Journal of Cardiology Cases 18 (2018) 60–64

Contents lists available at ScienceDirect

Journal of Cardiology Cases

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

Case Report

Dynamic left ventricular outflow tract obstruction complicated with takotsubo cardiomyopathy: The acute phase of takotsubo cardiomyopathy manifests latent left ventricular outflow tract obstruction

Yuta Sakaguchi (MD)*, Kazuyuki Ozaki (MD), Toshiki Takano (MD), Hitomi Namba (MD), Komei Tanaka (MD), Takao Yanagawa (MD), Takuya Ozawa (MD), Tohru Minamino (MD, FJCC)

Department of Cardiovascular Biology and Medicine, Niigata University Graduate School of Medical and Dental Sciences, Niigata, Japan

ARTICLE INFO

Article history:
Received 22 December 2017
Received in revised form 12 April 2018
Accepted 24 April 2018

Keywords:
Dobutamine
Dynamic left ventricular outflow tract obstruction
Sigmoid-shaped septum

ABSTRACT

Dynamic left ventricular (LV) outflow tract (LVOT) obstruction is sometimes complicated with takotsubo cardiomyopathy (TC). The present case involves a 70-year-old woman with chest discomfort. Seven years earlier, transthoracic echocardiography revealed LVOT obstruction due to a sigmoid-shaped septum. She underwent urgent cardiac catheterization for suspected acute coronary syndrome. She was diagnosed as having TC with LVOT obstruction. After undergoing conservative treatment, her LV function normalized and the LVOT obstruction resolved. After the LV wall motion normalized, administering an intravenous infusion of dobutamine again provoked LVOT obstruction. In this situation, the presence of TC manifested latent LVOT obstruction.

<**Learning objective:** Although dynamic left ventricular outflow tract (LVOT) obstruction is the important compication of takotsubo cardiomyopaty (TC), the mechanism of LVOT obstruction remains unclear. This case had latent LVOT obstruction due to sigmoid-shaped septum, and LVOT obstruction might be manifested in the acute phase of TC. This phenomenon has potential for mechanism of LVOT obstruction complicated with TC.>

Introduction

Takotsubo cardiomyopathy (TC), also called stress-induced cardiomyopathy, is characterized by transient systolic left ventricular (LV) dysfunction and by abnormal electrocardiography (ECG) findings that mimic acute coronary syndrome in the absence of obstructive coronary artery disease [1]. Some patients with TC develop cardiogenic shock due to severe systolic dysfunction or LV outflow tract (LVOT) obstruction [2,3]. The mechanism of dynamic LVOT obstruction complicated with TC remains unknown; however, we hypothesize that the acute phase of TC manifests latent LVOT obstruction [4]. In this paper, we report a true case of LVOT obstruction due to the acute phase of TC.

Case report

A 70-year-old woman was admitted to our hospital because of chest discomfort. Seven years earlier, she had undergone transthoracic echocardiography (TTE) to examine a heart murmur. At that time, the TTE examination revealed a sigmoid-shaped septum with LVOT obstruction (e.g. the LVOT peak Doppler velocity was 4.7 m/s). This time, she had experienced diarrhea and anorexia for 1 week before her admission to our hospital. Her blood pressure was 79/49 mmHg and her heart rate was regular at 92 beats/min. The chest examination revealed a Levine II/VI ejection systolic murmur at the apex. Electrocardiography (ECG) revealed sinus tachycardia and ST elevation in leads $V_{4–6}$, II, III, and aV_F (Fig. 1A). Chest X-ray imaging revealed a cardiothoracic ratio of 49%, but no evidence of pulmonary congestion or pleural effusion. The TTE findings showed that the basal interventricular septum bulged into the basal LVOT, which suggested a sigmoid-shaped septum (Fig. 1B). LV hypertrophy (LVH) was not detected. LV asynergy with mid-apex anterior, anteroseptal,

* Corresponding author at: Department of Cardiovascular Biology and Medicine, Niigata University Graduate School of Medical and Dental Sciences, 1-757 Asahimatidori, Chuo-ku, Niigata, 951-8510, Japan.
E-mail address: yu-sakaguchi372@tatikawa.or.jp (Y. Sakaguchi).

