradycardia is a rare adverse effect, several case reports have described amlodipine-associated bradyarrhythmia, particularly in elderly patients with multiple comorbidities. We present the case of an 84-year-old male who developed junctional bradycardia while receiving amlodipine as part of a multidrug antihypertensive regimen. This case underscores the importance of considering amlodipine-induced bradycardia as a potential, though uncommon, reversible cause of bradyarrhythmia, particularly in vulnerable patient populations.

2. Case Presentation

An 84-year-old male with a past medical history significant for chronic resistant hypertension, hyperlipidemia, chronic kidney disease stage III, allergic rhinitis, hypothyroidism, impaired fasting blood glucose, testicular hypofunction, chronic anemia, gastroesophageal reflux disease, and osteopenia presented to the clinic for routine follow-up. During examination, he was found to have asymptomatic bradycardia with a heart rate in the low 30 s beats per minute range (bpm). Of note, prior to starting amlodipine, the patient's baseline heart rate ranged in the 70 bpm. Electrocardiogram (EKG) obtained at the office demonstrated junctional bradycardia (**Figure 1**). The patient reported no symptoms of dizziness, fatigue, syncope, or chest pain, and had been chronically bradycardic since initiation of amlodipine.

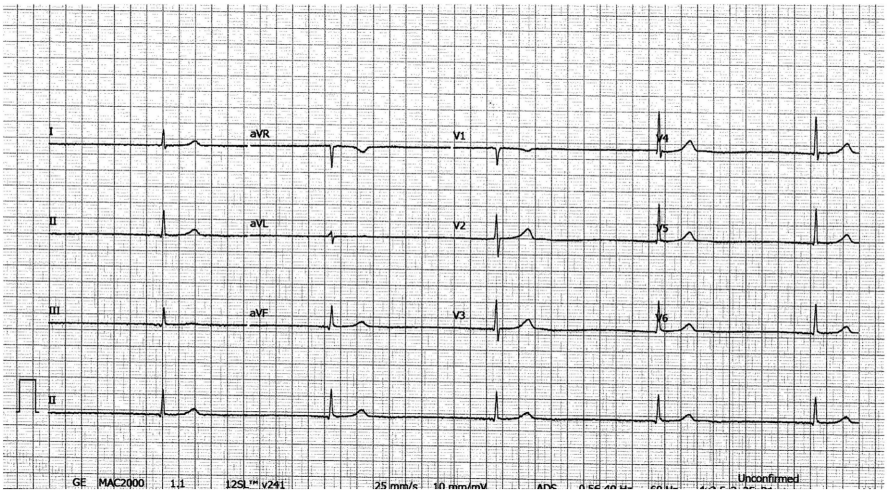

Figure 1. EKG prior to amlodipine being discontinued, showing junctional bradycardia with absent P waves.

The patient had non-contributory social and surgical history. He was a lifelong non-smoker but reported chronic alcohol consumption of 3 to 4 drinks daily. His antihypertensive regimen included aspirin 81 mg daily, hydrochlorothiazide 25 mg daily, doxazosin 2 mg daily, hydralazine 100 mg three times daily, lisinopril

40 mg twice daily, and amlodipine 10 mg daily. Additional medications included Vytorin (ezetimibe/simvastatin) 10/20 mg daily for hyperlipidemia, alendronate 70 mg weekly for osteopenia, levothyroxine 112 mcg daily for hypothyroidism, omeprazole 20 mg daily for GERD, bupropion XL 300 mg daily for depression, and intramuscular testosterone 100 mg weekly for hypogonadism. He was referred to the hospital for further evaluation of severe junctional bradycardia.

Initial laboratory studies prior to hospitalization demonstrated sodium 138 mEq/L, potassium 5.3 mEq/L, chloride 106 mEq/L, blood urea nitrogen (BUN) 32 mg/dL, and creatinine 1.79 mg/dL. Thyroid studies showed a thyroid-stimulating hormone (TSH) of 4.74 μIU/mL and free thyroxine (T4) of 7.7 ng/dL, both within normal limits.

