case report

Takotsubo cardiomyopathy resembling acute high lateral myocardial infarction

Erkan Ilhan, Ufuk Gürkan, Hale Yaka Yılmaz, Tolga Sinan Güvenç, Yigit Çanga, Osman Bolca

From the Department of Cardiology, Dr. Siyami Ersek Cardiovascular and Thoracic Surgery Training and Research Hospital, Istanbul, Turkey

Correspondence: Erkan Ilhan · Department of Cardiology, Dr. Siyami Ersek Cardiovascular and Thoracic Surgery Training and Research Hospital, Tibbiye caddesi, Haydarpasa, 34668, Istanbul, Turkey · M: +905054365384, F: +902164183317 · erkan.ilhan@yahoo.com.tr

Ann Saudi Med 2012; 32(4): 424-426

DOI: 10.5144/0256-4947.2012.424

A 65-year-old female patient admitted to the emergency department was diagnosed with acute high lateral myocardial infarction, but later Takotsubo cardiomyopathy (TC) was discovered. She had squeezing chest pain that started shortly after an emotional stress. The electrocardiogram revealed a loss of R wave voltage in leads V1 to V4 and an ST-segment elevation in I and aVL. After an urgent coronary angiography and ventriculography, TC was considered, and supportive anti-ischemic treatment was started. The severe left ventricular systolic dysfunction improved and normalized during the follow-up. She was discharged without any complications. TC is a new entity of acute cardiac events, and patients usually recover completely without sequelae with proper diagnosis and management. An exact diagnosis may also prevent an inappropriate application in the setting of recurrences.

Takotsubo cardiomyopathy (TC), also known as transient left ventricular apical ballooning syndrome, is one of the acute cardiac events that resemble acute coronary syndromes. The characteristics of this syndrome are electrocardiographic ST-T changes mimicking acute coronary syndromes, reversible wall motion abnormalities that cannot be explained by a single major coronary artery occlusion, and increased cardiac enzymes, but absence of obstructive or thrombotic lesions on coronary angiography. We present a TC case mimicking acute high lateral myocardial infarction (MI).

CASE

A 65-year-old female patient without any medical history was admitted to our emergency department with a complaint of mild squeezing chest pain lasting 1 hour. Her chest pain started soon after an emotional stress, when she learned that her mother had died. The heart rate and blood pressure were 79 bpm and 130/80 mm Hg, respectively. The physical examination was normal. The electrocardiogram (ECG) revealed a loss of R wave voltage in leads V1 to V4 and a 0.5 mV ST-segment elevation in I and aVL, without any reciprocal changes (**Figure 1a**). The corrected QT (QTc) interval was 464 ms. An urgent coronary angiography showed only a nonobstructive proximal left anterior descending coronary artery plaque without any coronary spasm, but the left ventriculography revealed characteristic apical ballooning due to hyperkinesis of basal segments and akinesis of other segments (**Figure 2**). The left ventricular end diastolic pressure was 10 mm Hg.

At bedside, the transthoracic echocardiography (TTE) in the intensive care unit showed a severely depressed left ventricular systolic function with an ejection fraction (EF) of approximately 20% to 25%. The mid and apical segments of all left ventricular walls were akinetic, while basal segments were hyperkinetic (**Figure 3**). The peak troponin I level was found as 4.97 ng/dL (reference 0-0.06 ng/dL) during hospitalization. Fasting glucose, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, very low-density lipoprotein cholesterol, and triglyceride levels were 98 mg/dL, 102 mg/dL, 50 mg/dL, 18 mg/dL, and 90 mg/dL, respectively. She had no history of cigarette smoking or any drug usage. The left ventricular systolic functions showed progression during serial TTE examinations under acetylsalicylic acid, clopidogrel, and metoprolol treatment without any inotropic support. She was discharged from the hospital with dual antiplatelet and beta-blocker medications at the end of the first week, with a possible diagnosis of TC. During serial visits, the TTE showed a progressive improvement and normalization

of wall motion 2 weeks after the index event (**Figure 3**). The EF was calculated as 60%-65%. The ECG at that time showed a normal R wave progression in precordial leads, inverted T waves in leads I, aVL, and V1to V6, and a positive T wave in aVR (**Figure 1b**). The QTc was calculated as 436 ms at that time.Thus, she was diagnosed with TC. The patient did not experience health problems over a 3-month follow-up period.

