Int J Clin Exp Med 2018;11(11):12723-12728
www.ijcem.com /ISSN:1940-5901/IJCEM0073883

Case Report

A case report of Takotsubo syndrome with ventricular thrombosis and changes in wall thickness

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Received May 30, 2017; Accepted June 1, 2018; Epub November 15, 2018; Published November 30, 2018

Abstract: Takotsubo syndrome has been described during the last 25 years. Echocardiography of this syndrome is characterized by akinetic myocardium in the apex. This study aimed to report the case of a 62-year-old woman with a ventricular thrombus and changes in ventricular wall motion and thickness. The patient suffered from headache, nausea, transient unconsciousness, and high fever. She was initially accepted as pituitary apoplexy caused by pituitary adenoma. However, LV apex thrombosis having wall motion abnormality was discovered. With a series of echocardiographic examinations, the thrombus disappeared without any symptoms. The left ventricular apex wall motion changed from akinetic to resembling hypertrophic cardiomyopathy, and then normalized during recovery accompanied by T-wave inversion and changes in prolonged QT interval. She was diagnosed with Takotsubo syndrome and identified by her normal coronary angiography CT scan. She was followed up for more than 6 years and had no headache or chest pain. In this Takotsubo syndrome case we found the wall thickness, changed from normal or thin in the first mimic myocardial infarction to resembling hypertrophic cardiomyopathy to normal, accompanied with ECG changes during recovery. The thickness changes of the LV wall required extensive investigation.

Keywords: Echocardiography, stress cardiomyopathy, Takotsubo syndrome

Introduction

Takotsubo syndrome is a cardiac syndrome characterized by the absence of obstructive coronary disease with changes in electrocardiography (ECG) which can mimic acute myocardial infarction (MI). Takotsubo syndrome patients may have transient left ventricular (LV) dysfunction, minimal release of myocardial enzymes, and complete recovery [1, 2]. The syndrome is commonly reported [3]. While changes of ECG had been described in detail [2, 4], the present case was of a stroke patient with series echocardiographic changes in an LV thrombus and the thickness and motion of LV apex was accompanied by changes in ECG.

Case report

A 62-year-old housewife suffered from intermittent left temporal headache, nausea, and vomiting zero to two times each year for 10 years without vision loss and visual field defects. All these symptoms were aggravated 3 months before she was admitted to Beijing Hospital for detailed examination in June 2010.

One month prior, the patient was admitted to the Emergency Department of the HuaBei Shiyou Hospital because of transient unconsciousness and high fever (39.0°C). Emergency computed tomography (CT) scan of the head did not show any abnormalities. The patient had apparent anorexia and fatigue. Blood pressure (BP) was 90/55 mmHg. The laboratory analysis showed that serum sodium was 106 mmol/L (normal range, 135-145 mmol/L). After fluid therapy and administration of inotropic agents (dopamine, atropine, aramine), BP remained 70/50 mmHg. Because of suspected acute adrenocortical hypofunction, the patient was treated with the subsequent infusion of 100 mg cortisol twice daily until she was admitted to the Beijing Hospital. BP recovered to 100/70 mmHg thereafter. Similarly, serum sodium increased to 133 mmol/L, but the patient started experiencing euphoria, sleeplessness, convulsions, and disorientation. She was trans-

Table 1. Laboratory tests related to hypopituitarism

Test	Test value	Reference value
Blood coagulation phase		
D-dimer	750 ng/mL↑	<500 ng/mL
Serum luteinizing hormone	0.92 mIU/mL↓	20-50 mIU/mL (menopause)
Follicle-stimulating hormone	2.23 mIU/mL↓	30-60 mIU/mL (menopause)
Prolactin	1.61 ng/mL↓	4.5-12.6 ng/mL (at 50 years)
Estradiol	1.99 pg/mL	<15 pg/mL (menopause)
Testosterone	0 nmol/L↓	1-1.5 nmol/L (for women)
Free triiodothyronine	1.02 pg/mL↓	2.3-4.2 pg/mL
Free thyroid hormone	0.44 ng/dL↓	0.89-1.76 ng/dL
Thyroid-stimulating hormone	0.48 μIU/mL	0.35-5.5 μIU/mL
Cortisol	<1 μg/dL (8 a.m.)	10-20 μg/dL (8 a.m.)
	<1 μg/dL (4 p.m.)	3-10 μg/dL (4 p.m.)
	<1 μg/dL (the day after 8 a.m.)	
Adrenocorticotropic hormone (ACTH)	<10 pg/mL (when 8 a.m. cortisol is <1 μg/dL)	0-46 pg/mL

