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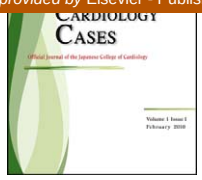

Case Report

Coronary spasm as a cause of takotsubo cardiomyopathy and intraventricular obstruction

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KEYWORDS
Coronary spasm;
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Summary A 79-year-old man presented to the emergency room because of chest pain on 3 successive mornings. An electrocardiogram (ECG) showed ST segment elevation in leads II, III, and aVF. Laboratory findings including cardiac enzymes, were within normal limits, except a positive result for the troponin T test. Two-dimensional echocardiography revealed akinesis of the left ventricular apex and hyperkinesis of the basal wall. Doppler echocardiography revealed a significant subaortic pressure gradient. Emergent coronary angiography showed no significant coronary artery stenosis, but the ergonovine test induced a right coronary artery spasm with exaggeration of the ST segment elevation in II, III, and aVF leads. The computed tomography performed 2 weeks later showed normal left ventricular wall motion with sigmoid septum. The patient was diagnosed with takotsubo cardiomyopathy and intraventricular obstruction due to coronary spasm; he was treated with calcium channel blockers and nitrates. This case suggests the importance of differential diagnosis of the pathogenesis of takotsubo cardiomyopathy.

Introduction

Takotsubo cardiomyopathy is an acute myocardial infarction-like disorder showing transient akinesis of the left ventricular apex without significant coronary artery stenosis. Although the exact mechanisms of takotsubo cardiomyopathy are unknown, excessive sympathetic stimulation and catecholamine release are considered to be the major causes [1–4]. We report a case of takotsubo cardiomyopathy caused by coronary spasm.

Case report

A 79-year-old man presented to the emergency room because of chest pain on 3 successive mornings. Physical examination revealed a grade 3 systolic murmur at the right sternal border in the second intercostal space. The lungs were clear. An electrocardiogram (ECG) showed: poor R wave progression in leads V1 to V4; ST segment elevation in leads II, III, and aVF; and negative T waves in leads V4 to V6 (Fig. 1, left). Laboratory findings, including car-

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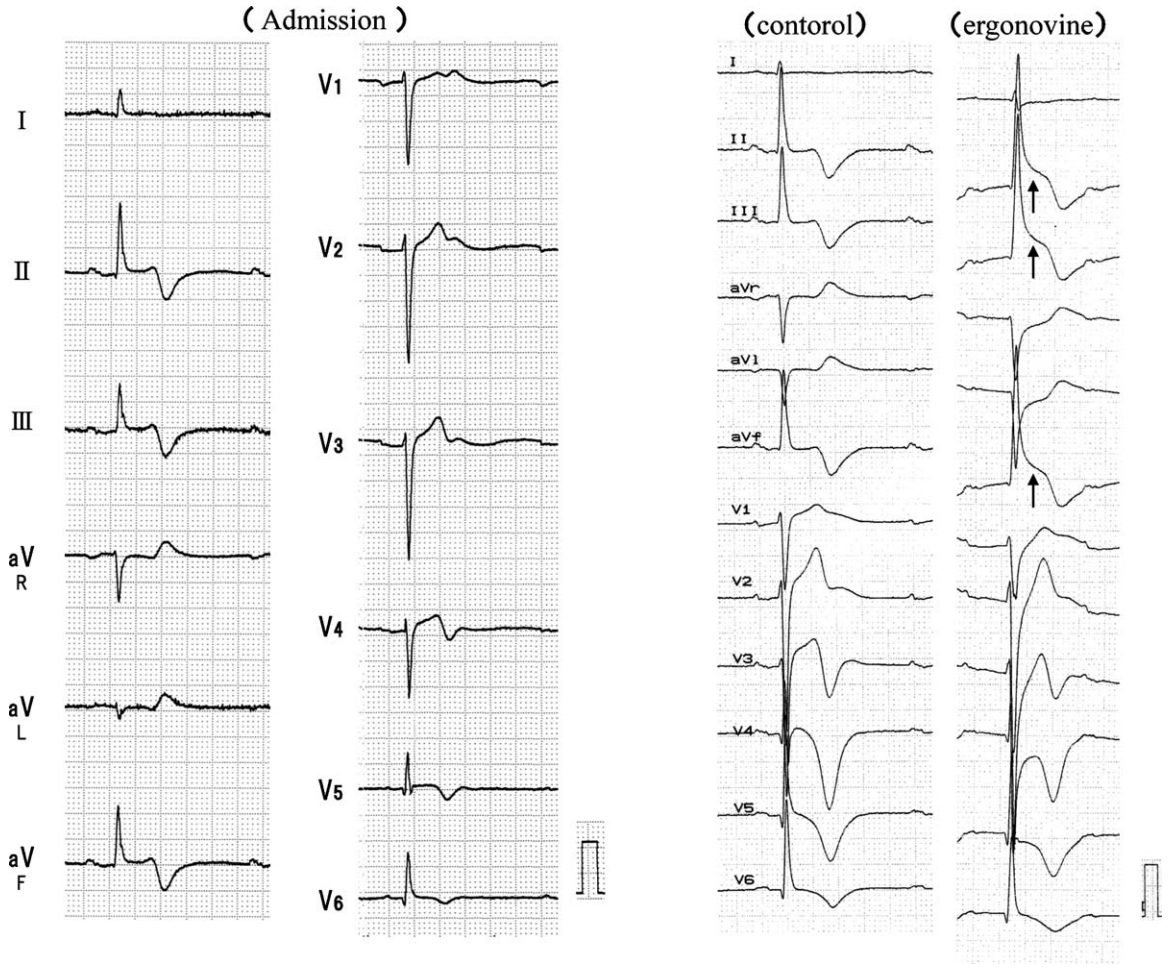

