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

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A B S T R A C T

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.

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 posterodorsal branch of the right coronary artery, possibly caused by CAE (Fig. 2A, Vdeo 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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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. 1. Twelve-lead electrocardiogram on admission (A) and on the second day (B).

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 ventriculography 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 (TI) and iodine-123-beta-methyl iodophenyl pentadecanoic acid (BMIPP) dual scintigraphy showed mismatched uptake in the apical inferior area of the left ventricle (F).
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.

References

[1] Komamura K, Fukui M, Iwasaki T, Hirotani S, Masuyama T. Takotsubo cardiomyopathy: pathophysiology, diagnosis and treatment. World J Cardiol 2014;6:602–9.
[2] Y-Hassan S, Henareh L. Spontaneous coronary artery dissection triggered post ischemic myocardial stunning and takotsubo syndrome: two different names for the same condition. Cardiovasc Revasc Med 2013;14:109–12.
[3] Templin C, Gadhir JR, Diekmann J, Napp LC, Bataiosu DR, Jaguszewski M, et al. Clinical features and outcomes of takotsubo (stress) cardiomyopathy. N Engl J Med 2015;373:929–38.
[4] Shibata T, Kawakami S, Noguchi T, Tanaka T, Asaumi Y, Kanaya T, et al. Prevalence, clinical features, and prognosis of acute myocardial infarction attributable to coronary artery embolism. Circulation 2015;132:241–50.
[5] Hwang JJ, Ko FN, Li YH, Ma HM, Wu GJ, Chang H, et al. Clinical implications and factors related to left atrial spontaneous echo contrast in chronic nonvalvular atrial fibrillation. Cardiology 1994;85:69–75.
[6] Herath HMNTB, Pahalagamage SP, Lindsay LC, Vinothan S, Withanawasam S, Senaratne V, et al. Takotsubo cardiomyopathy complicated with apical thrombus formation on first day of the illness: a case report and literature review. BMC Cardiovasc Disord 2017;17:176.
[7] Kosuge M, Ebina T, Hibi K, Motita S, Okuda J, Iwashita N, et al. Simple and accurate electrocardiographic criteria to differentiate takotsubo cardiomyopathy from anterior acute myocardial infarction. J Am Coll Cardiol 2010;55:2514–6.
[8] Gadhir JR, Wittstein IS, Prasad A, Sharkey S, Dote K, Akashi YJ, et al. International expert consensus document on takotsubo syndrome (part): diagnostic workup, outcome, and management. Eur Heart J 2018;39:2047–62.