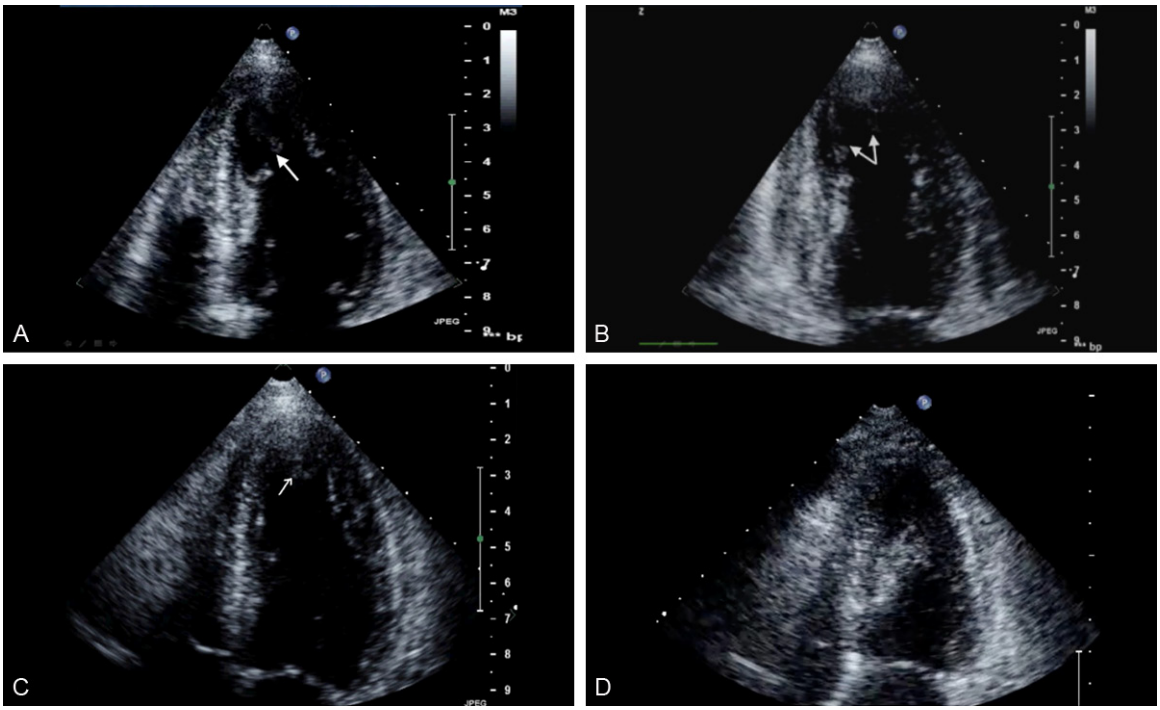

Figure 1. Echocardiography of the patient when the patient in the hospital. (A-C) were taken on the seventh day after admission. (A) Akinetic segmental wall motion of the left ventricular (LV) apex was seen in the systolic phase. (A and B) Two thromboses were adhering to the LV apex as the arrow showed. (C) Apex thrombus diminished obviously 4 hours after the first echocardiography (A and B). (D) On the 18th day after admission, echocardiography showed no LV thrombus, and the LV apex was thicker than any other part of the left ventricle.

ferred to the Department of Endocrinology, Beijing Hospital, for further treatment. She had no hypertension or a history of diabetes or coronary heart disease. She had never had chest pain. Her menarche occurred at the age of 14 years. She had experienced a spontaneous abortion with massive hemorrhage (hemoglobin dropped to about 40 g/L) when she was 45 years old and had menopause at 50 years of age.

