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【 CASE REPORT 】

The Significance of Recognizing Myocardial Bridge in the Coronary Spasm Diagnosis in Myocardial Infarction with Nonobstructive Coronary Arteries

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Abstract:
A 61-year-old man experienced chest oppression for 1 hour. He was positive for troponin T and underwent emergent coronary angiography (CAG), which did not reveal significant coronary stenosis. He was diagnosed with myocardial infarction with nonobstructive coronary arteries (MINOCA). We performed a spasm-provocation test, which revealed a focal spasm at the segment of the myocardial bridge. After receiving a calcium-channel blocker, he exhibited a good clinical course. Coronary spasm is considered an underlying cause of MINOCA; therefore, the presence of a myocardial bridge may help with the diagnosis.

Key words: coronary spasm, vasospastic angina, myocardial infarction with nonobstructive coronary arteries, myocardial bridge

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Introduction

Recently, myocardial infarction with nonobstructive coronary arteries (MINOCA) has garnered some interest. It has multifactorial causes, including coronary plaque disease, coronary dissection, coronary spasm, and coronary microvascular angina (1, 2). Of these, coronary spasm is considered to be one of the major causes of MINOCA (3, 4); however, in some emergency cases, it is challenging to determine the correlation between the coronary spasm and MINOCA because of prior nitroglycerin (NTG) administration. Furthermore, a correlation between the coronary spasm and the myocardial bridge has been reported (5-12).

We herein report the case of a patient with MINOCA in whom a spasm-provocation test (SPT) during emergent coronary angiography (CAG) revealed coronary spasm as the cause, with the myocardial bridge being a clue.

Case Report

A 61-year-old man was referred to our hospital by his home physician because he had been experiencing chest pain for 1 hour, starting at 7 AM in mid-February 2019. On admission at our institution, he described his chest pain as 1 (0: free from chest pain, 10: maximum). In his interview, he revealed that he sometimes felt chest discomfort during the night; however, he had not consulted his home physician. He had coronary risk factors of smoking (20 cigarettes/day for 30 years) and untreated dyslipidemia.

His medical examination revealed the following data: height, 166.8 cm; weight, 60.8 kg; body mass index, 21.9 kg/m^2; blood pressure, 118/68 mmHg; and pulse rate, 84/min. All other vital signs were stable, no cardiac murmur or abnormal respiratory sounds were noted, and no abnormal abdominal findings were observed. A blood examination revealed a high level of low-density lipoprotein cholesterol (194 mg/dL) and marked elevation in the troponin T level (0.176 ng/mL). An electrocardiogram (ECG) revealed a normal sinus rhythm with no significant ST-T changes. However, echocardiography revealed mild hypokinesis of the anteroapical left ventricle with a left ventricular ejection fraction (LVEF) of 60%. Based on these findings, the patient was diagnosed with non-ST elevation myocardial infarction.

After the intravenous administration of 0.3 mg of NTG, emergent CAG revealed mild coronary stenosis at the mid-

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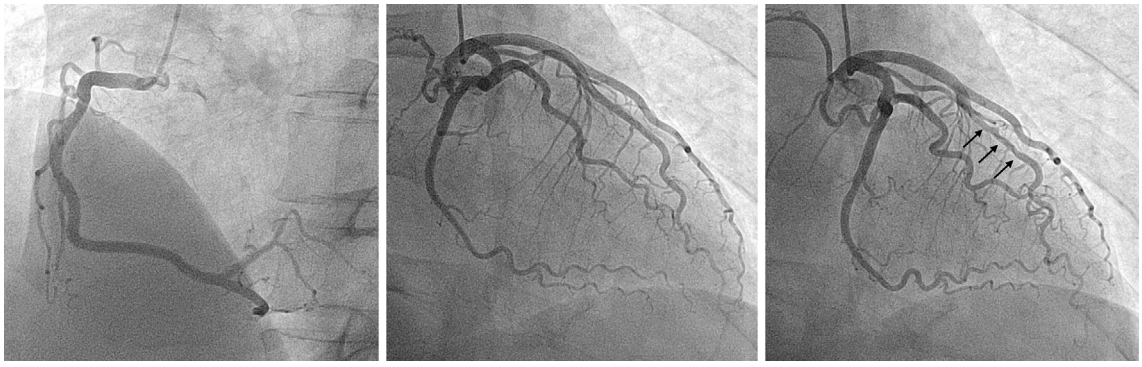

