Acute Pericarditis in the Recovery Phase of Transient Left Ventricular Apical Ballooning Syndrome (Takotsubo Cardiomyopathy)

Takahisa Maruyama, Takeshi Hanaoka and Hiroyuki Nakajima

Abstract

A 64-year-old woman was admitted because of persistent chest pain and ST-elevation in the precordial and inferior leads on electrocardiogram. Emergent coronary angiography demonstrated that there was no obstruction, and left ventriculography showed apical akininesis and basal hyperkinesis. She was diagnosed with takotsubo cardiomyopathy. Chest pain subsided within the day of admission followed by T-wave inversion on electrocardiogram. Nevertheless, chest pain and ST-elevation reappeared late on the third hospital day, accompanied by fever and small amounts of pericardial and pleural effusions. Under the administration of nonsteroidal anti-inflammatory drug, the inflammation lessened. This is the first report of acute pericarditis in the recovery phase of takotsubo cardiomyopathy.

Key words: takotsubo cardiomyopathy, acute pericarditis, complication, apical ballooning

(DOI: 10.2169/internalmedicine.46.0184)

Introduction

A syndrome with transient left ventricular dysfunction mimicking acute myocardial infarction has been reported since the 1990s, and many of the cases were from Japan (1-4). In the acute stage of the syndrome, there is no obstruction in the coronary arteries, and the left ventricle is shaped like a takotsubo, a Japanese word for a pot (tsubo) with round bottom and narrowed neck used to fish for octopus (tako). Therefore, this syndrome has been called “takotsubo cardiomyopathy” as well as “transient left ventricular apical ballooning syndrome”. Although sympathetic nerve activity and catecholamines are suggested to play an important role in this syndrome, the precise mechanism has been unknown (5). The prognosis of this syndrome seems to be favorable, but there are several complications including left-sided heart failure (2, 4). Among them, acute pericarditis in the recovery phase has not been reported.

Case Report

A 64-year-old woman consulted our hospital complaining of persistent chest pain, which had occurred while she was singing in the previous day afternoon, and then somewhat attenuated during the night. Her pulse rate was 62 beats/min and the blood pressure was 118/70 mmHg. She was afebrile, and neither heart murmurs nor friction sounds were heard. Electrocardiogram (ECG) demonstrated a normal sinus rhythm and ST-segment elevation in leads II, III, aVF, and V3 through V6 (max.+2.5 mm in V5) without abnormal Q-wave formation (Fig. 1). The chest x-ray study did not show any pulmonary congestion or pleural effusion. On echocardiography, the apical region of the left ventricle was akinetic, and there was no evidence of pericardial effusion. Laboratory studies demonstrated small increases in cardiac enzymes: creatine kinase, 317 IU/l (normal, 32-170 IU/l); creatine kinase-MB isoenzyme, 33 IU/l (<16 IU/l); troponine T, positive (by qualitative analysis), whereas the C-reactive protein was not elevated (0.15 mg/dl). Emergent cardiac catheterization study was performed during persistent chest pain and ST-elevation. Coronary angiography demonstrated only minimal sclerotic changes and good flow in the arteries. On left ventriculography, the apex was akinetic with a ballooning appearance on systole, the extent of which was not matched with coronary arterial distribution,

Received for publication March 22, 2007; Accepted for publication July 25, 2007
Correspondence to Dr. Takahisa Maruyama, maruyama@hospital.nagano.nagano.jp
whereas the basal region was hyperkinetic (Fig. 2). Left ventricular end-diastolic pressure was 5 mmHg, and there was no intra-ventricular pressure gradient. Thus, transmural ischemia due to organic obstruction or spasm was ruled out, and takotsubo cardiomyopathy was diagnosed.

Heparin was administered intravenously to prevent the formation of left ventricular mural thrombus, but vasoactive agents were not prescribed because the hemodynamic condition was stable. Chest pain was completely resolved by the evening of the day of admission. The level of creatine kinase was decreased on serial samplings. On ECG obtained the next day, T-waves were inverted in leads where the ST-segment had been elevated (Fig. 1).

At midnight on the third hospital day, the patient complained of chest pain again. In contrast with the previous symptoms on admission, this recurrent pain radiated broadly to the anterior chest and back, and was exacerbated by inspiration or body movements. The ECG showed re-elevation of the ST-segments in leads I, II, III, aVL, aVF, and V2 through V6, which became more prominent the following
day (Fig. 1). On echocardiography, a small amount of pericardial effusion was present, although left ventricular apical movement had almost normalized (Fig. 3A). A slight pleural effusion was also noted on chest x-ray study (Fig. 3B). Her temperature rose to 38.3°C, followed by the elevation of C-reactive protein level (Fig. 1). Administration of nonsteroidal anti-inflammatory drug relieved the chest symptoms and fever. The signs of the inflammation improved within several days, and the T-wave on ECG again inverted deeply (Fig. 1).

Discussion

In the present case, a postmenopausal woman complained of prolonged chest pain with ECG showing ST-elevation, although there was no obstruction in her coronary arteries. Transient akinesis of the left ventricular apex was documented, with a small increase in cardiac enzyme levels. These were typical features compatible with takotsubo cardiomyopathy (2-4). Although an acute emotional or physical stress often precedes the onset of takotsubo cardiomyopathy, in about a quarter of the patients with this syndrome the triggering episodes are not remarkable, as in the present case (2).

The most important complication of this cardiomyopathy is left-sided heart failure leading to pulmonary congestion or cardiogenic shock (2, 4). In some patients, transient intraventricular pressure gradient or mitral regurgitation may deteriorate the hemodynamic status. Ventricular or atrial arrhythmias are also reported. Mural thrombus can be formed in the akinetic area of left ventricle (6). Cardiac rupture was reported as a rare complication (7). However, there are no reports describing acute pericarditis in the recovery phase of takotsubo cardiomyopathy.

In our patient, chest pain subsided and the ECG finding of ST-elevation evolved to inverted T wave once within the early days of hospitalization. Nevertheless, chest pain and ST-elevation reappeared with fever, a rise in C-reactive protein level, and symptoms and signs suggesting pleural involvement, which were compatible with acute pericarditis (8, 9).

Regarding the sequence of the phenomena in the present case, takotsubo cardiomyopathy developed first and acute pericarditis occurred subsequently. However, the relationship between two conditions, takotsubo cardiomyopathy and acute pericarditis, was not clear. We speculated on the following two possibilities. First, as in the symptom order, takotsubo cardiomyopathy might come first. Transmural myocardial damage might extend to the inflammation of pericardium, as though a transmural acute myocardial infarction is complicated with acute pericarditis in the early stage of infarction (10). In previous reports and in our experiences with takotsubo cardiomyopathy, the durations of ST-elevation are variable (3). In some cases, the ST-elevation persisted for several days, although in other cases the ST-elevation promptly evolved to giant negative T wave. In the former cases, the asymptomatic inflammation of pericardium might contribute to the prolonged ST-elevation.

Second, the course of the present case might be interpreted as myopericarditis (11). The transient apical ballooning might be a form of myocardial inflammation and the pericardial involvement followed, although the etiological association of myocarditis with takotsubo cardiomyopathy is not supported by the previous studies (3, 12).

Takotsubo cardiomyopathy involves several characteristic features with unique left ventricular movement, but is not defined precisely (4). An effort to make guidelines for diagnosis of this syndrome is currently underway in Japan (13). Associations between takotsubo cardiomyopathy and various clinical conditions will be further reported in the future.

References


© 2007 The Japanese Society of Internal Medicine
http://www.naika.or.jp/imindex.html