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Case Report OMICS International

A Case of Vasospastic Angina Involving Erosion and Thrombus within the Spastic Segment Detected by Coronary Angioscopy

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Abstract

A-58-year-old female who had been treated for vasospastic angina had difficulty in breathing and palpitations during rest at night. She was admitted to our institution for a detailed examination of her symptoms. Coronary angiography showed no obvious coronary stenosis. Thereafter, we performed the spasm provocation test (SPT), firstly in the right coronary artery (RCA) and then in the left coronary artery (LCA), using intracoronary infusions of acetylcholine. SPT revealed a focal spasm at the distal segment of RCA and diffuse spasms at the left anterior descending coronary artery (LAD) and left circumflex artery. An intracoronary infusion of nitroglycerin relieved coronary spasms. Coronary angioscopy revealed erosions and white thrombus at the spastic segment of the proximal segment of LAD. Administration of two types of coronary vasodilators improved chest symptoms. Recent studies have focused on acute coronary syndrome without plaque rupture, and coronary spasm has been proposed as one of the underlying mechanisms responsible. The findings of the present case may support a relationship between coronary spasm and coronary erosion with thrombus formation.

Keywords: Coronary spasm; Vasospastic angina; Coronary erosion; Coronary thrombus; Coronary angioscopy

Introduction

Intracoronary thrombus formation is known to contribute to the development of acute coronary syndrome (ACS). Further, thrombus formation is most commonly caused by plaque rupture [1]. Conversely, ACS without plaque rupture has been reported [2], and coronary spasm has been considered one of the causes of ACS without plaque rupture [2-4]. However, a little is known regarding the relationship between coronary spasm and thrombus formation [5, 6].

Herein, we report a case of vasospastic angina, in which erosions and thrombus were detected within the spastic segments using coronary angioscopy (CAS).

Case Report

A 58-year-old female had a history of anterior chest pain at dawn during rest that had begun in her 40s. Five years ago, she had been diagnosed with vasospastic angina (VSA) and treated with nicorandil at another hospital. From 4 months ago, she had difficulty in breathing from the neck to the chest and palpitations during rest at night. She subsequently presented at our institution for the evaluation of her symptoms in May 2016. She consumed 350 ml of beer and a glass of wine per day but had no history of smoking. She had been diagnosed with hypothyroidism and had undergone surgeries for inguinal hernia and uterine prolapse. She had a family history of coronary artery disease, and her mother also had a history of angina. Blood examinations revealed hypokalemia (3.3 mEq/L) as well as elevated fasting blood sugar (112 mg/dL) and brain natriuretic peptide (86 pg/mL) levels. An electrocardiogram revealed sinus rhythm, with a negative T-wave in the precordial leads. Although the cardiothoracic ratio on chest X-ray was 54.5%, echocardiograhy showed normal findings. For the evaluation of progression of coronary atherosclerosis and continuation of a high activity of coronary spasm, she was admitted to our institution for coronary angiography (CAG) and spasm provocation test (SPT). Coronary vasodilator administration was stopped one day prior to admission.

Initial CAG showed no obvious coronary stenosis of the right coronary artery (RCA) or left coronary artery (LCA) (Figure 1A and D). To clarify the cause of her chest symptoms, we then performed SPT using acetylcholine (ACh). Intracoronary infusion of 20 and 50 μg ACh into RCA caused a focal spasm at the distal segments (Figure 1B) accompanied with the usual chest pain, which had disappeared spontaneously without intracoronary nitroglycerin (NTG) injection. Continuous intracoronary infusions of 50 and 100 μg ACh into LCA caused diffuse spasms at the left anterior descending coronary artery (LAD) and left circumflex artery (Figure 1E) accompanied with the usual chest symptoms. Intracoronary infusion of 0.2 mg NTG relieved the coronary spasms (Figure 1C and 1F).

Based on these findings, we diagnosed multi-vessel coronary artery spasm. We performed CAS to observe intracoronary imaging after obtaining informed consent from the patient. Erosions were observed at the spastic segments of the proximal LAD, with white thrombus at both the proximal and distal erosions (Figure 2). The patient was initiated on 8 mg/day benidipine hydrochloride and was carefully followed up as an outpatient. Thereafter, the frequency of chest symptoms reduced, but remained despite medication. Therefore, we changed her medication to 40 mg/day nifedipine and isosorbide dinitrate patches, which led to complete disappearance of her chest symptoms.

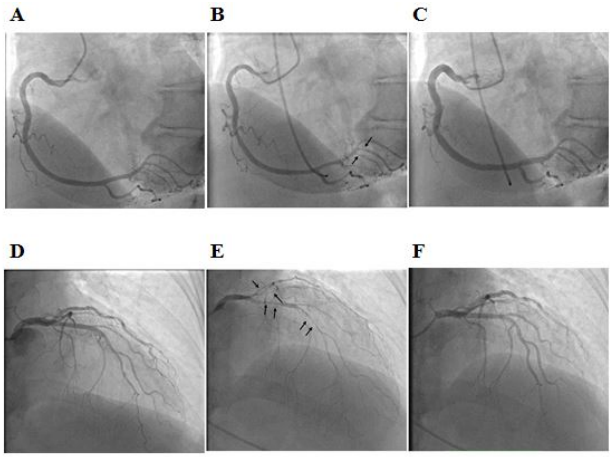

Figure 1: Coronary angiography and spasm provocation test. Above: Right coronary artery. A: There were no atherosclerotic changes at baseline. B: After intracoronary infusion of 50 μg acetylcholine (ACh), a focal spasm occurred at the distal segment (indicated with arrows). C: After the intracoronary infusion of nitroglycerin, no significant coronary stenosis was observed. Below: Left coronary artery. D: There were no atherosclerotic changes at baseline. E: After the intracoronary infusion of 100 μg ACh, a diffuse spasm occurred at the left anterior descending coronary artery (LAD) and left circumflex artery accompanied with chest symptoms. F: After the intracoronary infusion of nitroglycerin, no significant coronary stenosis was observed.