Figure 1. **Coronary angiography at the baseline after the intravenous administration of nitroglycerin (NTG). Coronary angiograms reveal mild coronary stenosis at the mid-portion of the right coronary artery (left panels) and an angiographically normal left coronary artery, except for a myocardial bridge at the mid-segment of the left anterior descending coronary artery (mid-panel: diastole; right panel: systole). Arrows, segments of the myocardial bridge.**

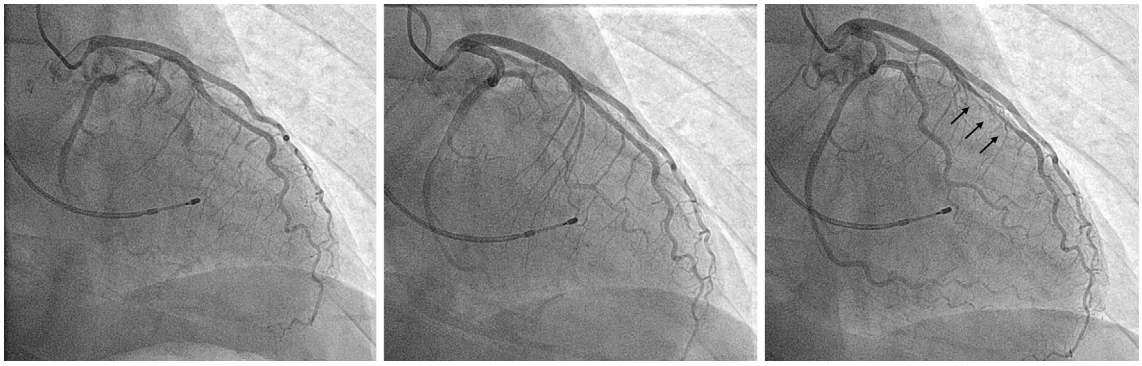

Figure 2. **Coronary angiography during the spasm-provocation test (SPT). First, infusions of acetylcholine (Ach) at doses of 50, 100, and 200 μg into the LCA did not cause any chest symptoms or angiographic coronary spasm (left panel). Second, infusions of ergonovine maleate (EM) at doses of 20, 40, and 60 μg into the LCA did not cause any chest symptoms or angiographic coronary spasm (middle panel). Finally, the infusion of ACh at a dose of 200 μg into the LCA caused chest pain, ST-T changes on an ECG, and angiographically severe vasoconstriction at the segment of the myocardial bridge (right panel). Arrows, spastic segments. LCA: left coronary artery, ECG: electrocardiogram**

portion of the right coronary artery. He had an angiographically normal left coronary artery (LCA); however, a myocardial bridge was present at the mid-segment of the left anterior descending coronary artery (LAD; Fig. 1). Based on the initial angiograms, he was diagnosed with MINOCA.

To determine the cause of MINOCA, we focused on the presence of the myocardial bridge at the mid-segment of the LAD. We hypothesized that a coronary spasm at the myocardial bridge may have resulted in MINOCA. We therefore first performed SPT using acetylcholine (ACh) infusion. ACh was injected in incremental doses of 50, 100, and 200 μg into the LCA. CAG revealed no significant coronary spasm in the LCA (Fig. 2, left panel). Next, we performed SPT using ergonovine maleate (EM); 20, 40, and 60 μg EM was injected into the LCA, and no positive findings were observed in response to EM (Fig. 2, middle panel). Finally, we injected an additional 200 μg of ACh into the LCA following EM provocation of the LCA. The patient experienced chest pain, which was similar to that before admission, and the ECG revealed negative T in V2-V4. CAG exhibited severe narrowing at the mid-segment of the LAD, which corroborated the segment of the myocardial bridge (Fig. 2, right panel). Upon NTG injection, the coronary spasm resolved, and the patient was diagnosed with vasospastic angina (VSA), which was the cause of MINOCA. He was discharged four days later and prescribed long-acting nifedipine and a strong statin. He has not experienced any chest pain in the five months since his discharge.