https://doi.org/10.1016/j.jccase.2018.04.010
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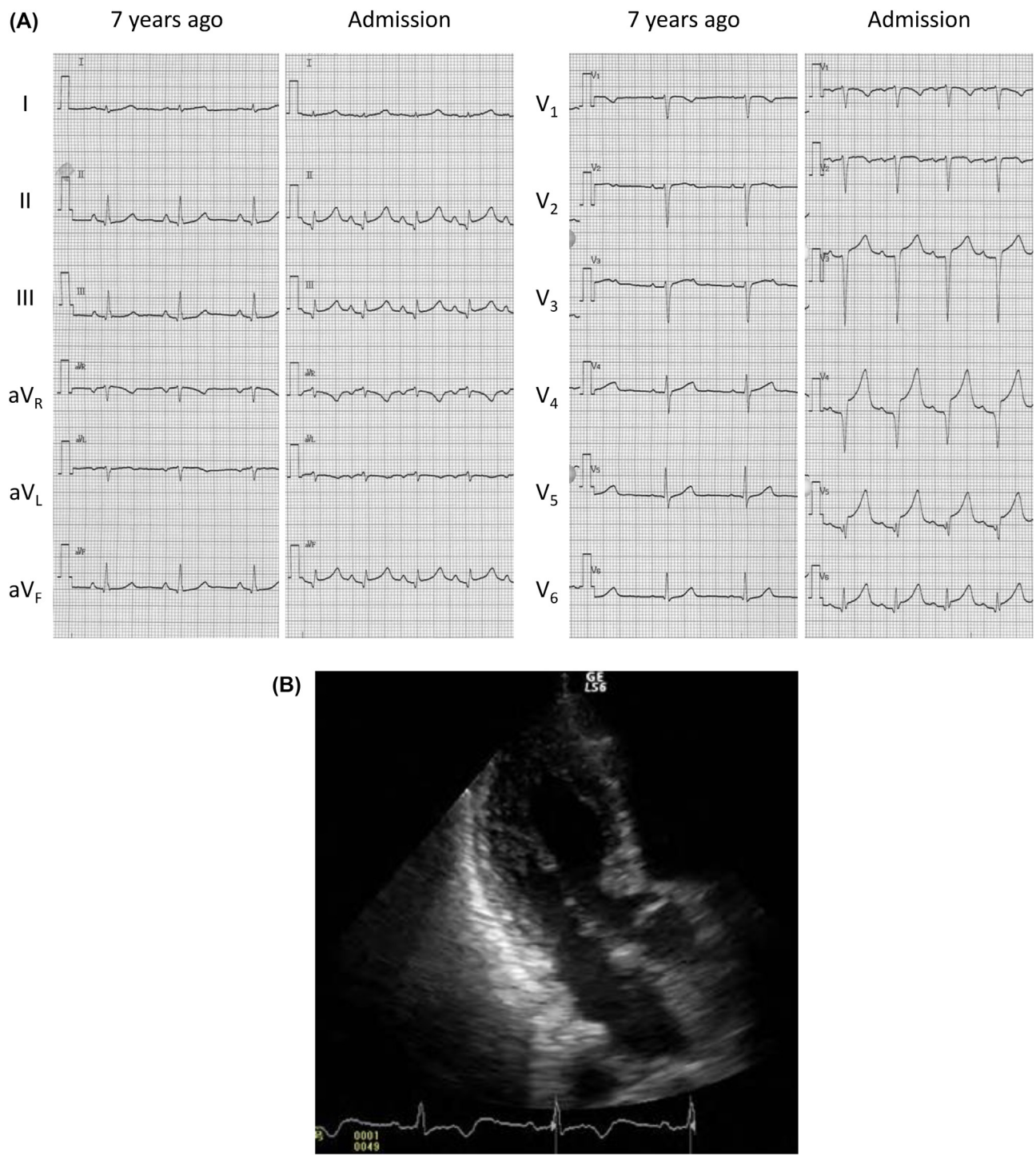

Fig. 1. The electrocardiography and the transthoracic echocardiography findings on admission. (A) The electrocardiography findings on admission (left) and 7 years earlier (right). On admission, electrocardiography shows ST elevation in leads II, III, aVF, and V_{4-6}. (B) The transthoracic echocardiography findings on admission. The apex long-axis view in echocardiography shows a sigmoid-shaped septum. The chamber size and wall thickness are within normal limits with no evidence of left ventricular (LV) hypertrophy. Mid-apex anterior, anteroseptal, and inferior LV wall motion shows severe hypokinesis. The LV ejection fraction is 45%. Mitral regurgitation is mild.