DISCUSSION

TC was first described in the Japanese population in 1990, and it is seen far more commonly in Japan.[1] However, folllowing increased awareness of the syndrome more patients with TC were reported in Europe and North America. The mean age was reported as 67 years in one of the largest series.[2] According to that study, 63% of the patients had physiologic or emotional stress and 86% of cases were women. Chest pain was the most common presenting symptom (67%), but isolated dyspnea or syncope was also reported in the published studies.[3] The ST-segment elevation is seen frequently on admission (90%), especially in precordial leads, yet the isolated ST elevation in inferior or lateral leads is seen rarely.[2] In the same study, which had a relatively large patient number, Tsuchihashi et al found that creatinine kinase was elevated in 56% of cases,[2] but some of later series using troponin as a cardiac marker showed elevation of that cardiac biomarker in virtually all patients.[4] The peak level of troponin in TC patients is generally disproportionately low given the extent of wall motion abnormality, and almost all patients typically develop T-wave inversions in most leads on the subsequent days.[2]

The transient wall motion abnormality (akinesis or dyskinesis) usually affects apical and mid portions of the left ventricle. However, some variant TC cases in the published studies propose that the apical wall motion may be spared in some patients.[5,6] It was shown that no ECG criteria can reliably discriminate between TC and ST-segment elevation MI.[7] Although no single noninvasive techniques are available for differentiation of these two entities, cardiac MRI may be helpful. Some studies showed that in contrast to the typical findings in acute MI or myocarditis, TC patients do not show any delayed gadolinium hyperenhancement.[8,9] Still, an invasive coronary arteriography is usually needed in most patients in routine clinical practice.

The precise etiology and real frequency of the syndrome are not known. Since the syndrome commonly occurs immediately after an acute emotional and/or physical stress, catecholamine stimulation seems to be an important component of the pathophysiology of TC. Wittstein et al showed that patients with TC have

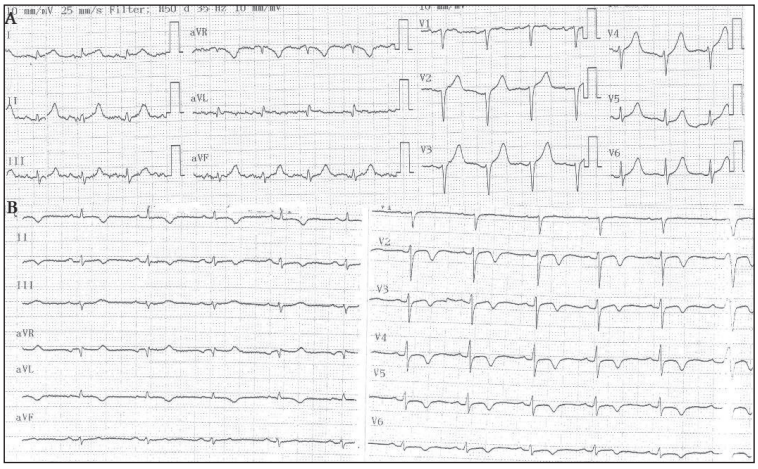

Figure 1. Twelve lead electrocardiogram (ECG) showing minimal 0.5 mm ST segment elevation in leads aVL, I and loss of R wave voltage in leads V1-V4 (A). Twelve lead ECG showing restoration of R wave voltages in precordial leads and typical negative T waves in most of the leads (B).

