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관상동맥 연축에 의하여 발생한 발작성심방세동과 동서맥 1예

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A Case of Paroxysmal Atrial Fibrillation and Sinus Bradycardia due to Coronary Artery Spasm

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Paroxysmal atrial fibrillation may be induced by coronary spasm presenting with typical angina-like pain and palpitations. It is typically treated using rate or rhythm control strategies, although sustained coronary spasm can induce sinus bradycardia with dizziness and syncope. In the present case, we reached a diagnosis of paroxysmal atrial fibrillation and sinus bradycardia due to coronary artery spasm using the methyl-ergonovine provocation test during angiography. While the treatment of coronary spasm can resolve paroxysmal atrial fibrillation, sinus bradycardia, and variant angina, the mechanism remains unclear, although it may be associated with sinus node ischemia. Similar symptoms, particularly chest discomfort, should be carefully considered in cases of paroxysmal atrial fibrillation. (Korean J Med 2015;89:79-84)

Keywords: Atrial fibrillation; Bradycardia; Coronary vasospasm; Ischemia

INTRODUCTION

Atrial fibrillation is the most common type of sustained arrhythmia, and its increasing incidence has made it a global healthcare concern [1]. Current interventions for atrial fibrillation are targeted towards treating the symptoms and reducing the risk of tachycardia-induced cardiomyopathy and stroke [2], either through control of the ventricular heart rate or restoration of the sinus rhythm [1]. Here, we report the first Korean case of recurrent paroxysmal atrial fibrillation and sinus bradycardia due to coronary spasm, and the successful resolution of the atrial fibrillation following treatment for the coronary spasm.

CASE REPORT

A 69-year-old male with severe chest discomfort and exertional dyspnea visited our emergency room in the morning. He had suffered from intermittent chest discomfort and dyspnea for the

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previous 5 years and had previously visited two other local clinics. He was diagnosed as normal at the first visit after coronary angiography. However, following coronary angiography at the second visit, he was diagnosed with atrial fibrillation and treated with medication. Upon admission to our hospital, he also had hypertension and dyslipidemia treated with flecainide, aspirin, atorvastatin, candesartan, hydrochlorothiazide, bisoprolol, and warfarin.

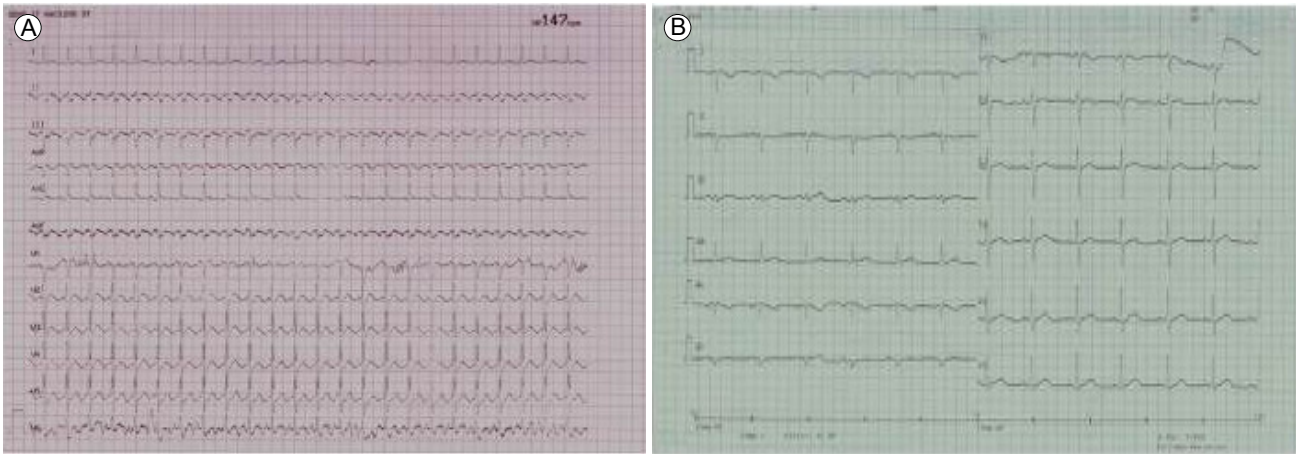

Figure 1. A 12-lead electrocardiogram recorded upon admission of the patient to the emergency room. (A) Atrial flutter with tachycardia was visible at admission. (B) After 4 hr, a normal sinus rhythm was observed.