and inferior severe hypokinetic wall motion was also detected. The pressure gradient on admission was 60 mmHg with continuous wave pressure. There were no morphological abnormalities with papillary muscle and mitral valves. Blood examination exhibited an elevated white blood cell count of 8630/μL, creatine kinase level of 520 IU/L, creatine kinase-muscle brain form level of 72 IU/L, and brain natriuretic peptide level of 93.5 pg/mL. She underwent urgent coronary angiography for suspected acute coronary syndrome, and we confirmed the absence of an occlusive coronary artery (Fig. 2A). Left ventriculography revealed anterobasal and posterobasal segment hyperkinesis and apical akinesis (Fig. 2B). The ejection fraction was 42% and mitral regurgitation was mild. There was a 66 mmHg peak-to-peak systolic pressure gradient in the LVOT on catheter pullback. Based on these findings, she was diagnosed as having TC

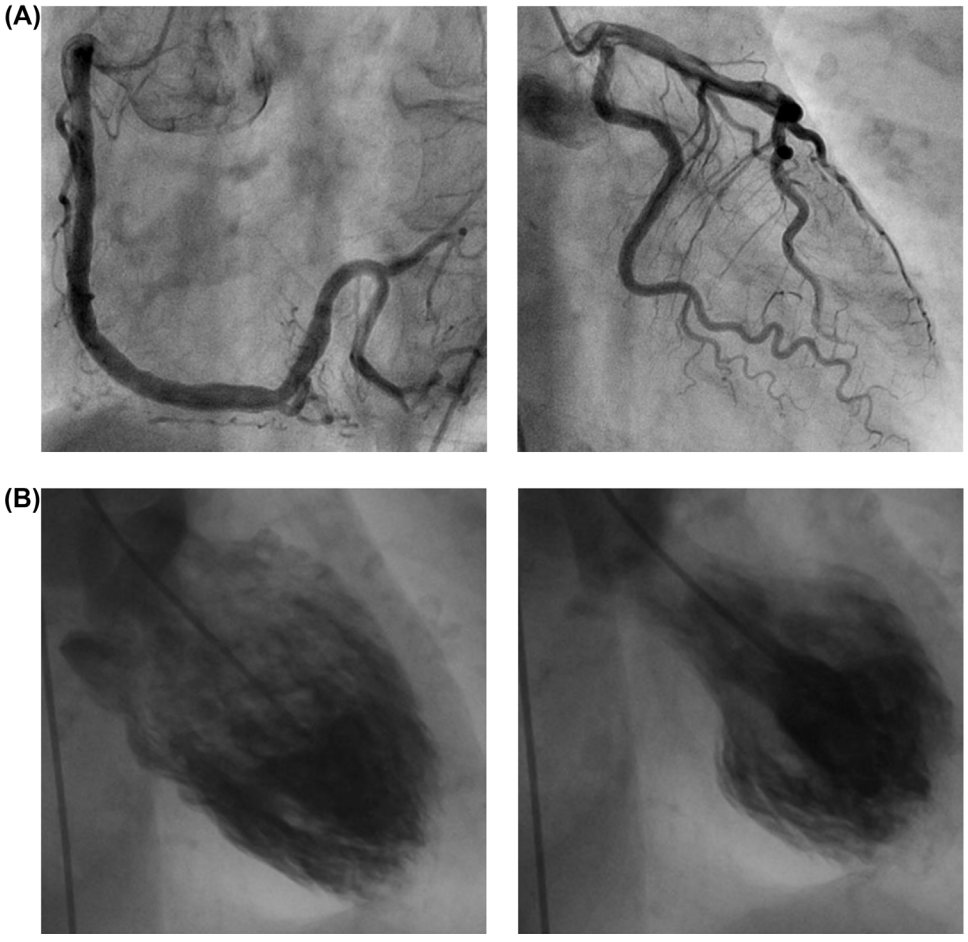

Fig. 2. The urgent coronary angiography. (A) The findings of urgent coronary angiography for the left coronary arteryin the 30° RAO/30° cranial view (right), and the right coronary artery in the 30° LAO/30° cranial view (left). Coronary angiography reveals the absence of an occlusive coronary artery. (B) The findings of left ventriculographyin the RAO view. The left ventriculography findings in diastole (left) and systole (right) show apical akinesia with anterobasal and posterobasal hyperkinesis. LAO, left anterior oblique; RAO, right anterior oblique.

with LVOT obstruction. With conservative treatment, her LV wall motion was normalized and LVOT obstruction was absent on day 10.