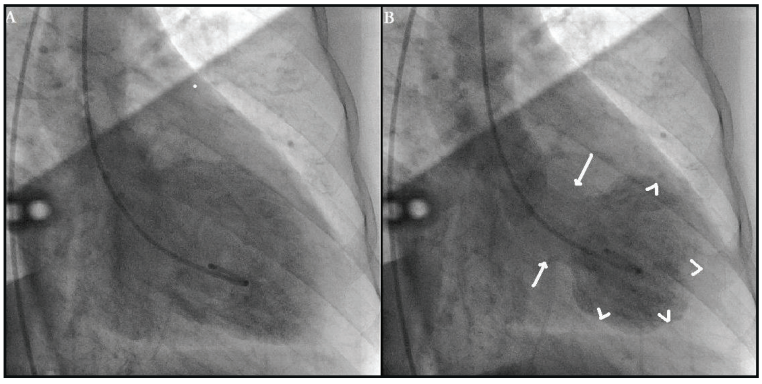

Figure 2. Left ventriculogram in the right anterior oblique projection during diastole (A) and systole (B) showing akinesis of the midventricular and apical segments (arrowheads) but hyperkinesis of basal segments (arrows) giving the shape of apical ballooning.

2- to 3-fold higher catecholamine levels compared to acute MI patients who have the same degree of heart failure.[10] Furthermore, the endomyocardial biopsy of TC patients showed mononuclear infiltrates and contraction-band necrosis, which are consistent with the catecholamine-mediated cardiotoxicity.[10]

Overall, the prognosis seems favorable. In the largest series including 88 patients, the mortality rate was reported to be 1%.[2] In the same study, heart failure or pulmonary edema were seen in 22% of patients, and 8% of cases needed intra-aortic balloon pump. Eight patients (9%) exhibited ventricular tachycardia or fibrillation. Only 1 patient (1%) had heart failure at discharge, and recurrences of TC were seen in 2 patients (2%) after discharge. However, some rare complications, such as left ventricular wall rupture and severe mitral regurgita-

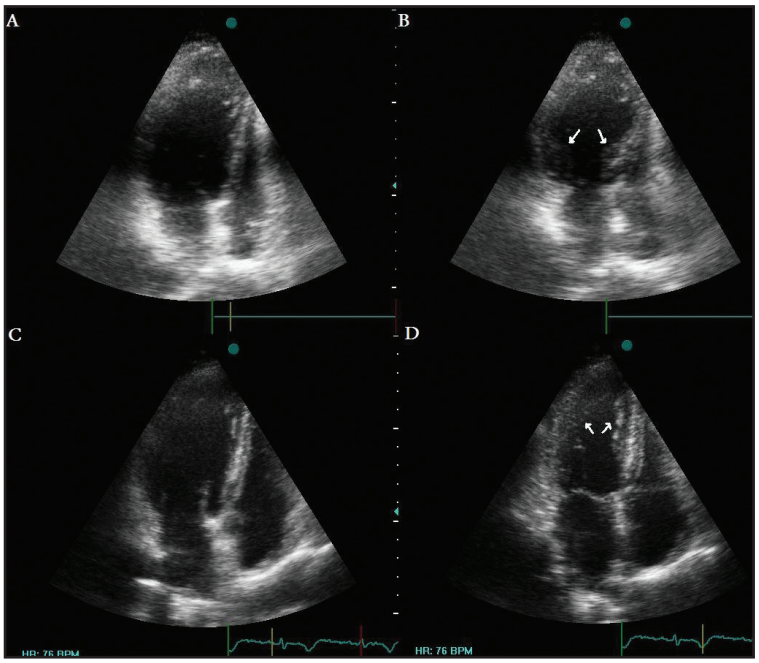

Figure 3. Diastolic (A) and systolic (B) frames of apical 4-chamber view of transthorasic echocardiography in intensive care unit showing akinetic mid and apical segments and hyperkinetic basal segments (arrows). After serial visits, apical 4-chamber view of control transthoracic echocardiography showing normal left ventricular wall motions during diastole (C) and systole (D). Arrows indicate normal thickening of previously abnormal segments

tion due to acute rupture of the posteromedial papillary muscle, were also reported in published studies.[11,12]