Discussion

We encountered a case of VSA diagnosed by an SPT after intravenous administration of NTG in emergent CAG. We thus established that the coronary spasm caused MINOCA. In addition, the presence of the myocardial bridge prompted us to aggressively perform an SPT in the present

case.

MINOCA is considered a working diagnosis with multiple potential causes (1, 2, 13). In the present case, potential causes of MINOCA correlating with the coronary artery were considered, including coronary plaque disease, coronary dissection, coronary spasm, and coronary microvascular angina. Indeed, coronary spasm is one of the major causes of MINOCA, being observed in 3% to 95% of patients with MINOCA, depending on the timing of SPT and use of provocative drugs (1-3, 13). Recently, Montone et al. (14) reported that positive SPT results were found in 46.2% of patients with MINOCA, and epicardial coronary spasm was detected in 30% of patients with MINOCA. In addition, they reported that MINOCA patients with positive SPT results exhibited a worse prognosis than those with negative SPT results (14). This emphasizes the significance of performing SPT to determine the cause of MINOCA.

In the present case, we performed the SPT during emergent CAG after the intravenous administration of NTG and determined that the coronary spasm had caused MINOCA. The SPT timing in our case might be controversial because intravenous NTG had been administered before the SPT. Furthermore, according to the VSA guidelines (15), the SPT is generally not recommended for patients with acute coronary syndrome. When the coronary spasm does not disappear immediately in patients with an impaired LVEF, there may be another important underlying problem. Taking these findings into consideration, we believe that it would be more appropriate to perform emergent CAG to assess significant coronary stenosis. In addition, a second CAG session must be performed a few days later to determine the presence of coronary spasm. However, in reality, performing CAG twice in one patient might not be feasible at all hospitals. Furthermore, the SPT is reportedly safe in the acute phase in patients with MINOCA (14). The present case had a preserved LV systolic function; therefore, we performed the SPT, including a sequential SPT, after the intravenous administration of NTG. The sequential SPT involves spasm provocation using an initial ACh infusion, followed by an EM infusion, and finally another ACh infusion, which causes a higher frequency of spasm provocation without increasing the risk of severe complications (16, 17). Under such conditions, if NTG has been administered before the SPT (as in our case), a sequential SPT is extremely useful (16-19).

Several recent studies have focused on the correlation between the myocardial bridge and coronary spasm, demonstrating the presence of coronary spasm at segments with a myocardial bridge (5-12, 20). The precise mechanism underlying the correlation has not yet been established. However, abnormal coronary vasomotor at the segments of the myocardial bridge, including endothelial dysfunction (5) and/or abnormal adventitial vasa varum of the coronary artery (21), might be associated with myocardial bridge-related coronary spasm. Naturally, a number of cases of harmless myocardial bridges may be reported in the clinical setting; however, the possibility of coronary spasm should be suspected in patients with chest symptoms and myocardial bridge.

Conclusion

In conclusion, we herein report case of a patient with MINOCA, in whom sequential SPT in emergent CAG after administering NTG revealed that coronary spasm caused MINOCA. We used the myocardial bridge as a hint for the diagnosis. It is critical to determine the cause of MINOCA, as its prognosis can be affected by the cause. Therefore, cardiologists should remember to perform an SPT, including a sequential SPT, to determine the cause of MINOCA. Furthermore, the presence of a myocardial bridge might be a clue for diagnosing a coronary spasm.

The authors state that they have no Conflict of Interest (COI).

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