

A CASE OF PERSISTENT APICAL BALLOONING COMPLICATED BY APICAL THROMBUS IN TAKOTSUBO CARDIOMYOPATHY OF SYSTEMIC LUPUS ERYTHEMATOSUS PATIENT

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Takotsubo cardiomyopathy, which is also known as “transient apical ballooning”, is a cardiac syndrome associated with emotional and physical stress that occurs in postmenopausal women. It may mimic acute coronary syndrome but coronary angiography reveals normal epicardial coronary arteries. The prognosis is favorable with the normalization of wall motion abnormalities within weeks. We report a case of persistent apical ballooning complicated by an apical thrombus in Takotsubo cardiomyopathy of systemic lupus erythematosus patient. Takotsubo cardiomyopathy may not be always transient and left ventricular thrombus can occur in the disease course as our patient.

KEY WORDS: Takotsubo cardiomyopathy · Systemic lupus erythematosus · Persistent apical ballooning · Thrombus.

INTRODUCTION

Takotsubo cardiomyopathy has clinical features that resemble an acute coronary syndrome, such as chest pain, ST-segment changes in the anterior precordial leads on electrocardiogram, mild elevation of serum cardiac enzymes, and transient left ventricular dysfunction with marked apical ballooning. The general prognosis is considered to be favorable, although some investigators have reported cases with various complications.¹⁾

This is a case of a 63-year-old woman with systemic lupus erythematosus (SLE) who suffered from persistent apical ballooning complicated by an apical thrombus in a suspected takotsubo cardiomyopathy. This case may be important because left ventricular thrombus may occur occasionally and not all takotsubo cardiomyopathy may recover completely.

CASE

A 63-year-old Korean woman with a past medical history of hypertension and a 25-year history of SLE presented with a 3-week history of shortness of breath. She had been treated with hydroxychloroquine 400 mg/day and varying doses of

prednisone between 5 and 15 mg/day for the SLE. Additionally, the hypertension was under control with carvedilol 25 mg/day. On examination, her blood pressure was 110/70 mmHg, pulse rate was 112 beats/min, respiratory rate was 24 breaths/min, and body temperature was 36.5°C. Jugular venous distention was noted on inspection. On cardiac auscultation, her rhythm was noted to be tachycardic but regular, weak summation gallops were heard at the cardiac apex, and no pericardial friction rubs were appreciated. Blood tests showed a white blood cell count of 4000/mm³ (normal, 4300-9400/mm³), hemoglobin of 11.5 g/dL (normal, 12-14.3 g/dL) and platelet count of 67000/mm³ (normal, 169-365/mm³). The C-reactive protein level was found to be 0.29 mg/L (normal, 0-0.75 mg/L). A blood chemistry panel revealed a blood urea nitrogen level of 25.0 mg/dL (normal, 7-20 mg/dL), creatinine of 1.3 mg/dL (normal, 0.5-1.5 mg/dL), total protein of 6.7 g/dL (normal, 6.0-8.3 gm/dL), and albumin of 3.2 g/dL (normal, 3.5-4.5 mg/dL). Analysis of the urinary sediment revealed 1-4 white blood cells/high power field (hpf), many red blood cells/hpf, and trace levels of proteinuria. Cardiac enzyme

• Received: January 5, 2013 • Revised: July 3, 2013 • Accepted: August 12, 2013

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labs were drawn and found to be elevated: CK-MB of 8.7 U/L (normal, 0.6-6.3 U/L), troponin-I of 0.35 ng/mL (normal, 0.0-0.2 ng/mL), and pro-brain natriuretic peptide of 8110 pg/mL (normal, 0-125 pg/mL). Chest X-ray revealed an enlarged cardiac silhouette and an electrocardiogram revealed ST elevation in leads V1-6 (Fig. 1A). Immunofluorescence tests were negative for double-stranded DNA antibodies and anti-extractable nuclear antigen antibodies (anti-Ro and anti-La). Complement levels were found to be low (C3 0.39 g/L, normal 0.8-1.7 g/L; C4 0.04 g/L, normal 0.12-0.36 g/L). Viral markers for cytomegalovirus, Coxsackie virus B type 2, herpes simplex virus, and Epstein-Barr virus were all negative. Echocardiography demonstrated moderate left ventricular systolic dysfunction

[left ventricular ejection fraction (LVEF) was 42%] with apical akinesia but no evidence of pericardial effusion (Fig. 2A and B). Echocardiography performed 2 years earlier showed mild concentric left ventricular hypertrophy with a LVEF of 70%. A coronary angiography showed normal coronary arteries.