On day 15, she underwent cardiac catheterization again. Left ventriculography revealed normal LV wall motion (Fig. 3A). The spasm provocation test was positive for the right coronary artery when using 50 μg of acetylcholine (Fig. 3B), and was negative for the left coronary artery when using 100 μg of acetylcholine and 50 μg of ergonovine. There was no pressure gradient between the apex and ascending aorta. After receiving an intravenous infusion of dobutamine, a 100 mmHg peak-to-peak systolic pressure gradient was provoked in the LVOT (Fig. 3C). She was diagnosed as having vasospastic angina and latent LVOT obstruction.

She was treated with benizipine 8 mg/day and bisoprolol 2.5 mg/day. Neither an anginal attack nor worsened LVOT pressure gradient occurred after the administration of benizipine and bisoprolol. Later that day, the treadmill exercise test demonstrated she had sufficient exercise tolerance without provoked LVOT obstruction. She remained symptom-free after discharge.

Discussion

TC mimics acute coronary syndrome, and presents as transient LV dysfunction and ST-T changes on ECG [1]. This condition predominantly affects postmenopausal women. Emotional/physical stress at the onset is common, although a triggering event is not always present. There are some hypotheses for TC mechanism such as multivessel epicardial vasospasm [5] and coronary microvascular dysfunction catecholamine-triggered myocyte injury [6]. However, its pathogenesis is not well understood. The patient in this report had no emotional stress but had experienced gastroenteritis for weeks. We believed that physical stress was involved in the development of TC. However, the spasm provocation test was positive at the right distal coronary artery, whereas blood perfusion of the distal right coronary artery in the cardiac muscle involved a small region. For these reasons, coronary spastic angina was not a direct cause of TC in this patient.

Patients with TC often present with LVOT obstruction, and its prevalence ranges from 10% to 50% in previous reports [3]. The typical patient is an elderly, hypertensive female with sigmoid deformity of the intraventricular septum. The recurrence rate is higher among patients with TC and LVOT obstruction than in patients with TC without LVOT obstruction. In one report [3], patients who took a β-blocker did not have LVOT obstruction at the time of recurrent TC. A LVOT obstruction can be induced by stressors such as exercise or dobutamine infusion in patients with latent LVOT obstruction who do not exihibit LVOT obstruction [7]. After the LV wall motion had normalized in this patient, the dobutamine provocation test revealed LVOT obstruction. These findings indicated this patient had latent LVOT obstruction. In this patient, latent LVOT obstruction without LVH was primarily associated with a sigmoid-shaped septum. A sigmoid-shaped septum may be a normal part of the aging process and does not