An EKG obtained during admission demonstrated junctional bradycardia with absent P waves, narrow QRS complexes, and a ventricular rate of 30 beats per minute (**Figure 1**). Chest radiography revealed no acute cardiopulmonary findings, and the cardio-mediastinal silhouette was unremarkable. The attending cardiologist recommended permanent pacemaker placement; however, the patient declined invasive intervention, preferring conservative medical management. Amlodipine 10 mg was discontinued as it was the most recent addition to his hypertension regimen, and the patient was discharged with instructions for close outpatient follow-up.

At his 48-hour follow-up, repeat EKG demonstrated restoration of sinus rhythm with first-degree atrioventricular (AV) block (**Figure 2**) and a heart rate of 64 beats per minute.

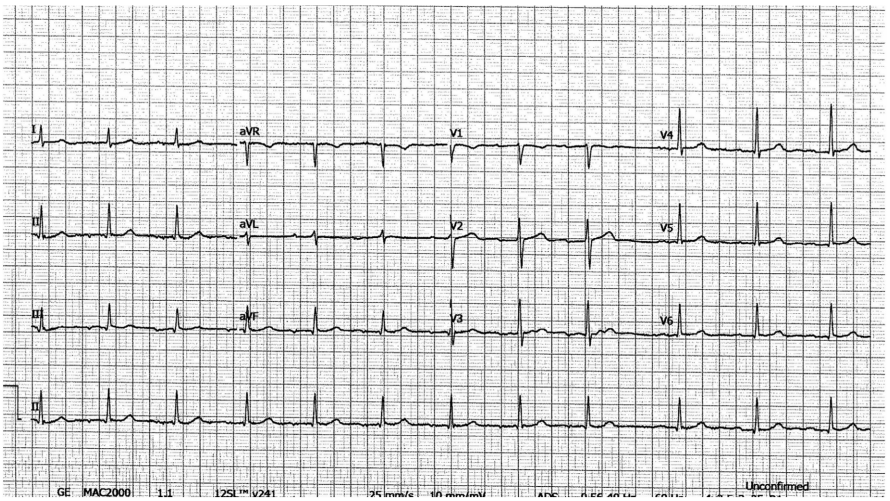

Figure 2. EKG after amlodipine was discontinued, showing sinus rhythm with sinus arrhythmia and first-degree AV block.

Approximately one month after discontinuation of amlodipine, follow-up laboratory studies revealed hyponatremia and hypochloremia with sodium 126 mEq/L and chloride 92 mEq/L, while BUN and creatinine had improved to 19 mg/dL and 1.11 mg/dL, respectively. The hyponatremia and hypochloremia were attributed

to the start of hydrochlorothiazide 25 mg twice a day, following the cessation of amlodipine to regulate his blood pressure. The patient has had no follow-ups since then to determine if the hyponatremia and hypochloremia have resolved. No repeat thyroid function tests were obtained at that time.

3. Discussion

Amlodipine primarily inhibits voltage-dependent L-type calcium channels, decreasing calcium influx into vascular smooth muscle and promoting vasodilation, which lowers blood pressure [1]. Unlike non-dihydropyridine CCBs such as diltiazem and verapamil, which act on the sinoatrial (SA) and atrioventricular (AV) nodes, amlodipine generally exerts minimal effects on cardiac conduction due to its vascular selectivity. However, in certain patients, particularly those with advanced age, renal dysfunction, or polypharmacy, its off-target effects may impair pacemaker cell activity, leading to the <1% but >0.1% of cases that present with bradycardia [2].

The patient's inpatient EKG (**Figure 1**) shows a junctional rhythm with absent P waves and significant bradycardia. The absence of P waves, in addition to the narrow QRS morphology at a rate of 30 beats per minute, suggests a junctional bradycardia. Although this deviates from the mechanism of action, the removal of amlodipine restored the SA node activity and resolved the patient's bradycardia, thus indicating that even at non-toxic levels, amlodipine can impact the pacemaker cells of the heart.

