Journal of Cardiology Cases 22 (2020) 45–47

Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

Takotsubo syndrome triggered by coronary artery embolism in a patient with chronic atrial fibrillation

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ARTICLE INFO

Article history:
Received 6 September 2019
Received in revised form 6 January 2020
Accepted 11 January 2020

Keywords:
Takotsubo syndrome
Coronary artery embolism
Chronic nonvalvular atrial fibrillation
Spontaneous echo contrast

ABSTRACT

Although takotsubo syndrome is defined as a reversible heart failure syndrome with the absence of obstructive coronary artery disease, some cases of concomitant takotsubo syndrome and acute myocardial infarction have been reported. We herein describe the case of a patient with chronic nonvalvular atrial fibrillation who was not receiving anticoagulant therapy, who developed takotsubo syndrome triggered by acute myocardial infarction probably due to coronary artery thromboembolism. <**Learning objective:** Takotsubo syndrome triggered by acute myocardial infarction rarely occurs. Patients with a broad infarct-area require careful management because of the development of adverse events such as cardiogenic shock.>

Introduction

Takotsubo syndrome (TTS) is a reversible heart failure syndrome mimicking acute myocardial infarction (AMI) that occurs in the absence of obstructive coronary artery disease [1]. Some patients with concomitant TTS and AMI [2] have been reported. We herein describe a case of TTS that was triggered by physical stress probably due to coronary artery embolism (CAE) leading to AMI in a 73-year-old male patient with chronic nonvalvular atrial fibrillation (NVAF) in whom anticoagulant therapy had been discontinued.

Case report

A 73-year-old man was referred to our hospital with sudden-onset chest oppression, which occurred while eating ice cream at home in the evening. He had a history of hypertension and chronic NVAF with CHADS2 and CHA2DS2-VASc scores of 1 and 2. Anticoagulant therapy using dabigatran had been discontinued by his primary care doctor approximately three years previously. Upon arriving at our hospital, the patient exhibited chest oppression without any neurological abnormalities. A physical examination revealed that his blood pressure was 146/68 mmHg, his pulse rate was 72 beats/min and irregular, and his body temperature was 36.0 °C A 12-lead electrocardiogram (ECG) showed slight ST-segment elevation in the inferior lead (Fig. 1A), and echocardiography demonstrated diffuse wall motion abnormality of the left ventricle (LV) with an ejection fraction of 45% with grade 2 mitral regurgitation. Laboratory tests revealed that the patient's serum creatinine (0.8 mg/dl), creatinine kinase (CK, 86 U/L), CK-MB (9 U/L) levels, and plasma D-dimer level (0.7 μg/ml) were within the normal range, while his troponin T level increased to 0.128 ng/ml. Thus, he was diagnosed with AMI. The patient underwent urgent coronary angiography two hours after the onset of chest oppression, which revealed abrupt occlusion in the posterodescending branch of the right coronary artery, possibly caused by CAE (Fig. 2A, Video S1), with no atherosclerotic stenosis outside of the culprit lesion (Fig. 2B, C, Video S1). We did not perform percutaneous coronary intervention because we considered the infarct area to be small. Subsequently, a left ventriculogram showed loss of LV wall motion at the apex beyond the infarct-vessel perfusion area with hyperkinetic wall motion at the base, which seemed to represent apical-ballooning of the LV (Fig. 2D, E, Video S2). We suggested that TTS occurred following the onset of AMI, probably due to coronary thromboembolism from a left atrial thrombus in a patient with chronic NVAF who had not received anticoagulant therapy. On the second day, 12-lead ECG showed deep negative T-wave in the inferior and anteroposterior leads with T-wave inversion (Fig. 1B), as is typical for the course of TTS.

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https://doi.org/10.1016/j.jccase.2020.01.005