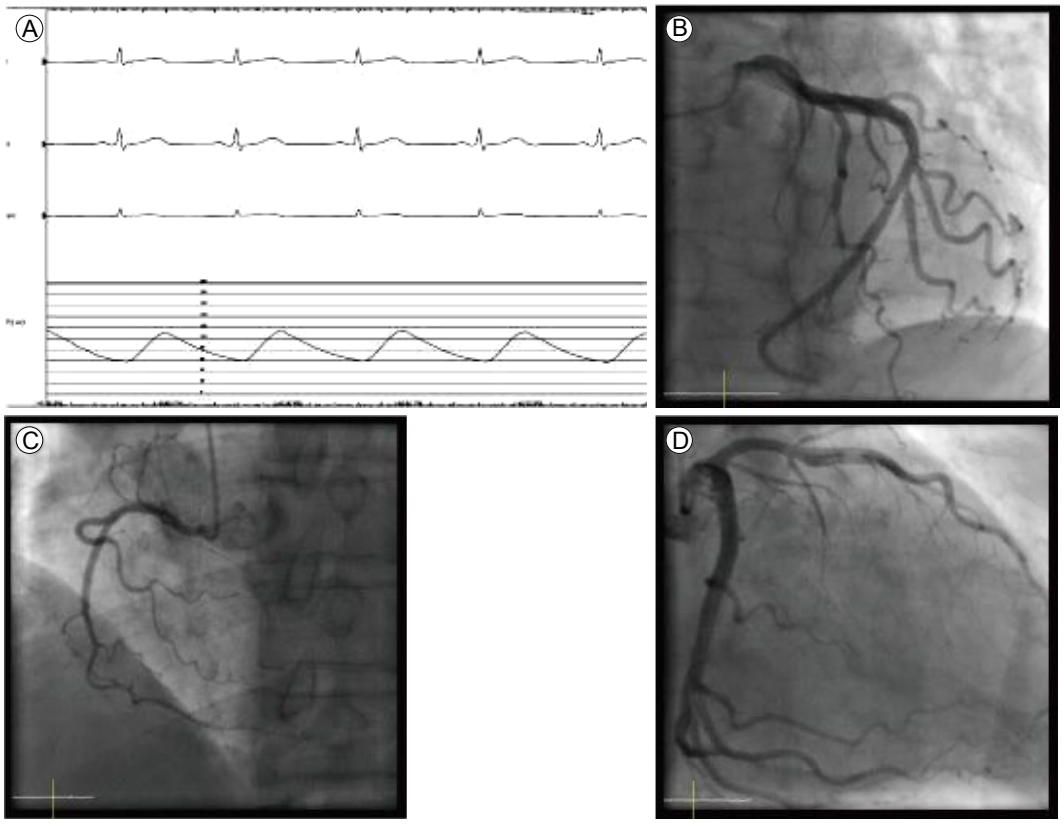

Figure 2. The initial electrocardiogram (A) showed a normal sinus rhythm with stable vital signs. Initial coronary angiography showed no stenosis in the (B) left anterior descending artery, (C) left circumflex artery, and (D) right coronary artery.

At the time of admission, the patient reported feeling chest discomfort and palpitations, typically in the morning. He subsequently experienced syncope, which lasted ~20 min. After the patient regained consciousness, his chest discomfort persisted, along with a radiating pain in his left arm and back. An initial electrocardiogram (ECG) revealed an atrial flutter with tachycardia (Fig. 1A), although a follow-up ECG 4 hr later revealed a normal sinus rhythm (Fig. 1B). His other vital signs, as well as the results of a physical examination and echocardiography, were all normal. His blood test results, cardiac enzyme levels, and thyroid function were also normal, although his brain natriuretic peptide levels were slightly elevated.