During admission, the physical examination was normal except that the heart rate was 100 beats per minute. The laboratory tests indicated hypopituitarism (**Table 1**). Pituitary magnetic resonance imaging (MRI) revealed saddle ab-

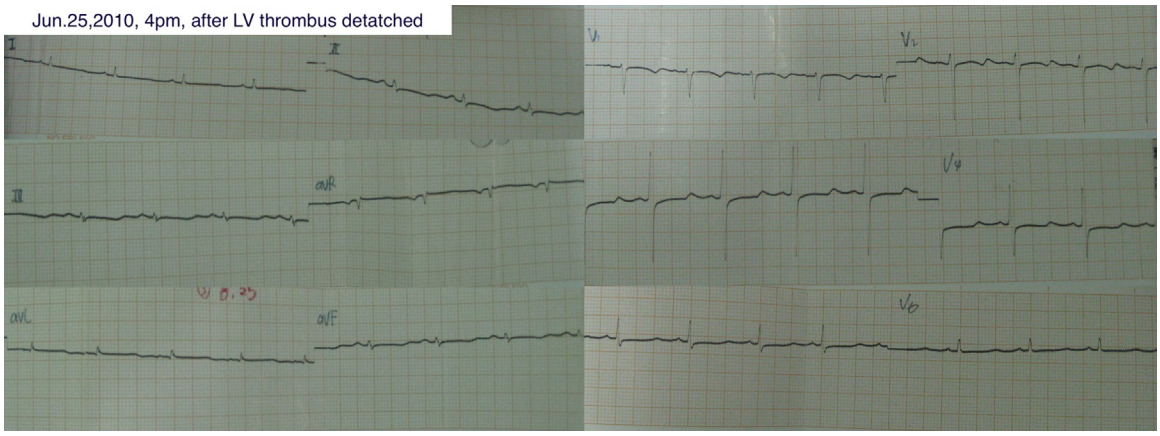

Figure 2. ECG on the seventh day after admission showed flat T waves in chest leads.

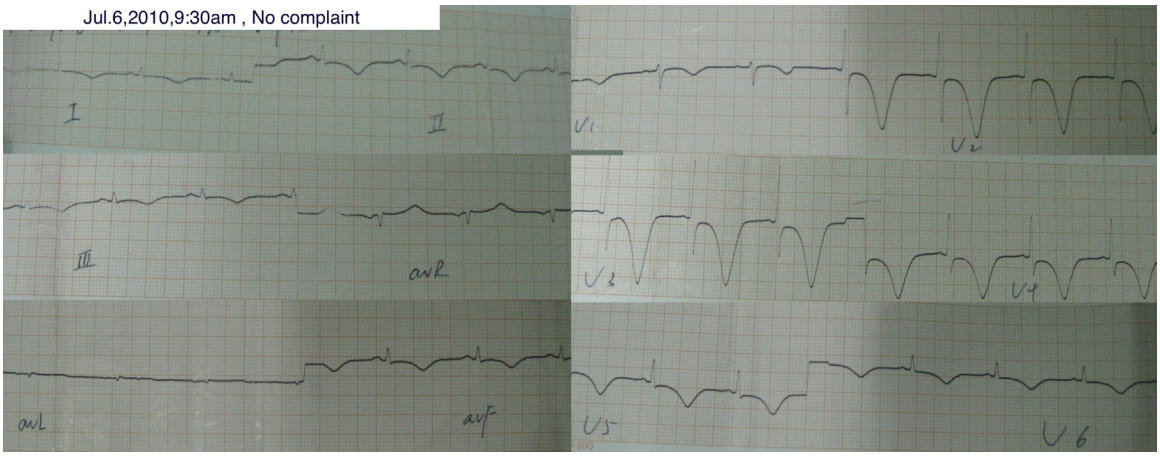

Figure 3. ECG on the 17th day after admission. A typical deep T-wave inversion and prolonged QT interval changes of precordial leads of ECG.

normal signals in shadow (about the size of 1.9 × 1.7 × 1.3 cm^3, borders, T1W1 obvious high signal on T1W1, and a slightly uneven high signal on T2W1).