We suspected takotsubo cardiomyopathy. However there was no trigger event as physical and emotional stress. The patient was treated with angiotensin converting enzyme inhibitor, furosemide, and intravenous nitrates. The dose of glucocorticoids was between 0.5 to 1 mg/kg for the control of SLE activity. Her dyspnea gradually improved, however, a three-week follow-up echocardiography test revealed persistent apical ballooning and a newly developed apical thrombus (size,

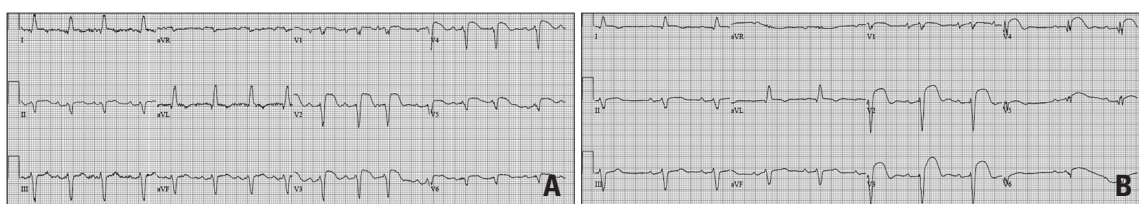


Fig. 1. Electrocardiography showing persistent ST segment elevation during the first admission (A) and 3 months follow-up (B).