Figure 1 Electrocardiogram (ECG) on admission (left) and during the ergonovine test (right). On admission, ECG showed poor R wave progression in leads V1 to V4; ST segment elevation in leads II, III, and aVF; and negative T waves in leads V4 to V6. After intracoronary administration of ergonovine, exaggeration of ST segment elevation was observed.

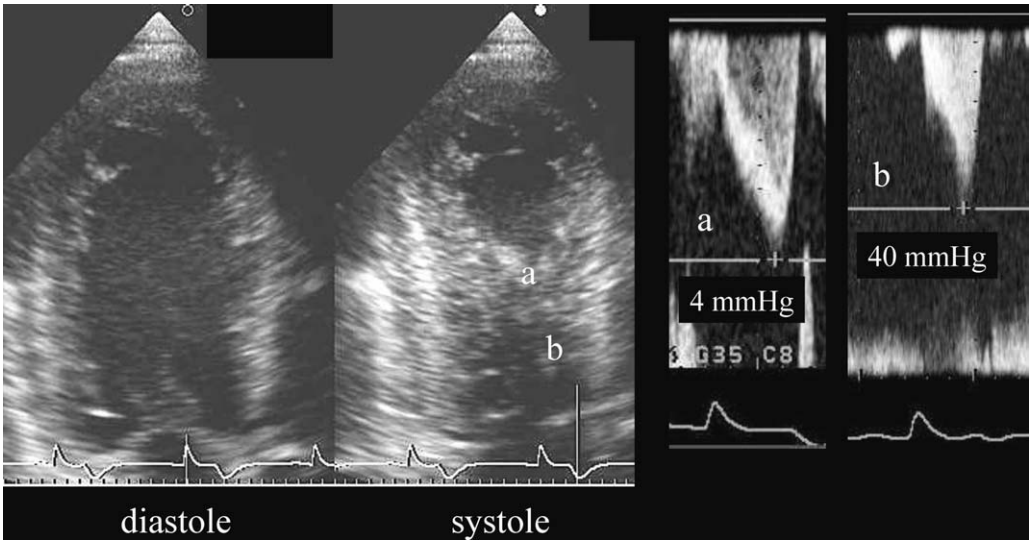

Figure 2 Transthoracic echocardiography on admission. Two-dimensional echocardiography (left) showed apical ballooning at the apex and hyperkinesis of basal wall. Doppler echocardiography (right) showed a slight pressure gradient at the mid-ventricle (a; 4 mm Hg) and significant pressure gradient at the left ventricular outflow (b; 40 mm Hg).