Seven days after admission, the patient had routine echocardiography to exclude thromboembolism of cerebral artery. It revealed an apex thrombus with akinetic segmental wall motion in the LV apex (**Figure 1A** and **1B**), and ECG (**Figure 2**) showed no myocardial infraction. The second echocardiography on the same day, 4 hours after the first echocardiography, showed that the akinetic segmental wall motion in the LV apex was the same, but the thrombus had diminished in size (**Figure 1C**). An LV thrombus with acute MI was suspected. However, the patient had no chest pain or a headache after admission. Simultaneously, the troponin I level was elevated to 0.14 ng/mL (normal <0.04 ng/mL) on the 7th day after admission, which normalized on the 12th day after admission.

The patient was treated with Fragmin 5000 U every 12 hours after the second echocardiography.

On the 11th day after admission, the third echocardiogram showed no LV thrombus, and segmental wall motion abnormalities in the LV apex were almost recovered.

The ECG on the 18th day after admission showed a typical deep T-wave inversion and changes in prolonged QT interval in the precordial leads of ECG (**Figure 3**). The patient had no chest pain or thrombosis all these days. She refused to undergo a coronary angiography or CT scan at that time.

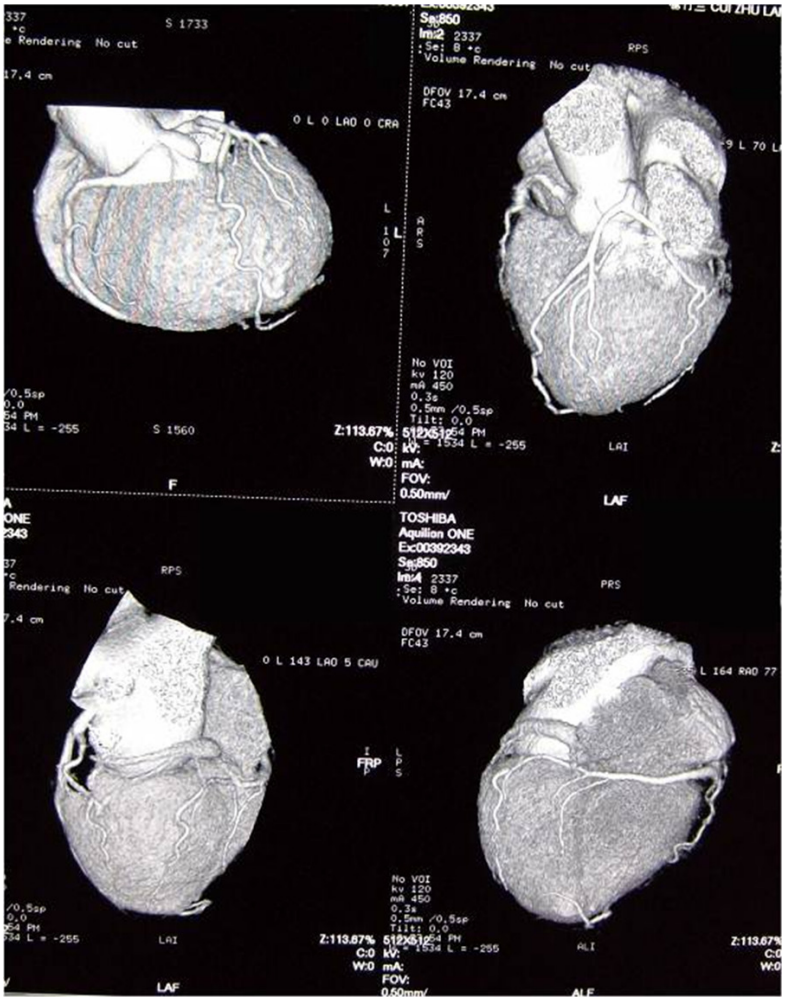

Figure 4. CT scan of the coronary artery five months after discharge showed that the coronary artery was completely normal.

On the 18th day after admission, the fourth echocardiogram showed no LV thrombus, and the LV apex was thicker than any other part of the left ventricle. The shape of the left ventricle was like that of the heart at the end of the systolic phase, resembling hypertrophic cardiomyopathy (**Figure 1D**).

The final diagnosis was pituitary apoplexy caused by pituitary adenoma and suspected Takotsubo cardiomyopathy with a transient apex thrombus. The patient refused surgical decompression of the pituitary and was then discharged with daily prednisone 5 mg/day, simvastatin 20 mg/day, Euthyrox 12.5 μg/day, and aspirin 100 mg/day on the 20th day after admission.