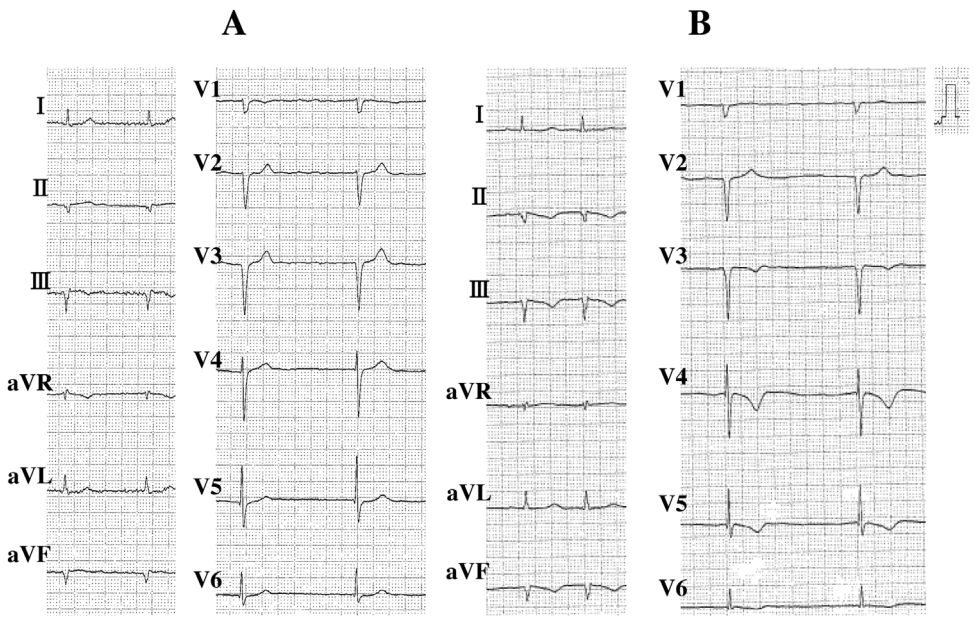

Fig. 1. Twelve-lead electrocardiogram on admission (A) and on the second day (B).

On the third day, the patient started to take a direct oral anticoagulant (apixaban 5 mg, twice a day) following the continuous intravenous administration of heparin. Subsequently, the patient's CK level rose to 239 U/L, and the CK-MB level rose to 44 U/L. On the fourth day, myocardial thallium-201 and iodine-123-beta-methyl iodophenyl pentadecanoic acid dual scintigraphy revealed mismatched uptake in the apical inferior area of the LV (Fig. 2F), which suggested an inferior AMI because it did not seem to represent a typical sign of TTS. On the 7th day, transesophageal echocardiography was performed to evaluate the intracardiac embolic source, which revealed spontaneous echo contrast (SEC) in the left atrium (LA) with the absence of thrombus in the left appendage, patent foramen ovale, and vegetation (Fig. 3, Video S3). The patient was discharged on foot on the 12th day; at the time, the wall motion of the LV remained impaired on echocardiography. Approximately 2 months after the onset of TTS, the wall motion of the LV had mostly improved, with hypokinesis of the apical inferior of the LV (Video S4).

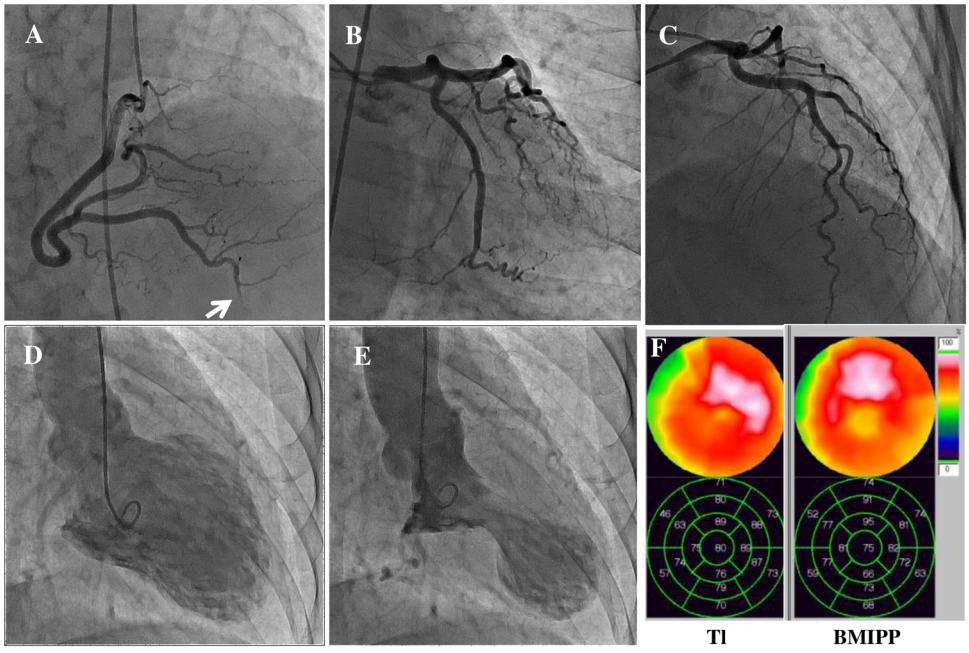

Fig. 2. Diagnostic coronary angiography showed abrupt occlusion (arrows) in the distal posterodescending artery of the right coronary artery and significant stenosis in the left coronary artery (A–C). Left ventriculogrphy revealed akinesis of the middle and apical segments with hyperkinesis of the base during diastole and systole (D, diastole and E, systole). Myocardial thallium-201 (Tl) and iodine-123-beta-methyl iodophenyl pentadecanoic acid (BMIPP) dual scintigraphy showed mismatched uptake in the apical inferior area of the left ventricle (F).