As the patient had typical angina at admission, we decided to perform coronary angiography. When the patient was tested at our catheterization laboratory, he had no chest pain, and his initial vital signs and echocardiogram results were normal, with a stable sinus rhythm (Fig. 2A). Coronary angiography revealed no stenosis in the left anterior descending artery, the left circumflex artery, or the right coronary artery (Fig. 2B, 2C, and 2D). However, the patient complained of definite symptoms of angina; therefore, we provoked coronary spasm by injecting 80 μg of methyl-ergonovine into the right coronary artery. Coronary artery spasm (> 90% reduction) was observed 2 min after provocation (Fig. 3A). The patient still complained of chest discomfort, although the symptoms were not the same as at the time of admission. Three minutes after provocation, echocardiography indicated a sudden onset of atrial fibrillation, with a rapid ventricular response (heart rate: 130 bmp; Fig. 3B and 3C) and the patient reported palpitations with chest discomfort. Four minutes after provocation, the coronary angiogram still indicated near total occlusion due to severe coronary spasm in the right coronary artery (Fig. 4A). At this point, the echocardiogram had changed to sinus bradycardia (heart rate: 32 bpm; Fig. 4B) with decreased blood pressure (68/35 mmHg), and the patient reported dizziness. We immediately injected 100 μg of nitroglycerin via the intracoronary artery and administered sublingual nitroglycerin. After 10 s, the coronary spasm had abated, the echocardiogram results were normal (Fig. 4C and 4D), and the patient complained of no significant symptoms.