To further assess causality, we applied the Naranjo Adverse Drug Reaction Probability Scale, which yielded a score of 7, indicating a probable relationship between amlodipine and the observed adverse event [3] (**Table 1**). Hyponatremia and hypochloremia were only noted after the withdrawal of amlodipine; hence, these electrolyte disturbances could not have contributed to the bradycardia. According to the 2018 ACC/AHA guidelines, pacemaker placement for bradyarrhythmia due to sinus node dysfunction or AV blocks is indicated when the cause is irreversible, and no reversible causes are found [4]. This is an invasive procedure, and this case shows how medical management could potentially be as effective, even if the mechanism of action of dihydropyridines is not commonly associated with bradycardia.

Table 1. Naranjo adverse drug reaction probability scale.

Question	Response	Score
1) Are there previous conclusive reports on this reaction?	Yes	+1
2) Did the adverse event appear after the drug was administered?	Yes	+2
3) Did the adverse reaction improve when the drug was discontinued or a specific antagonist was administered?	Yes	+1
4) Did the adverse reaction reappear upon re-administration?	Not tested	0

Continued

5) Are there alternative causes that could have caused the reaction?	No	+2
6) Did the reaction reappear with placebo?	Not tested	0
7) Was the drug detected in any body fluid (e.g., blood)?	Not tested	0
8) Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	Not Tested	0
9) Did the patient have a similar reaction to the same or similar drugs in the past?	Not Tested	0
10) Was the adverse event confirmed by objective evidence?	Yes	+1
Total Score		**7**

Several prior reports have described bradycardia associated with amlodipine use, though it remains an uncommon adverse event. Ramadan and Quyyumi reported a 42-year-old woman who developed symptomatic bradycardia while taking 10 mg of amlodipine daily, which resolved within 72 hours of discontinuation [5]. Tu and Adjovu described a 71-year-old man with worsening fatigue, dizziness, and bradycardia that resolved within 24 hours after stopping amlodipine [6]. Similarly, Algahtani *et al.* reported a case of sinus bradycardia and junctional rhythm in a 60-year-old man on amlodipine, which improved after withdrawal of the medication [7]. Lee *et al.* also documented bradyarrhythmia associated with amlodipine in a patient with underlying renal impairment [8]. These cases, along with our report, suggest that elderly patients with multiple comorbidities and polypharmacy may be more susceptible to this rare but potentially reversible adverse effect.

Although amlodipine is not classically categorized as a potent AV nodal blocking agent, emerging reports highlight its potential role in precipitating BRASH syndrome (Bradycardia, Renal failure, AV nodal blockade, Shock, and Hyperkalemia) under the right clinical circumstances. In elderly patients with multiple comorbidities, particularly those with chronic kidney disease, even medications like amlodipine may contribute to a synergistic downward spiral. This patient, with stage III CKD and on a multidrug antihypertensive regimen including lisinopril and amlodipine, exhibited features consistent with an early or incomplete form of BRASH syndrome [9]. While overt hyperkalemia and shock were absent, the progressive bradycardia and renal dysfunction suggested a pathogenic loop potentially initiated by mild volume depletion or medication accumulation.

Early recognition and intervention, such as discontinuing contributory agents and supporting renal function, can often reverse the trajectory without invasive measures like pacing or dialysis. Clinicians should maintain a high index of suspicion in frail or elderly patients, where minor metabolic derangements and polypharmacy can amplify each other, triggering a cascade that may initially present subtly but

worsen rapidly if not addressed.

4. Conclusion

In this case, an 84-year-old male with resistant hypertension developed junctional bradycardia while taking amlodipine as part of a multidrug antihypertensive regimen. Discontinuation of amlodipine alone led to complete resolution of the bradycardia without requiring invasive intervention. Although amlodipine rarely affects cardiac conduction, this case highlights the importance of recognizing it as a potential reversible cause of bradycardia, especially in elderly patients with multiple comorbidities and polypharmacy. Early identification and medication adjustment may prevent unnecessary procedures such as pacemaker implantation.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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