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Case Report cardiology

Difficult management of a patient presenting with recurrent syncope caused by diffuse vasospasm

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ABSTRACT

Spontaneous and simultaneous multivessel coronary artery spasm may present with multisite myocardial ischemia, atrioventricular block, acute lung edema, cardiogenic shock, or ventricular fibrillation. In a case of syncope caused by vasospasm, the underlying mechanism may be complex, such as atrioventricular block and/or ventricular arrhythmia. Dual implantable cardioverter defibrillator (ICD) placement should be considered along with optimal medical treatment. This report is a description of a 57-year-old male patient who was admitted to the hospital with chest pain followed by loss of consciousness. As the patient had bradycardia, a diffuse spasm, and life-threatening ventricular arrhythmia during ischemic episodes, a dual ICD device was implanted. ICD treatment may be a good option in cases with a diffuse spasm that is hard to control with medical treatment due to the risk of life-threatening ventricular arrhythmia.

Keywords: Complete atrioventrikuler block; syncope; vasospasm.

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Both coronary vasospasms and beta blockers can cause cardiac syncope. Here, we present the case of a patient with recurrent syncope suspected due to sinus bradycardia caused by the chronic use of beta blockers. However, we noticed that the underlying mechanism of syncope was diffuse vasospasm causing complete atrioventricular block and ventricular arrhythmia. The patient was successfully managed with dual implantable cardioverter-defibrillator (ICD) implantation as well as oral nitrate and high-dose nondihydropyridine calcium channel blocker administration. In the case of syncope caused by vasospasm, the underlying mechanism may be complex, involving atrioventricular block and/or ventricular arrhythmias. Hence, dual ICD implantation should be considered along with optimal medical treatment.

CASE REPORT

A 57-year-old male was admitted to our hospital with chest pain followed by loss of consciousness. It was stated that loss of consciousness was not related to the patient's position, and according to the eyewitness' statement, the patient's eyes were open at that time. Episodes lasted for approximately 2–3 min. During the episode, fecal and urinary incontinence, tongue-biting, and tonic–clonic seizures were not observed. The medical history revealed that the patient was taking 5 mg amlodipine and 50 mg metoprolol succinate due to hypertension; there was no history of alcohol or cigarette consumption. The patient was then hospitalized and admitted to the cardiology department. Sinus bradycardia (45/min) was detected on electrocardiogram (ECG) at admission, and the patient's blood pressure was 165/63 mmHg. Then, beta blocker

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treatment was stopped. There were also no electrolyte disturbances on admission. No other pathology except sinus bradycardia was detected during 24-h ambulatory rhythm monitoring. On the third day of hospitalization, ST segment elevation at DII-DIII as well as aVF derivations and complete atrioventricular (AV) block were detected on ECG (Fig. 1). Emergency coronary angiography was performed, and diffuse spasm was detected at the left anterior descending, circumflex, and right coronary arteries (Fig. 2). After intracoronary administration of 300 mcg nitrate, vasospasm disappeared and ST elevation and AV block regressed (Figs. 3, 4). Because the patient had bradycardia, diffuse spasm, and life-threatening ventricular arrhythmia during ischemic episodes, dual implantable cardioverter-defibrillator (ICD) was implanted. Then, 50 mg isosorbid 5-mononitrate and 90 mg diltiazem 2×1 po treatment was initiated. Episodes of syncope continued, and ventricular fibrillation reoccurred during an ischemic episode because of vasospasm. During follow-up, the diltiazem dose was increased up to 480 mg, and episodes were taken under control . In the sixth-month follow-up, the patient had no complaints of angina and syncope and his vasospastic episodes were under control with treatment.

