Transient left ventricular apical ballooning syndrome is a unique, reversible cardiomyopathy usually found in postmenopausal women, presenting with evolitional electrocardiographic changes and elevated cardiac biomarkers suggestive of acute ST-segment elevation myocardial infarction without angiographic coronary artery stenosis. We present a 62-year-old diabetic female with typical pictures of transient left ventricular apical ballooning syndrome. Acute anterior myocardial infarction was diagnosed initially based on the marked electrocardiographic changes and elevated troponin. However, coronary angiography showed no significant stenosis. ST-segment elevation of the electrocardiogram returned to baseline with development of T-wave inversion about one week later. We used three-dimensional images by dual-source multidetector computed tomography to well demonstrate the unique, reversible change of the left ventricle. The patient had a good recovery under supportive therapy. Transient left ventricular apical ballooning syndrome is an important differential diagnosis of acute coronary syndrome.

Key Words: Acute myocardial infarction • Apical ballooning syndrome • Multidetector computed tomography • Takotsubo cardiomyopathy

INTRODUCTION

Transient left ventricular (LV) apical ballooning syndrome, also named “Takotsubo cardiomyopathy”, is characterized by transient LV wall motion abnormalities accompanied by electrocardiographic (ECG) changes and elevated cardiac biomarkers that can mimic acute myocardial infarction (AMI) in the absence of angiographic coronary artery stenosis.1 “Takotsubo” means octopus-trapping pot in Japanese. Owing to its clinical presentation, this syndrome is indistinguishable from an acute coronary syndrome and acknowledged by the American College of Cardiology and American Heart Association as a unique form of reversible cardiomyopathy.2 We report a 62-year-old female admitted due to diabetic ketoacidosis with typical transient left ventricular apical ballooning syndrome. We demonstrated the three-dimensional, reversible changes of apical ballooning by dual-source multidetector computed tomography (DSCT) instead of left ventriculography to show a more clear appearance of “takotsubo”.

CASE REPORT

This 62-year-old female had a history of diabetes mellitus. She had received irregular diabetic control previously. Since four days before this admission, she had complained of dizziness, general malaise and nausea. She was told she had a cold by the local clinic. However, progressive dyspnea and change of consciousness were noted in the morning on April 18, 2007. Initial labora-
tory data at our emergency room showed arterial blood gas PH 6.778, PCO₂ 15.9 mmHg, PO₂ 59 mmHg, HCO₃⁻ 2.3 mmol/L; blood sugar 496 mg/dl; serum ketones 80 mg/dl; creatinine 1.4 mg/dl; sodium 131 mmol/L; potassium 7.3 mmol/L; hemoglobin 14.7 g/dl and troponin I 0.471 ng/ml. The patient was then admitted to our intensive care unit under the impression of diabetic ketoacidosis. She underwent intubation due to respiratory failure. Physical examination was unremarkable except for fever and stupor consciousness. The brain computed tomography showed right bundle branch block. Chest X-ray (CXR) showed normal heart size and no obvious pulmonary congestion. On the second day after admission, new electrocardiographic change with diffuse ST-segment elevation, especially in the precordial leads, was noted (Figure 1A). Bedside echocardiography revealed anterior and apical hypokinesis. Under the impression of acute myocardial infarction of the extensive anterior wall, the patient underwent emergent cardiac catheterization. Coronary angiography displayed patent coronary artery. Maximal troponin I release was 4.79 ng/ml. The cardiac computed tomography exhibited characteristic apical ballooning of the left ventricle in the systolic phase (Figure 2A). “Takotsubo”-like shape of the left ventricle was demonstrated in three-dimensional reformatted image (Figure 2B). ST-segment elevation of the electrocardiogram lasted for 3 days and then returned to baseline with development of T-wave inversion gradually (Figure 1B). The patient’s condition was stabilized under supportive therapy. She was discharged with good recovery two weeks later. The follow-up images after one month indicated nearly complete regression of apical ballooning (Figures 2C & 2D).

**DISCUSSION**

Transient left ventricular apical ballooning syndrome was first described in the Japanese population by Sato et al. and Dote et al.⁴ and has been reported now worldwide. This syndrome was initially named as Tako-
tsubo cardiomyopathy for the similarities in morphology with the characteristic appearance of the left ventricle during systole. In patients presenting with an acute coronary syndrome, 1.5% to 2.2% appeared to have apical ballooning syndrome. It is possibly induced by acute emotional or physical stress, usually in postmenopausal women, who accounted for 82% to 100% of patients, with an average age of 62 to 75 years. ECG abnormalities with ST-segment elevation in the precordial leads were noted in > 90% of patients. Evolutionary deep symmetrical T-wave inversions develop within 24 to 48 hours after presentation. A minor elevation in cardiac biomarkers is common. Despite the clinical presentation mimicking acute myocardial infarction, patients do not have obstructive coronary artery disease identifiable from the angiography. The left ventriculography shows regional systolic dysfunction involving the mid and apical portions (arrows). Excess residual contrast found in the apex of the right ventricle (RV) may implicate RV involvement. (C) and (D) show the apical ballooning regressed and LV returned to normal morphology in the follow-up study. (B) and (D) are three-dimensional reconstruction images shown in the acute and recovery stages.

In dual-source multidetector computed tomography, the contrast agent is filled in the left atrium (LA), left ventricle (LV), and aortic root (Ao). (A) and (B) show the characteristic takotsubo-like shape of LV during the systolic phase, indicating myocardial akinesia or dyskinesia in the mid and apical portions (arrows). Excess residual contrast found in the apex of the right ventricle (RV) may implicate RV involvement. (C) and (D) show the apical ballooning regressed and LV returned to normal morphology in the follow-up study. (B) and (D) are three-dimensional reconstruction images shown in the acute and recovery stages.

Figure 2.

In conclusion, the manifestations of transient left ventricular dysfunction (akinesia or dyskinesia) during systole are characteristic of takotsubo cardiomyopathy. The underlying pathophysiology remains unknown, and it is likely related to enhanced sympathetic tone and excessive catecholamine stimulation. The proposed mechanisms include multi-vessel epicardial spasm, coronary microvascular spasm, and catecholamine-induced myocardial stunning. The recurrence rate is less than 10%. Similar cardiac changes were also noted in cases of cerebrovascular accidents and pheochromocytoma. The optimal management is supportive therapy. According to its possible pathophysiology, long-term β-blocker therapy for preventing recurrence is reasonable. The transient use of angiotensin-converting enzyme inhibitors or angiotensin receptor blockers should be considered for impaired LV function. Anticoagulant may be considered for preventing LV thrombus formation and embolization. There is usually a good prognosis in the absence of significant underlying comorbid conditions. The in-hospital mortality is 1% to 3%. Normalization of LV function occurs in 1 to 3 months mostly.

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ventricular apical ballooning syndrome mimic acute myocardial infarction, and those who have ECG changes like our patient may risk undergoing thrombolytic therapy in hospitals without an on-site interventional cardiologist. It is important to keep this in mind in the differential diagnosis of acute coronary syndrome.

REFERENCES


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