Based on this observation, we diagnosed the patient with atrial

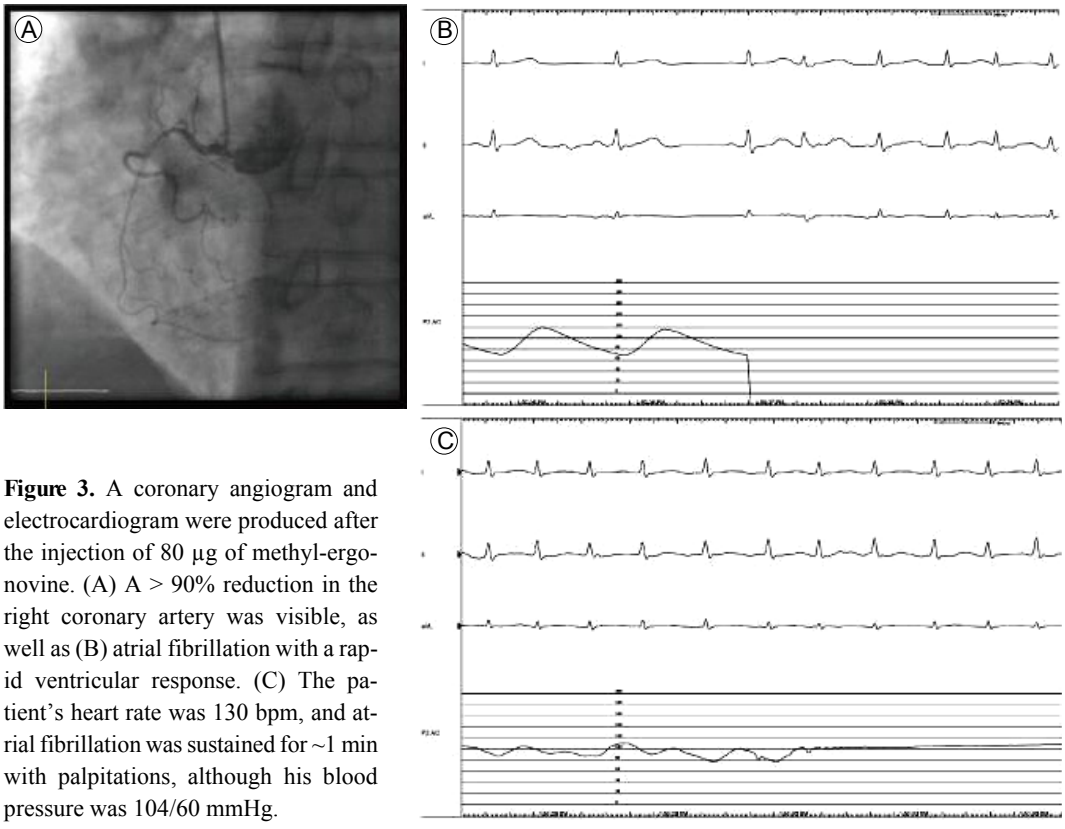

Figure 3. A coronary angiogram and electrocardiogram were produced after the injection of 80 μg of methyl-ergonovine. (A) A > 90% reduction in the right coronary artery was visible, as well as (B) atrial fibrillation with a rapid ventricular response. (C) The patient's heart rate was 130 bpm, and atrial fibrillation was sustained for ~1 min with palpitations, although his blood pressure was 104/60 mmHg.

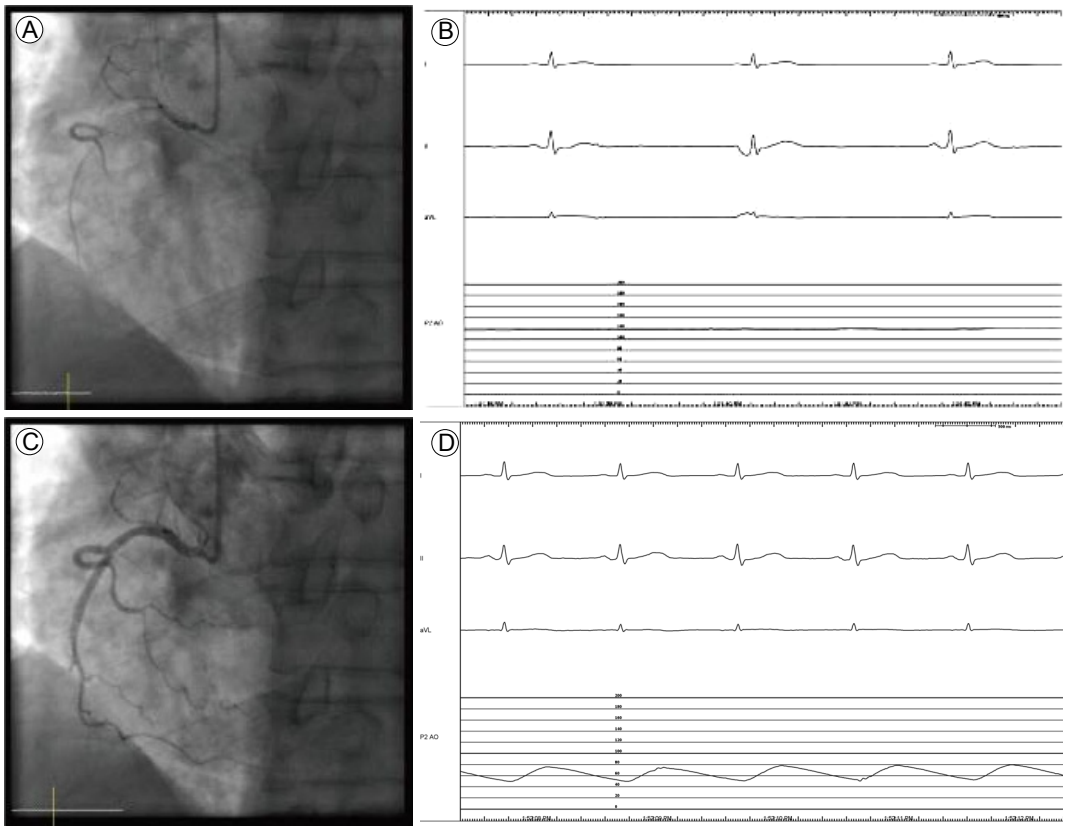

Figure 4. A coronary angiogram and electrocardiogram were produced 4 min after the provocation test and after the administration of 100 μg of intracoronary nitroglycerin. (A) The right coronary artery was almost completely occluded 4 min after the provocation test. (B) Paroxysmal atrial fibrillation was observed as sinus bradycardia with dizziness and a marked decrease in blood pressure (68/35 mmHg). Intracoronary nitroglycerin (100 μg) was administered, and (C) the right coronary flow and lumen size recovered immediately, with (D) a normal sinus rhythm, stable vital signs, and no notable symptoms.

fibrillation and sinus bradycardia due to severe coronary spasm. We changed the patient's medication to nicorandil, isosorbide mononitrate, and diltiazem for spasm treatment [3], and stopped the bisoprolol, flecainide, and warfarin, although aspirin and statin treatment was maintained. After altering his regimen, the patient did not experience a recurrent episode of chest discomfort or palpitations, and a normal sinus rhythm was detected during follow-up echocardiography. Since being discharged 6 months ago, the patient has not reported any further symptoms (palpitations, dizziness, and chest discomfort).