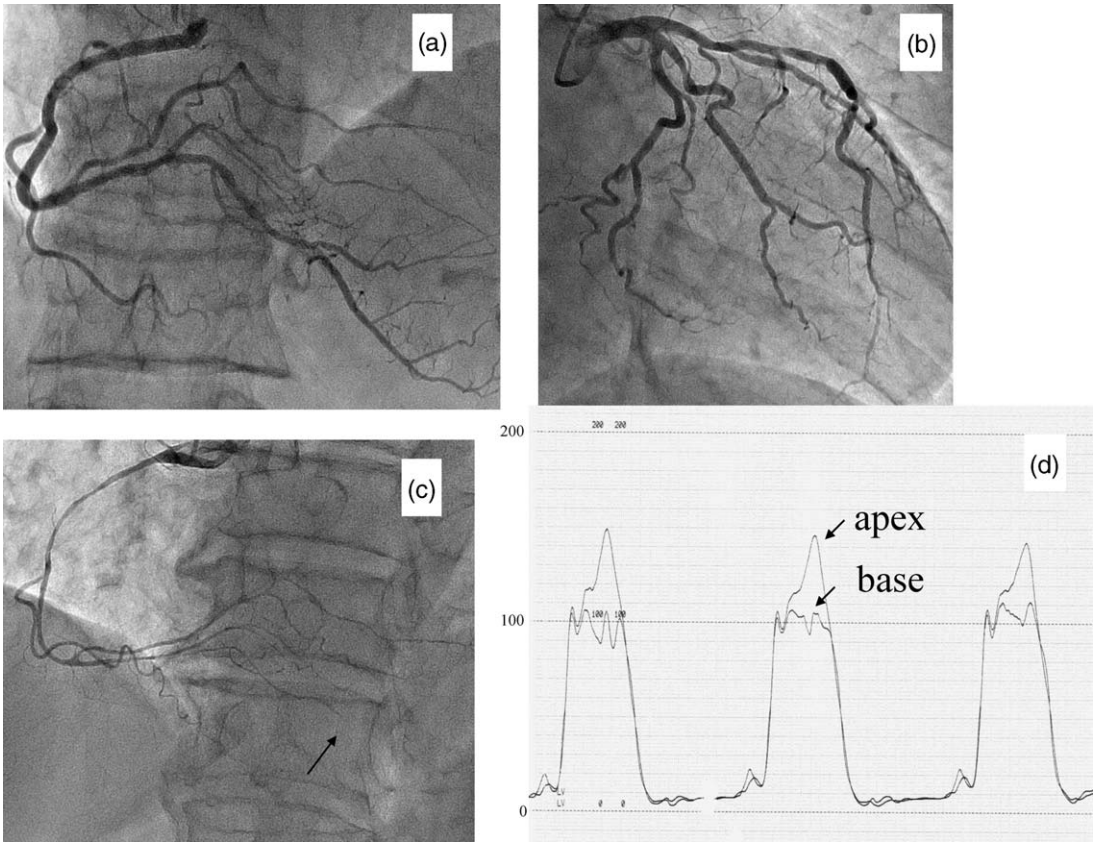

Figure 3 Emergent coronary angiography showed no significant coronary artery stenosis (a and b). After intracoronary administration of ergonovine, the right coronary artery showed diffuse spasticity, with total occlusion of the posterior descending artery (c, arrow). A pressure study revealed an intraventricular pressure gradient (d).

