A case of doxazosin-induced acute coronary syndrome in a patient with myocardial bridging

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Adv Interv Cardiol 2016; 12, 1 (43): 75–76
DOI: 10.5114/pwki.2016.56957

Introduction

Myocardial bridging (MB) is an anatomical variation characterized by narrowing during systole of some of the epicardial coronary arterial segments running in the myocardium. It can be encountered in 0.5% to 16% of routine coronary angiographies [1]. Although it is considered as a benign anomaly, it may lead to such complications as myocardial ischemia, acute coronary syndromes, coronary spasm, exercise-induced dysrhythmias such as supraventricular tachycardia, ventricular tachycardia, syncope, or even sudden death [2].

In this report, we present a previously unreported case of a 51-year-old man with doxazosin-induced acute coronary syndrome, who was diagnosed with myocardial bridging overlying the left anterior descending artery.

Case report

A 51-year-old man was admitted to the coronary intensive care unit due to chest pain and syncope without any prodromal symptoms after taking the first doxazosin dose. Blood pressure was 100/60 mm Hg and heart rate was 100 bpm at initial evaluation. He had had hypertension for over ten years. He was on losartan. The electrocardiogram showed sinus rhythm with biphasic T wave on precordial derivations and negative T wave in leads DI and aVL (Figure 1 A). High-sensitivity troponin T level was elevated (42 ng/l, 0–14 ng/l). Kidney function tests were normal. A transthoracic echocardiogram showed left ventricular ejection fraction of 60% with normal wall motion, left ventricular hypertrophy, and diastolic dysfunction. Coronary angiography revealed that myocardial bridging was confined to the left anterior descending artery (LAD) with severe systolic compression (90%) (Figures 1 B, C). Other coronary arteries were normal. Doxazosin was discontinued. The patient was initially treated with metoprolol and aspirin. Myocardial perfusion scintigraphy was found to be normal under β-blocker treatment. The protocol was performed at rest and during exercise, with 99mTc sestamibi. Cardiac enzyme level was decreased at follow-up. The patient was discharged with β-blocker and acetylsalicylic acid. He has been followed up without any symptoms for 1 month.

Discussion

Coronary arteries that tunnel through the myocardium are seen in as many as 40% to 80% of cases on autopsy; however, functional MB is less commonly observed on angiography (0.5% to 16.0%) [1]. Although it is considered as a benign anomaly, it may lead to such complications as myocardial ischemia, acute coronary syndromes, and coronary spasm. Various pathophysiological changes can induce symptoms of myocardial ischemia in previously asymptomatic patients. Aging, hypertension, coronary atherosclerosis, coronary vasospasm, microvascular dysfunction, endothelial dysfunction, plaque development proximal to the bridge, and negative remodeling can induce myocardial ischemia in patients with MB. Left ventricular hypertrophy might affect epicardial constriction through remodeling and growth of heart muscle covering the coronary artery. Left ventricular diastolic dysfunction may impair the diastolic flow time in the coronary artery.

Although percutaneous coronary intervention, myotomy or coronary artery bypass grafting can be considered for selected patients refractory to maximal medical therapy, for treatment of symptomatic patients with MB primarily pharmacological therapy is suggested [2].

In symptomatic patients, β-blockers relieve the hemodynamic disturbance caused by the MB by decreasing the heart rate, increasing the diastolic coronary filling period, and decreasing contractility and compression of the coronary arteries. In some cases calcium antagonists such as

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Received: 4.06.2015, **accepted:** 24.06.2015.