DISCUSSION

In this case, we concluded that paroxysmal atrial fibrillation was caused by coronary spasm, induced during the provocation test. The sustained coronary spasm appears to have induced symptomatic sinus bradycardia and paroxysmal atrial fibrillation, which subsequently disappeared when the patient was treated for the coronary spasm. Interestingly, the initial ECG indicated an atrial flutter with tachycardia, although this may have been a complication of the flecainide that was used to achieve atrial fibrillation rhythm control [4]. In addition, we were concerned about a coronary spasm-induced atrial flutter.

There are many factors associated with atrial fibrillation (Fig. 5, adapted from [5]), although our patient only exhibited hypertension. Various precipitating events can be identified in ~40% of patients with paroxysmal atrial fibrillation, including binge drinking, physical exertion, or emotional distress [1]. In contrast, our patient complained of palpations only after specific chest discomfort. Paroxysmal atrial fibrillation was induced after right

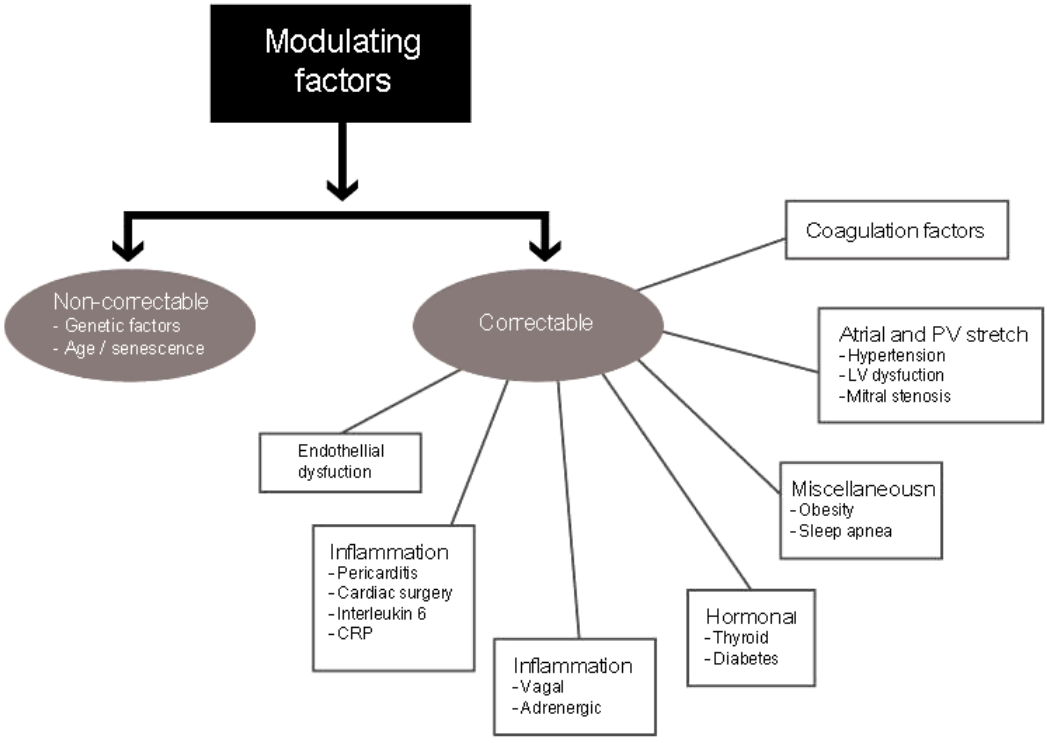

Figure 5. Schematic diagram illustrating the numerous factors that influence and may contribute to the pathogenesis of atrial fibrillation (adapted from: Mathew et al., Eur J Intern Med 2009 [5]). CRP, C-reactive protein; PV, pulmonary vein; LV, left ventricle.

coronary artery spasm, without a change in blood pressure, and was subsequently relieved after the spasm abated. In addition, sinus bradycardia occurred after the sustained coronary spasm, and was immediately improved after the administration of nitroglycerin. Therefore, we conclude that paroxysmal atrial fibrillation could have been induced by the coronary artery spasm, and subsequently alleviated by treatment of the spasm.

