Coronary Spastic Angina Causing Myocardial Stunning
Associated with Reversible Wall Thinning and Motion
Abnormality of the Left Ventricle: a Case Report

Toshiaki Isogai, Hiroyuki Tanaka, Motohiro Asaki, Tetsuro Ueda

We describe a rare case of coronary spastic angina (CSA) causing myocardial stunning with reversible wall thinning and motion abnormality of the left ventricle (LV). A 70-year-old woman presented with sudden resting angina. A 12-lead electrocardiogram (ECG) showed ST-segment elevation in leads I, aVL, and V2–V6 and reciprocal ST-segment depression in leads II, III, and aVF. The symptoms were resolved with an intravenous injection of isosorbide dinitrate. A transthoracic echocardiogram demonstrated akinesis in the region from the apex to the mid-anterior LV. Urgent coronary angiography did not demonstrate an obstructed coronary artery. Cardiac magnetic resonance imaging (MRI) identified thinning of the anterior wall in the akinetic region, without any late gadolinium enhancement. Cardiac single photon emission computed tomography (SPECT), using 123I-iodine beta methyl-iodophenyl pentadecanoic acid (123I-BMIPP) and 201thallium (201Tl), showed a mismatch congruent with the akinetic region. An acetylcholine provocation test performed during hospitalization revealed multivessel coronary spasms in all 3 epicardial arteries. After initiation of treatment with coronary vasodilators, the wall thinning and motion abnormality gradually recovered to the normal range. In the present case, CSA caused myocardial stunning associated with reversible wall thinning and motion abnormality in the acute phase. CSA can occasionally lead to two conditions associated with LV wall abnormalities: myocardial infarction and stunning. Although the findings of the 2 conditions are similar in the acute phase, stunning can be differentiated from infarction using multiple imaging modalities. Cardiac MRI and SPECT are useful tools for determining whether the myocardium is infarcted or stunned.

KEY WORDS: coronary spastic angina, myocardial stunning, myocardial infarction, magnetic resonance imaging, single photon emission computed tomography
To determine the cause of the patient’s resting angina, an acetylcholine provocation test was performed on hospitalization day 7. The test revealed multivessel coronary spasms in all 3 epicardial arteries (segment 7, 99% with delay; segment 14, 99%; segment 4AV, 99% with delay) in conjunction with her chest symptoms (Fig. 2, lower images). Therefore, we diagnosed her with coronary spastic angina (CSA). The patient began taking a calcium channel blocker (benidipine, 8 mg/day) and a potassium channel opener (nicorandil, 30 mg/day), and her chest symptoms did not reappear.

Over the course of hospitalization, the patient’s ECGs changed as shown in Fig. 1. The leads with ST-segment elevation showed T-wave inversion on hospitalization day 2. Subsequently, T-
waves with QT prolongation appeared deeper, especially in the precordial leads and persisted over 2 months. The T-waves slowly recovered, but the terminal T-waves remained slightly inverted 6 months after discharge.

The LV ejection fraction, as evaluated by echocardiography, gradually recovered during the patient’s hospitalization. On post-admission day 18, she was discharged without any symptoms. A 6-month post-discharge cardiac MRI showed full recovery of LV wall thickness and motion (Fig. 4). On the basis of these results, we concluded that CSA caused myocardial stunning associated with reversible wall thinning and wall motion abnormality of the LV without myocardial infarction.

**Discussion**

We treated a rare case of CSA that produced a reversible wall abnormality. Upon admission, the initial clinical suspicion was that myocardial infarction caused sudden resting angina and LV abnormality because of the typical episodes and ECG changes. However, urgent coronary angiography failed to show obstructed coronary arteries. During the acute phase, a cardiac MRI did not detect LGE, despite the wall abnormality. Cardiac SPECT also showed metabolic changes congruent with the damaged region. As a result, the clinical condition was determined to be myocardial stunning rather than infarction.

Myocardial stunning is a post-ischemic mechanical dysfunction that persists after reperfusion despite the absence of irreversible cell necrosis. The benefits of reperfusion therapy might be delayed, but occur within hours to days after reperfusion. Consequently, full recovery might be delayed for some weeks or months. The diagnosis of myocardial stunning requires evidence that (1) the contractile abnormality is reversible, with preservation of cardiac viability, and (2) the dysfunctional myocardium retains normal or near-normal flow. Myocardial stunning is caused by organic stenosis or coronary artery spasms, both of which are associated with severe, acute ischemia.

Prinzmetal et al first described CSA as a variant of angina in 1959. CSA is caused by episodic coronary artery spasms and is associated with transient ST-segment elevation. Each spasm is a reversible stenosis that limits coronary blood flow under resting conditions, occasionally causing myocardial infarction or stunning in cases of severe and prolonged ischemia. Because both myocardial infarction and stunning show similar chest symptoms, elevated cardiac markers, ECG findings, and LV wall abnormalities, cardiac MRI and SPECT are used to differentiate between the 2 conditions in our hospital.

Cardiac MRI with a gadolinium-based contrast agent offers high spatial resolution and can identify myocardial infarction or fibrosis. A previous study showed that LGE in cardiac MRI has a higher sensitivity and specificity for determining infarction than does SPECT. Despite the presence of myocardial stunning, myocardial injury without necrosis does not show LGE. In addition, cardiac MRIs clearly demonstrate mechanical changes in the heart. In our hospital, a patient with suspected myocardial stunning was treated with CSA.
dial infarction or stunning which demonstrates LGE in the transmural or subendocardial area covered by a stenotic coronary artery is diagnosed with myocardial infarction. Cardiac SPECT is useful to demonstrate myocardial stunning. At-rest imaging with \(^{123}\)I-BMIPP after an ischemic episode can identify regions of suppressed fatty acid metabolism as ischemic memory. Despite restoration of blood flow, as identified by at-rest imaging with \(^{201}\)TI, prolonged and persistent disturbances in fatty acid metabolism represent a scintigraphic marker of myocardial stunning.

In the present case, the patient’s heart was not infarcted, but stunning was evident based on her cardiac MRI and SPECT results. Subsequently, since she had experienced similar episodes of resting angina before her present admission, we performed an acetylcholine provocation test to determine the cause of her myocardial stunning. Results of this test showed that CSA caused her angina and severe ischemia on admission, subsequently leading to myocardial stunning. To prevent an angina attack or recurrent myocardial stunning, medical therapy for the CSA is critical.

Before reaching this diagnosis, we also considered the possibility that her clinical condition was a type of takotsubo cardiomyopathy (TC) triggered by physical and emotional stress, and associated with CSA. However, we diagnosed her with CSA because (1) the reciprocal changes observed in her ECG on admission are infrequently observed in TC cases; (2) CSA was identified as the cause of her resting angina, and (3) the LV wall motion in the acute phase did not show the typical TC apical ballooning pattern. Nevertheless, we could not rule out a diagnosis of TC because the mechanisms underlying TC remain unknown, and TC may account for some aspects of myocardial stunning.

In conclusion, the present case was interesting and instructive in demonstrating mechanical and metabolic changes associated with myocardial stunning through the use of cardiac MRI and SPECT. Myocardial stunning is difficult to differentiate from myocardial infarction in the acute phase, because of the similarities between the findings in the 2 conditions. However, differentiation should be attempted by using multiple imaging modalities. As demonstrated in the present case, cardiac MRI and SPECT are useful tools for achieving this differentiation and determining whether the myocardium is infarcted or stunned.

References

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