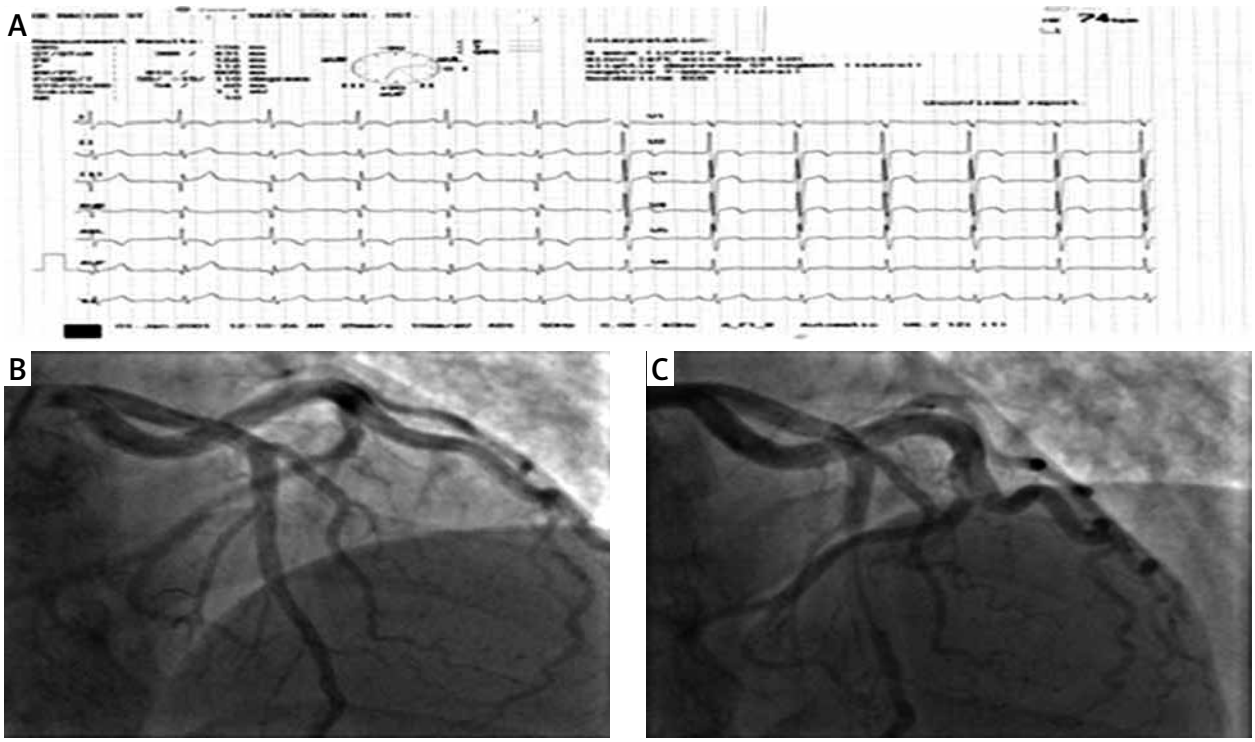

Figure 1. Biphasic T wave on precordial leads **(A)**, myocardial bridging on left anterior descending artery in diastole **(B)** and systole **(C)**

diltiazem or amlodipine in high dosage leads to reduction of angina due to relief of concomitant vasospasm [1].

In contrast, pure vasodilator agents such as nitroglycerin should be used cautiously in patients with MB. Although nitrates have antispasmodic features and can decrease pre-load, they can worsen symptoms by intensifying systolic compression of the bridged segment and the vasodilating segments proximal to the bridge, thereby exacerbating retrograde flow in the proximal segment and reducing the myocardial ischemic threshold [2].

Vasoconstriction develops in normal and atherosclerotic coronary arteries with the effect of an α_1-agonist. Doxazosin is known as an α_1-blocker of adrenergic receptors. It is reported that vasodilatation develops in normal coronary arteries and paradoxical vasoconstriction in the coronary arteries with endothelial dysfunction after α_1 blockage. There is no prominent effect of doxazosin on cardiac contractility. Furthermore, doxazosin induces reflex sympathetic release, which could evoke an increase in heart rate, myocardial oxygen demand and myocardial ischemia. Cardiac relaxation time becomes shorter due to tachycardia. Lee *et al.* [3] reported that doxazosin significantly decreased the coronary diameter in the stenotic segments, decreased coronary blood flow and increased coronary resistance caused by impaired endothelial function. In patients with MB, endothelial function is impaired and there is an increased tendency for atherosclerosis proximal to the bridge [4]. In the light of the present data, we concluded that our patient developed acute coronary syndrome due to increased myocardial oxygen demand as a result of reflex sympathetic release and vasodilatation in the normal coronary artery segments and paradoxical vasoconstriction in the coronary artery segments with endothelial dysfunction after the α_1 blockage therapy.

Conclusions

Caution is required in the use of α_1 blocker in patients with known MB. It is important to be alert for MB in patients developing acute coronary syndrome after α-blocker therapy. All factors influencing cardiac contractility or vasodilator function should be considered in patients with myocardial bridging, since left ventricular hypertrophy/diastolic dysfunction is also a risk factor in these patients.

Conflict of interest

The authors declare no conflict of interest.

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