diac enzymes, were within normal limits except a positive result for the troponin T test. Two-dimensional echocardiography revealed akinesis of the left ventricular apex and hyperkinesis of the basal wall. Doppler echocardiography revealed a mid-ventricular pressure gradient of 4 mm Hg and a subaortic pressure gradient of 40 mm Hg (Fig. 2). Emergent coronary angiography showed no coronary artery stenosis (Fig. 3a and b). After injection of 20 μg of ergonovine [5,6] into the right coronary artery, the patient complained of severe chest pain; an ECG showed exaggeration of the ST segment elevation in II, III, and aVF leads (Fig. 1, right), and coronary angiography revealed diffuse vasospasm with total

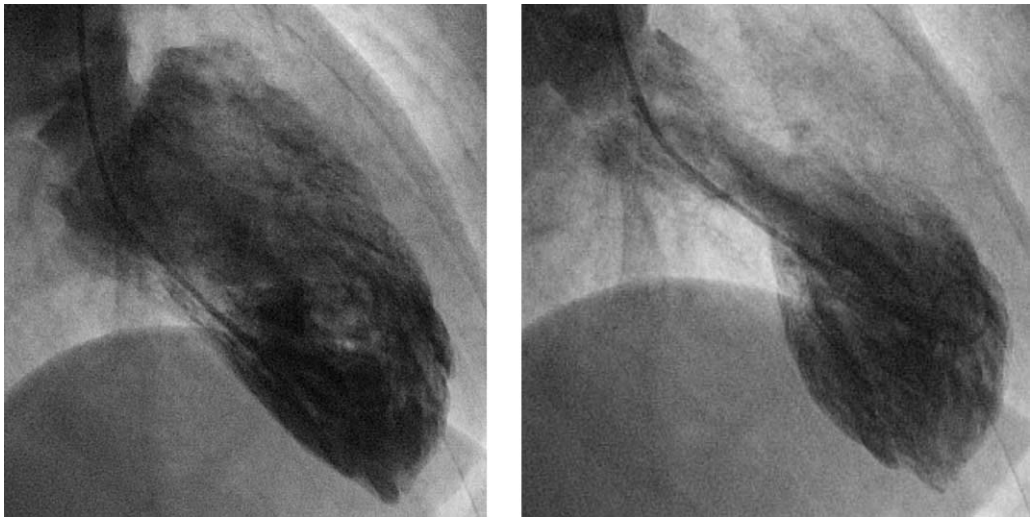

Figure 4 Left ventriculography showed systolic ballooning of the apex and hypercontraction of the basal segment.