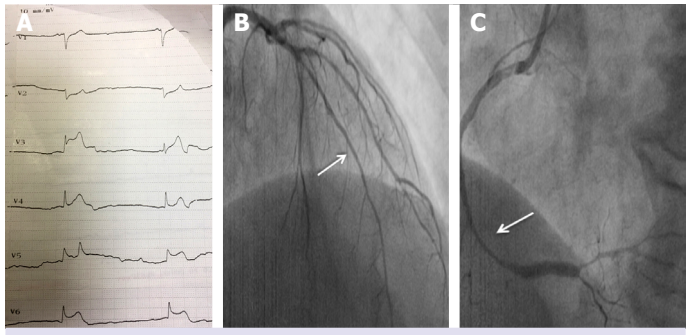

FIGURE 1. (A) Electrocardiogram with ST segment elevation and complete atrioventricular block. (B) Coronary angiographic views that show diffuse coronary vasospasm.

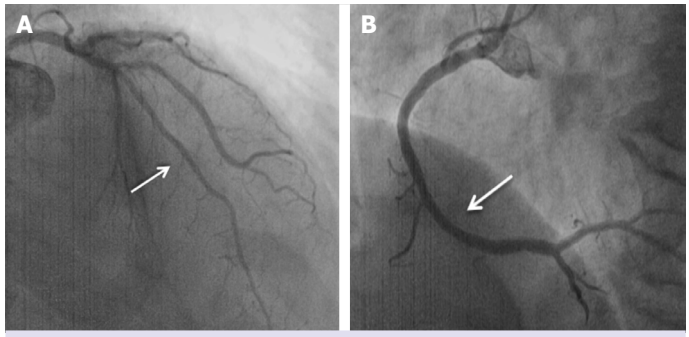

FIGURE 2. After intracoronary nitrate injection, coronary vasospasm is released.

DISCUSSION

Here, we present the case of a patient with recurrent syncope suspected due to sinus bradycardia caused by the chronic use of beta blockers. However, we noticed that the underlying mechanism of syncope was in fact diffuse vasospasm causing high-grade AV block treated with oral

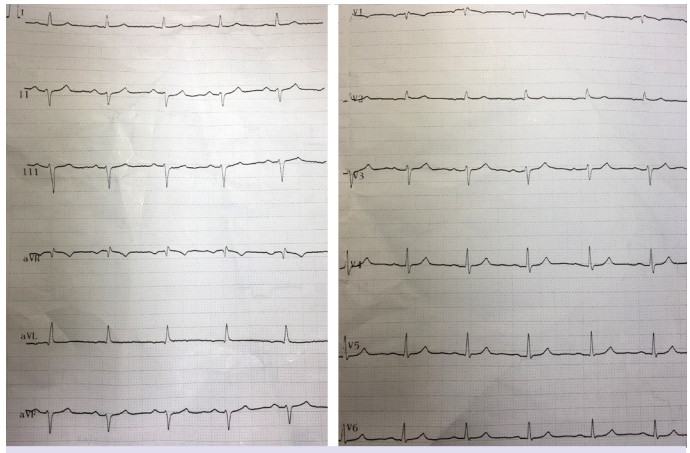

FIGURE 3. After intracoronary nitrate injection, ST segment elevation and atrioventricular block are resolved.