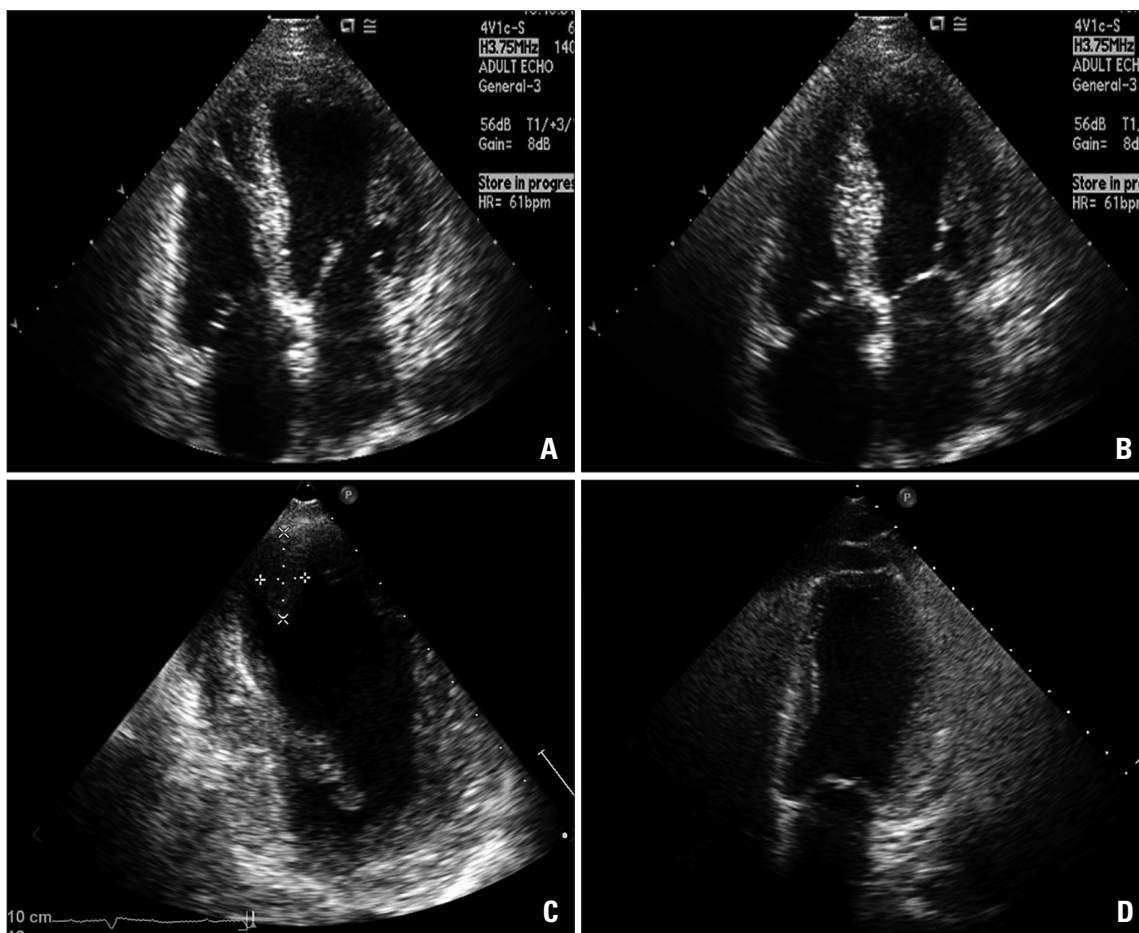


Fig. 2. Initial echocardiography showing apical ballooning at diastole (A) and at systole (B) of apical 4 chamber view. Follow-up echocardiography showing a newly developed thrombus in the left ventricular apex 3 weeks later (C). Akinesia of the left ventricular apex was persistent but slightly improved. Follow-up echocardiography 3 months later showing persistent apical ballooning with resolution of the thrombus (D).

1.10 × 2.12 cm) (Fig. 2C) with no significant improvement in LVEF. Heparin was then administered followed by oral anticoagulation therapy with warfarin. There were no embolic events during the patient's hospital stay. On the 35th day of hospital admission, follow-up echocardiography showed slightly improved wall motion of the left ventricular apex with a partially resolved thrombus and a LVEF of 50%. Although cardiac enzymes remained elevated (CK-MB 12.80 U/L and troponin-I 0.64 ng/mL), the patient was discharged on oral anticoagulation therapy.

Three months later, she was readmitted to the hospital due to a severe herpes zoster outbreak on her left shoulder. Cardiac enzymes were again found to be elevated (CK-MB 8.8 U/L and troponin-I 0.98 ng/mL). Electrocardiography revealed persistent ST segment elevation (Fig. 1B) and echocardiography revealed mild apical hypokinesia with a LVEF 50%, but no apical thrombus (Fig. 2D). During her hospital stay she developed a mild fever and candidemia which was treated with an intravenous antifungal agent. Unfortunately, she developed septic shock and expired on day 54 of hospital re-admission.

DISCUSSION

Takotsubo cardiomyopathy, which is also known as “transient apical ballooning”, is a cardiac syndrome associated with emotional and physical stress that occurs in postmenopausal women. It may mimic acute coronary syndrome but coronary angiography reveals normal epicardial coronary arteries.^{1,2)} In our case, the patient was a postmenopausal woman, however no triggering event was identified. A stressful trigger is often, but not always present. In up to 22% of patients, there was no identified triggering event.³⁾ The exact mechanism of takotsubo cardiomyopathy is not well known. Coronary vasospasm, disturbance of microcirculation, reperfusion injury and catecholamine overload are possible mechanisms.⁴⁾ In the acute phase, the treatment is generally supportive.²⁾ The prognosis is favorable with the normalization of wall motion abnormalities within weeks.⁵⁾ The complications of takotsubo cardiomyopathy are thought to be infrequent and different from those of the acute coronary syndrome, although there is inadequate literature evaluating the true incidence of these complications such as heart failure, cardiogenic shock, ventricular arrhythmias, ventricular rupture, and death.¹⁾

In our case, the finding of ST elevation and apical ballooning persisted over 3 months. The prolonged abnormal findings are rare in a typical case of takotsubo cardiomyopathy.^{1,4-9)} The mechanism of persistent ST elevation and apical ballooning is unclear. Some reports suggested that corticosteroid use might retard the improvement of left ventricular dysfunction.^{4,6)} However this is controversial, and a recent meta-analysis on corticosteroid use in myocardial infarction suggested that these

drugs had no harmful effects on clinical outcomes.¹⁰⁾ In this case, the steroid treatment was maintained with varying doses for the control of SLE activity.

Another interesting finding was the development of the apical mural thrombus. There are a few reports of thrombus associated with takotsubo cardiomyopathy.^{1,5,7,8)} It is thought that the thrombus may have been precipitated by the ventricular dyskinesia combined with an increased sympathetic activation which alters the coagulation cascade.⁵⁾ The clinical importance of this thrombus is that it may be a potential source of embolic events.⁷⁾ Echocardiography and cardiac magnetic resonance imaging may be useful techniques for the detection of an apical thrombus.¹⁾ Serial echocardiographic studies and anticoagulation therapy were useful for this complication, as were performed in this case.

This is a rare case of persistent apical ballooning complicated by an apical thrombus in takotsubo cardiomyopathy of SLE patient. Takotsubo cardiomyopathy may not be always transient and left ventricular thrombus can occur in the disease course as our patient. This is important for the treatment and management of patients with takotsubo cardiomyopathy.

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