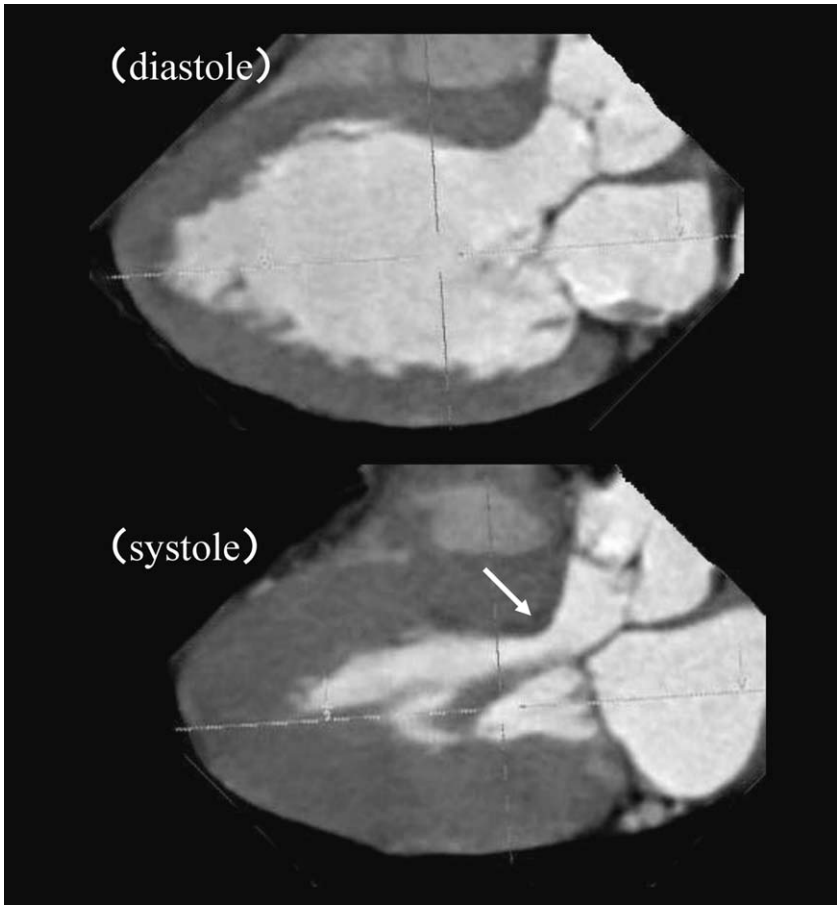

Figure 5 Left ventricular images in diastole (upper) and systole (lower) by computed tomography showing normal left ventricular wall motion with sigmoid septum (arrow). There was no longer left ventricular outflow obstruction.

obstruction at the posterior descending artery (Fig. 3c). Coronary spasm was resolved by the intracoronary administration of isosorbide dinitrate.

Left ventriculography showed systolic ballooning of the apex and hypercontraction of the basal segment (Fig. 4). A pressure study revealed a systolic pressure gradient between the apex and the basal segment (Fig. 3d). To disclose specifics of left ventricular outflow obstruction, the patient underwent computed tomographic (CT) scan 2 weeks later. Cine-CT images unveiled normal left ventricular wall motion with sigmoid septum. There was no longer left ventricular outflow obstruction (Fig. 5).

The patient was diagnosed with takotsubo cardiomyopathy and intraventricular obstruction due to coronary spasm; he was treated with calcium channel blockers and nitrates. After 1 month, echocardiography revealed normal left ventricular wall motion and no intraventricular outflow obstruction.

Discussion

Takotsubo cardiomyopathy is frequently observed in old women after acute emotional or physiological stress [7]; many clinical and laboratory findings have suggested the significance of catecholamine injury in the pathogenesis of this cardiomyopathy [1—4]. Although coronary spasm is observed in a few patients [8], its correlation with takotsubo cardiomyopathy is unclear because of the low proportion of and discrepancy in the sites of myocardial ischemia and ballooning apex [3].

The patient was male and did not have any stress, but he felt a typical chest pain in the early morning on 3 successive days. ECG on admission showed ST segment elevation in the inferior leads; this elevation became prominent after the ergonovine-induced coronary spasm. This change in the ECG findings suggests that the ECG findings on admission were due to right coronary artery spasm. Moreover, although examination for multivessel spasm was not performed because of the rapid administration of isosorbide, diffuse coronary spasm with distal occlusion is consistent with apical ballooning.

Intraventricular systolic pressure gradients were documented by both Doppler echocardiography and cardiac catheterization. The pressure gradient was mild at the mid-ventricle (4 mm Hg), and significant at the outflow tract (40 mm Hg). The ballooning apex could not produce a systolic pressure gradient at the mid-ventricle, whereas the compensatory hyperkinetic base produced a significant pressure gradient by vigorous ejection of blood and dynamic stenosis of the left ventricular outflow [8].

Patients with a sigmoid septum, small left ventricular outflow, and reduced left ventricular volumes have a geometric predisposition to dynamic left ventricular outflow obstruction [9], which may manifest in the setting of intense adrenergic stimulation or compensatory hyperkinesis of basal segment [10]. In this patient, sigmoid septum and coronary spasm caused transient left ventricular outflow obstruction, which increased oxygen demand at mid-to-apical cavity by elevating left ventricular filling pressures. Decreased oxygen supply by coronary spasm and microvascular dysfunction [11] also caused apical ischemia.

Although the exact mechanisms of takotsubo cardiomyopathy are unknown, coronary spasm and transient left ventricular obstruction played a pivotal role in this case. As medications for stress cardiomyopathy and coronary spasm are different, this case suggests the importance of diagnosing the pathogenesis of takotsubo cardiomyopathy.

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