Five months after discharge, the patient had a coronary artery CT scan which showed her coronary artery was completely normal as show in 3D vision (**Figure 4**). Another echocardiogram showed the heart size and structure and the thickness of the LV wall had been normalized. She was followed up for more than 6 years and had no headache or chest pain. She has been taking the same medicine as prescribed since she was discharged from the hospital.

Discussion

This study reports a case of a 62-year-old female patient who suffered from pituitary apoplexy. She was accepted as a stroke patient accidently found with LV apex thrombosis and having wall motion abnormality. With a series of echocardiographic examinations, the thrombus disappeared and wall motion recovered. During the recovery period, the thickness of the LV apex was found to increase accompanied by T-wave inversion and changes in prolonged QT interval. She was diagnosed with Takotsubo syndrome when her coronary angiography and CT scan were normal.

Although Takotsubo syndrome predominantly affects postmenopausal women (~90% of all cases reported, particularly in larger cohorts), the aforementioned features are not a mandatory part of the diagnostic criteria. Takotsubo syndrome is reversible - a critical feature that helps to differentiate it from other acute heart syndromes. Timelines of recovery vary depending on the severity of the acute episode. The LV ejection fraction usually recovers by 12 weeks, but changes in ECG and brain natriuretic peptide levels may take 6-12 months to recover [2, 4]. Many cases have been diagnosed with stress cardiomyopathy in the last 25 years, but the mechanism is still not clear [3].

The patient had typical echocardiogram features, and the thickness of the LV wall changed

with changes in ECG. This meant that changes in the thickness of the LV apex might be observed in patients having typical changes in deep reverse T-wave inversion and prolonged QT intervals.

Studies on the clinical features and etiology of Takotsubo syndrome have shown that several possible factors including abnormal anatomy of the coronary artery, activation of cardiac adrenoceptors, disturbance of sympathetic innervation, impairment of fatty acid metabolism, low estrogen level, and multi-vessel coronary spasm may contribute to the attack, although the definite mechanism is still unclear [2].

During the acute phase of Takotsubo syndrome, cardiovascular MRI showed edema of LV myocardium as high signal intensity, with a diffuse or transmural distribution consistent with the wall motion abnormality. These features helped distinguish Takotsubo syndrome from myocarditis and acute MI. Typically, late enhancement is usually absent in Takotsubo syndrome, distinguishing it from MI [5]. Edema might be the reason for dynamic T-wave inversions in patients with reversible LV dysfunction [6]. The thickness of the LV wall may be caused by edema and the activation of cardiac adrenoceptors, it had been reported LV wall thickness in patients with Takotsubo syndrome maybe showed worse prognosis compared to those without LV wall thickness during recovery from Takotsubo syndrome [7].

Abnormalities on ECG are present in more than 95% of patients with Takotsubo syndrome during the acute phase [8, 9]. The QT interval is important in risk stratification and may help distinguish Takotsubo syndrome from ST-segment elevation MI [9]. If the presentation is delayed, T-wave inversion and QTc prolongation without ST-segment elevation might be observed. Kurisu [8] reported that the ECG of patients with Takotsubo syndrome usually showed ST-segment elevation, especially in precordial leads of ECG V3-V6, during admission. The T-wave inversion deepened progressively to its first negative peak, which occurred at approximately 3 days. The T wave was shallow for several days and then deepened again, the second negative peak occurring in approximately 2-3 weeks. The QT interval was prolonged as the T wave deepened, and shortened as the T wave became shallow. This pattern was also seen in the patient reported in the present study.

In conclusion, in this case of Takotsubo, we found that the apex segmental wall motion changed from akinetic to normal. Additionally, this case showed wall thickness that changed from normal or thin like myocardial infarction to resembling hypertrophic myocardiography then recovery to normal, accompany with changes of ECG and LV thrombosis. The thickness changes of the LV wall may have been caused by edema and the activation of cardiac adrenoceptors, which requires extensive investigation.

Acknowledgements

Written informed consent was obtained from the patient and her relatives for publication of this report. All echocardiographic images and movies were obtained from the same echo lab.

Disclosure of conflict of interest

None.

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