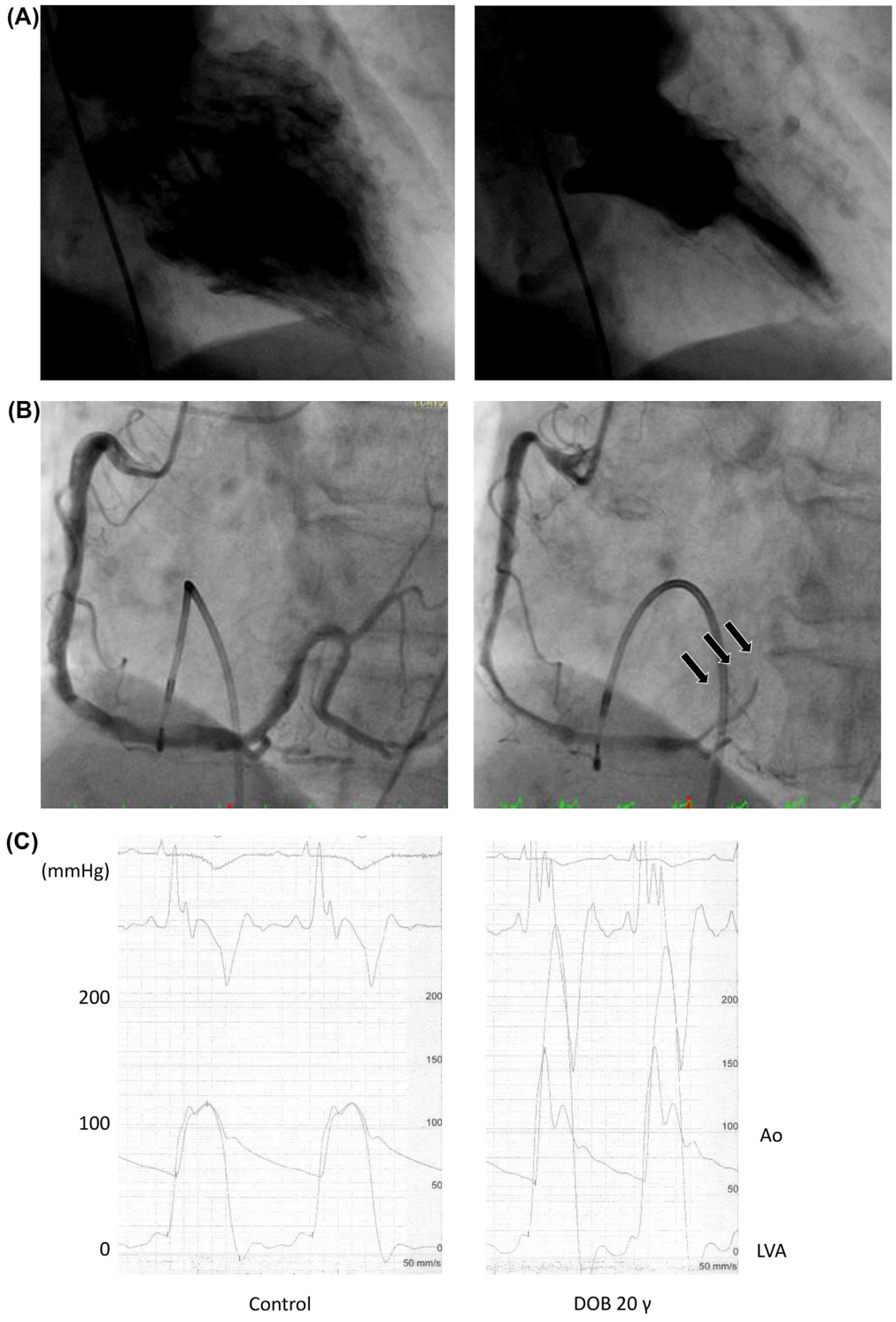

Fig. 3. The cardiac catheterization after the LV wall motion normalization. (A) The findings of left ventriculography in the RAO view on day 15. Left ventriculography in diastole (left) and systole (right) reveals normal left ventricular wall motion. (B) The findings of right coronary angiography in the 60° LAO view before (right), and after (left) 50 μg of acetylcholine infusion in the spasm provocation test. The spasm provocation test is positive in the distal right coronary angiography. (C) The findings of the PG between the LV apex and ascending aorta before (left) and after (right) dobutamine infusion. Dobutamine infusion provokes a 100-mmHg PG between the LV apex and the ascending aorta, which was not present before the infusion.
LV, left ventricular; LAO, left anterior oblique; RAO, right anterior oblique; PG, pressure gradient.

necessarily have any clinical importance, although several reports [8] have demonstrated the clinical importance of sigmoid-shaped septum as a potential cause of LVOT obstruction. Papillary muscle displacement of mitral valve is also an important mechanism of systoric anterior motion of the mitral valve [9]. In this case, morphologic abonormalities of papillary muscle and mitral valves were not evident.

The TTE performed 7 years earlier in this patient revealed a sigmoid-shaped septum with LVOT obstruction. This time, LVOT obstruction was again diagnosed with TTE and cardiac catheterization

in the acute phase of TC. Based on these findings, the mechanisms of LVOT obstruction in the acute phase of TC was a manifestation of latent LVOT obstruction caused by TC in this patient. The LV wall motion in the acute phase of TC is characterized by akinesis in the apical and mid-portions of the LV chamber that extends beyond one coronary artery region with hyperkinesis in the basal portion. The basal hyperkinesis narrows the systolic LVOT and accelerates blood flow through the LVOT, thereby promoting the Venturi effect and systolic anterior movement of the mitral valve and increasing the LVOT obstruction. Because of similar effects, anteroseptal acute myocardial infarction manifests as transient LVOT obstruction [10]. However, there are few reports of patients with previous LVOT obstruction and recurrent LVOT obstruction in the acute phase of TC. This finding is indicative of the manifestation of latent LVOT obstruction caused by TC. This case report revealed the clinical importance of latent LVOT obstruction due to a sigmoid-shaped septum.

Conflict of interest

The authors have no conflict of interest to declare.

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