Discussion

In the present case, SPT revealed multi-vessel coronary spasms and CAS demonstrated erosions and thrombus within the spastic segments. Thrombus formation due to coronary spasms has been reported as one of the mechanisms responsible for ACS without plaque rupture [2-4], with the findings of the present case supporting the presence of coronary spasm-induced thrombus formation.

There are several mechanisms responsible for coronary spasm-induced thrombus formation. In cases with coronary plaque rupture, coronary spasm may cause plaque rupture mediated by the external force of the coronary spasm itself; and this relationship was confirmed in an autopsy case report [7]. On the other hand, coronary erosions without plaque rupture are thought to contribute to coronary spasm-induced thrombus formation [2-4]. Using CAS, Etuda et al. [5] reported that intracoronary abnormalities, such as intracoronary hemorrhage, intimal flap, thrombus, or ulcers, were present in 40% of cases with variant angina, which is thought to be a type of angina with a high coronary spasm activity. In addition, using optical coherence tomography (OCT), Shin et al. [6] demonstrated the presence of OCT-defined erosions in 21 (26%) of 80 coronary vessels in 69 VSA patients. These reports indicated that intracoronary erosion and thrombus formation may occur within spastic segments. Using OCT, Tanaka et al. [8] demonstrated initial gathering, characterized by folding or gathering of the intima, at spastic segments during spasm. Therefore, coronary spasms may lead to hypercontraction and extension of the intima, which results in coronary erosion formation.

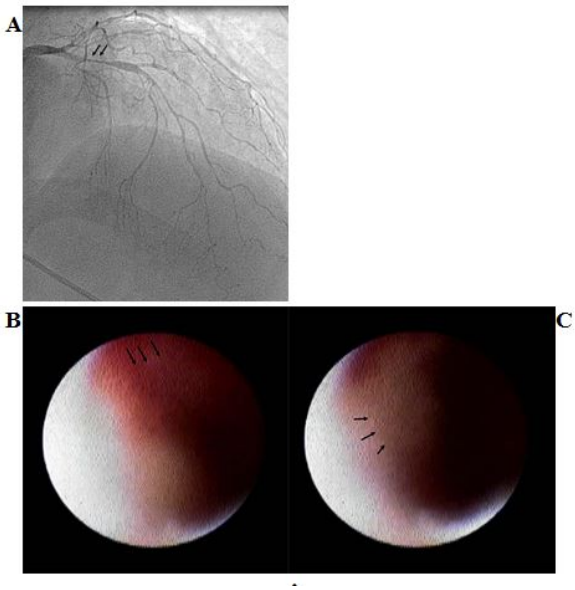

Figure 2: CAS at the proximal of left anterior coronary artery. A: Coronary angiogram of the left anterior descending coronary artery (LAD) during spasm. Arrows indicate the observed segment of CAS, as shown below. B: Coronary erosions in the coronary artery (red color, indicated with arrows). C: White thrombus within the coronary artery (indicated with arrows).

Accordingly, coronary erosions with thrombus formation may represent a cause rather than a result of coronary spasm. However, in the present case, coronary spasm occurred at diffuse segments of LAD, whereas coronary erosion and thrombus were found only within the proximal LAD, which was the most severely spastic site observed on angiography. This finding may indicate that the presence of coronary erosions and thrombus was the result of coronary spasm. Finally, there have been several reports on coronary spasms themselves promoting thrombus formation [9,10]. Miyamoto et al. [9] demonstrated that coronary spasms cause platelet aggregation in the coronary circulation, and Oshima et al. [10] revealed that coronary spasms activate blood coagulation and lead to thrombus formation. These factors may also contribute to thrombus formation at sites of coronary erosion caused by coronary spasms.

An autopsy study [11] reported that 30%–35% of patients who died of coronary causes had erosions. Furthermore, sudden death due to coronary erosion tended to be more frequently observed in women, younger patients, and in lesions with less severe narrowing, less calcification, and less thrombus [12]. These findings indicated that coronary spasms may causes erosions and lead to sudden death, even in young women. The present case was a relatively young woman with VSA that developed in her 40s and got worse in her 50s. Because she showed similar features to those shown in the above review [12], we deemed it necessary to perform careful and strict follow-up.

Because thrombus was found in the coronary artery by CAS, we considered the use of antiplatelet drugs in the present case. We considered frequent occurrence of coronary spasm to be the main cause of thrombus formation; therefore, we prescribed two types of coronary vasodilator in the present case rather than antiplatelet drugs.

A previous study reported that the administration of a low dose of aspirin does not influence the prognosis in VSA patients without significant coronary stenosis [13]. However, the present case had less frequent chest symptoms while taking one calcium channel blocker; therefore, it may be safer to add antiplatelet drugs during the acute phase.

Conclusion

We experienced a case of VSA who had increasingly frequent chest symptoms, in which coronary erosions and thrombus were detected within spastic segments using CAS. Coronary spasms may cause erosion and represent a cause of thrombogenesis and ACS. Taking these suggestions into consideration, cardiologists should treat VSA patients carefully.

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