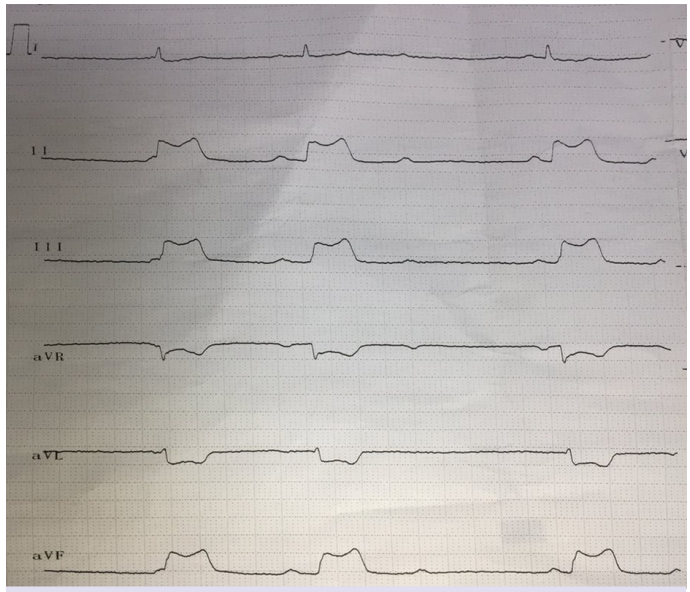

FIGURE 4. Electrocardiogram with ST segment elevation and complete atrioventricular block on inferior derivations.

nitrate and high-dose nondihydropyridine calcium channel blocker administration. The patient's arrhythmias were successfully managed with dual ICD implantation.

Endothelial dysfunction and enhanced vascular smooth muscle contractility are considered the major underlying mechanisms for the pathogenesis of coronary artery spasm; however, the variant angina has not been fully elucidated. In the literature, many primary pathophysiologic mechanisms that cause coronary vasospasms have been listed, such as imbalanced autonomic nervous activity, increased oxidative stress, chronic low-grade inflammation, magnesium deficiency, and genetic susceptibility [1, 2]. It is known that in a population with coronary artery disease with minimal atherosclerotic changes or even completely normal coronary arteries, myocardial ischemia associated with typical coronary vasospasm can result in severe clinical outcomes, such as SCD [2, 3]. Cardiac arrhythmias associated with myocardial ischemia have an important role in adverse cardiovascular events in patients with vasospastic angina. In their ambulatory ECG follow-up study, Onaka et al.[3] stated that vasospasm in multivessels or differentially localized coronary arteries is possible and that these patients can experience sudden cardiac death and malign arrhythmias. In a case presented by Ghadri et al. that includes a patient admitted with typical angina and high-grade AV block, coronary arteries were normal after nitrate administration, and they successfully treated the patient with long-term calcium channel blocker and nitrate administration [4]. In a case presented by Chuang et al., [5] a case with VF arrest due to vasospastic angina and a course of pulmonary edema, it was seen that coronary anatomy was normal after nitrate administration, and treatment was performed with high-dose calcium channel blocker administration and ICD implantation. In patients with vasospastic angina who are admitted with multivessel vasospasm and cardiac arrhythmia, in addition to the traditional treatment methods of calcium channel blocker and nitrate administration, dual ICD implantation has also been listed as a good treatment option in the literature. Similarly, Gul et al. successfully managed a patient who had recurrent angina attacks by bosentan therapy (an endothelin receptor antagonist), differently from conventional vasospastic angina treatment [6]. Simcha et al. reported on a patient with variant angina complicated by VF who was at a life-long risk for sudden death when exposed to myocardial ischemia [7, 8]. Although there are similar cases in the literature, our case differs. This is because after our patient was repeatedly examined with isolated syncope and his treatment was discontinued due to the risk of beta blocker-dependent bradycardia, his heart rhythm recovered, and we were planning his discharge when underlying Prinzmetal angina emerged and completely changed the course of clinical diagnosis and treatment. Therefore, we believe that it will be particularly helpful for clinicians to investigate the etiology of syncope in patients. In addition, during treatment management, it should be kept in mind that low-dose calcium channel blocker administration can be insufficient, as was the case in our patient, and ICD implantation should lower the rate of cardiovascular mortality that is unwanted in the long term.

CONCLUSION

Coronary vasospasm is one of the causes of cardiovascular syncope, and it is difficult to definitely diagnose the condition when it occurs in an angiographically normal-looking vessel. Due to the risk of life-threatening ventricular arrhythmia, ICD implantation can be performed in cases with diffuse spasm that is hard to control with medical treatment. In these patients, standard doses of calcium channel blockers are frequently insufficient, and high-dose nondihydropyridine calcium channel blocker administration is required.

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