The mechanism of atrial fibrillation is not fully understood, although several theories exist. The three main theories are the random re-entry model, the single or multiple rapidly firing ectopic foci model, and the multiple re-entrant model [5]. Atrial fibrillation can occur during acute myocardial infarction, due to occlusion proximal to the origin of the sinus node artery [6], which can result in impaired generation of the sinus impulse, sinoatrial conduction, and subsequent neural injury because of ischemia [7]. A sustained coronary spasm in the right coronary artery may therefore decrease atrial excitability; thus, we observed paroxysmal atrial fibrillation as symptomatic sinus bradycardia.

There are several reports describing paroxysmal atrial fibrillation associated with coronary spasm. For example, Previtali et al. [8] reported that paroxysmal atrial fibrillation induced an autonomic nervous system imbalance or alterations in myocardial metabolism, resulting in coronary spasm. In addition, Hung et al. [7] suggested that paroxysmal atrial fibrillation was induced by coronary spasm. Similarly, cardiac arrhythmias such as a ventricular arrhythmia or atrioventricular block have been frequently reported as a result of coronary vasospasm [9]. However, supraventricular arrhythmias related to coronary spasm are extremely rare, although Kawakami et al. [10] reported that coronary artery spasm was induced more frequently in patients with atrial fibrillation compared to in control patients (76.5 vs. 8.8%; odds ratio, 33.583; 95% confidence interval, 6.5732-171.58; $p < 0.0001$). Therefore, it is possible that a large number of patients with paroxysmal atrial fibrillation due to coronary spasm are not identified using current diagnostic criteria.

Interestingly, transient sinus node ischemia due to spasm was also observed in our patient, and this condition is known to predispose patients to atrial fibrillation. In addition, prolonged is-

chemia induces sinus bradycardia. Therefore, we concluded that the coronary spasm induced the paroxysmal atrial fibrillation and sinus bradycardia (along with the associated palpitations, chest discomfort, and dizziness), and treated the spasm with intracoronary nitroglycerin, which subsequently resulted in resolution of the paroxysmal atrial fibrillation and sinus bradycardia. We performed the provocation test via an intracoronary artery and the patient's blood pressure decreased, leading to presyncope; thus, we were unable to perform a provocation test on the left coronary artery. The patient's right coronary artery size was small and the left circumflex coronary artery was dominant. Thus, sinus bradycardia may have occurred via left coronary artery spasm with an atrioventricular node block.

Coronary spasm often induces life-threatening ventricular arrhythmias with cardiac arrest or syncope. In our case, the patient's syncope was thought to be related to sinus bradycardia. Once we reached this conclusion, we changed the patient's medication to target the coronary spasm without antiarrhythmic medication. Consequently, the patient did not experience any recurrence of palpitations, dizziness, or chest discomfort. If an arrhythmia had arisen again despite medical treatment, we would have considered ablation therapy.

In summary, we observed that paroxysmal atrial fibrillation, which may be induced by prolonged coronary spasm, can cause sinus bradycardia. The mechanism is unclear, although it may be related to sinus node ischemia, which can cause such symptoms as palpitations, syncope, and dizziness. In cases of paroxysmal atrial fibrillation with specific chest discomfort, coronary spasm should be considered, as atrial fibrillation might be resolved by treating the coronary spasm. A large population of patients with paroxysmal atrial fibrillation due to coronary spasm may exist; therefore, we believe that a large-scale study regarding the pathogenesis should be conducted.

중심 단어: 심방세동; 서맥; 관상동맥 연축; 허혈

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