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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

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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,

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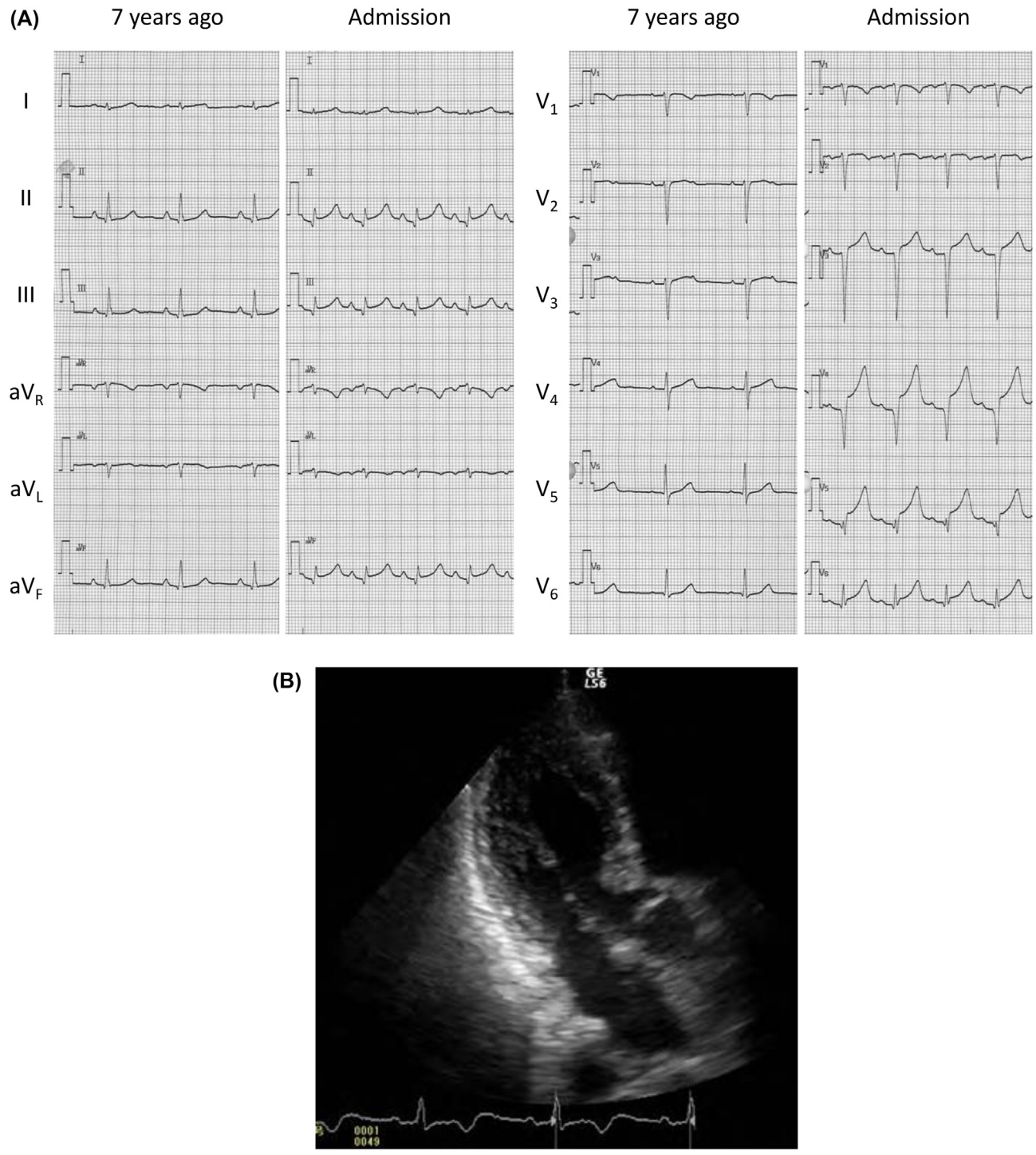

Fig. 1. The electrocardiography and the transthoracic echocardiography findings on admission. (A) The electrocardiography findings on admission (left) and 7 years earlier (right). On admission, electrocardiography shows ST elevation in leads II, III, aVF, and $V_{4–6}$. (B) The transthoracic echocardiography 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

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