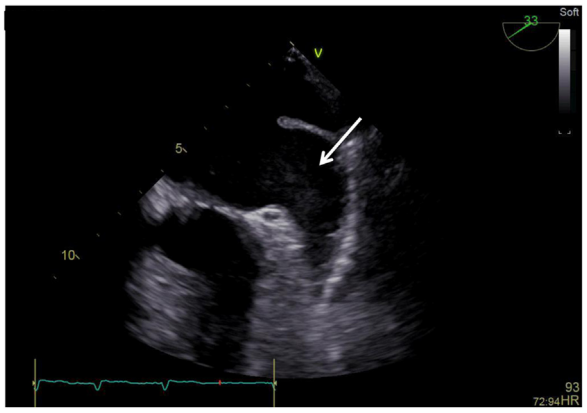

Fig. 3. Spontaneous echo contrast (arrow) with no evidence of thrombus in the left appendage on transesophageal echocardiography.

Discussion

TTS was first described by a Japanese cardiologist, Sato, in 1990 as a unique cardiomyopathy characterized by a reversible heart failure syndrome with symptoms and ECG changes mimicking AMI that is triggered by mental or physical stress in the absence of obstructive coronary artery disease [1]. According to the results of the International Takotsubo Registry (InterTAK Registry), however, approximately 15% of patients diagnosed with TTS had concomitant coronary artery disease [3]. Furthermore, the simultaneous occurrence of both TTS and AMI -as was observed in our patient- has been reported [2]. We assumed that TTS may have been triggered by the physical stress of AMI, probably due to CAE, in this case. To address this conclusion, two important issues should be solved: one is the etiology of AMI, the other is the causations between TTS and AMI.

CAE leading to AMI occurs infrequently because of unique anatomical features of the coronary arteries. The sudden onset of chest oppression while relaxing and eating ice cream at home suggested the development of systemic embolism in this case. Shibata et al. proposed the clinical diagnostic criteria and reported the clinical characteristics of CAE, in which 2.9% of patients developed an AMI due to CAE. These patients had few atherosclerotic risk factors, and the condition was most frequently caused by atrial fibrillation with low CHADS2 and CHA2DS2-VASc scores [4]. Our patient had the two minor criteria, and thus met the definition of "probable CAE", and his profile corresponded to these clinical features; thus, the etiology of AMI may have been CAE. In this case, the entity and source of the embolic materials was not confirmed. Furthermore, the patient was not evaluated for congenital and acquired coagulopathy. We hypothesize that it was thrombus from the LA because SEC associated with ischemic stroke in patients with NVAF [5] was observed in the LA with the absence of thrombus in the left atrial appendage. On the other hand, as Herath et al. reported that 5.3% of patients with TTS had complicating LV apical thrombus formation, and that in most of these cases LV apical thrombus formation occurred 24 h after the onset of TTS [6]. Thus, in the present case, it is unlikely that the AMI occurred due to apical thrombus of the LV complicated with TTS.

An increase in the catecholamine concentration triggered by mental and/or physical stress plays an important role in the occurrence of TTS [1]. According to the results of the InterTAK Registry, men were more triggered by physical stress and women by mental one, while approximately 28% of patients diagnosed with TTS did not have any evident triggers [3]. Kosuge et al. proposed a simple method using 12-lead ECG to differentiate between TTS and AMI: a significantly increased rate of ST-segment elevation in the -aVR lead with the absence of ST-segment elevation in the V1 lead in the acute phase of TTS [7]. Based on this, inferior AMI is considered to have occurred in this patient. Based on the diagnostic algorithm of TTS proposed by the international committee [8], TTS occurred in this patient. Thus, it is reasonable to suggest that the development of TTS was caused by physical stress probably due to CAE, although neither plasma catecholamine concentration nor autonomic nervous function was evaluated.

Patients with TTS usually have a good prognosis. Although the present case had a better outcome, it suggests the need for care in the management of some cases with a broad infarct area because there may be worse outcomes, including cardiogenic shock or heart failure.

Conflict of interest

The authors declare no conflict of interest in association with the present study.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.jccase.2020.01.005.

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