After prediagnosis, classical heart failure treatment including diuretics, inotropics, angiotensin-converting enzyme inhibitors, beta-blockers, and antiplatelet agents should be started and, if needed, left ventricular assist devices should be used.[13] While considering the inotropic support, clinicians should also be aware of the dynamic intraventricular obstruction due to hyperkinesis of basal segments, systolic anterior motion of mitral leaflets and chorda (SAM). This complication is seen in 18% of patients and may worsen after positive inotropic support by increasing the gradient or a worsening of possible mitral regurgitation caused by SAM.[2] The ventricular mural thrombus formation and thromboemboli are potential complications especially in those with the significant left ventricular systolic dysfunction.[14] Thus, short-term anticoagulation should be considered in most patients until the recovery of wall motions begins.[4]

In conclusion, TC is a recently defined acute cardiac event and should be suspected especially in individuals without an obvious heart disease. Despite its dramatic presentation, usually patients recover completely without sequelae with proper diagnosis and management. Since some recurrent TC cases were defined,[15] an exact diagnosis during the index event will also prevent inappropriate applications in the setting of recurrences.

REFERENCES

1. Satoh H, Tateishi H, Uchida T. Takotsubo-type cardiomyopathy due to multivessel spasm. In: Kodama K, Haze K, Hon M, editors. Clinical Aspect of Myocardial Injury: From Ischemia to Heart Failure (in Japanese). Tokyo: Kagakuhyouronsya Co.; 1990. p. 56-64.
2. Tsuchihashi K, Ueshima K, Uchida T, Oh-mura N, Kimura K, Owa M, et al. Transient left ventricular apical ballooning without coronary artery stenosis: A novel heart syndrome mimicking acute myocardial infarction. Angina Pectoris-Myocardial Infarction Investigations in Japan. J Am Coll Cardiol 2001;38:11-8.
3. Schneider B, Athanasiadis A, Schwab J, Pistner W, von Scheidt W, Gottwald U, et al. Clinical spectrum of tako-tsubo cardiomyopathy in Germany: Results of the tako-tsubo registry of the Arbeitsgemeinschaft Leitende Kardiologische Krankenhausärzte (ALKK). Dtsch Med Wochenschr 2010;135:1908-13.
4. Bybee KA, Prasad A, Barsness GW, Lerman A, Jaffe AS, Murphy JG, et al. Clinical characteristics and thrombolysis in myocardial infarction frame counts in women with transient left ventricular apical ballooning syndrome. Am J Cardiol 2004;94:343-6.
5. Cacciotti L, Camastra GS, Musarò S, Proietti I, Semeraro R, Martina C, et al. Stress cardiomyopathy: Transient basal ballooning. J Cardiovasc Med (Hagerstown) 2010;11:764-7.
6. Phanthawimol W, Ito H, Fukuyama O. A case with transient anterolateral wall ballooning syndrome: New variant form of Takotsubo cardiomyopathy? Hawaii Med J 2009;68:249-52.
7. Bybee KA, Motiei A, Syed I, Kara T, Lennon R, Murphy J, et al. Electrocardiography cannot reliably differentiate transient left ventricular apical ballooning syndrome from anterior ST-segment elevation myocardial infarction. J Electrocardiol 2007;40:38.e1-6.
8. Haghi D, Fluechter S, Suselbeck T, Kaden JJ, Borggrefe M, Papavassiliu T. Cardiovascular magnetic resonance findings in typical versus atypical forms of the acute apical ballooning syndrome (Takotsubo cardiomyopathy). Int J Cardiol 2007;120:205-11.
9. Sharkey SW, Lesser JR, Zenovich AG, Maron MS, Lindberg J, Longe TF, et al. Acute and reversible cardiomyopathy provoked by stress in women from the United States. Circulation 2005;111:472-9.
10. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Shulman SP, Gerstenblith G, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med 2005;352:539-48.
11. Shinozaki K, Tamura A, Abe Y, Yano S, Kadota J. Left ventricular free wall rupture in takotsubo cardiomyopathy. Int J Cardiol 2007;115:e3-4.
12. Nef HM, Möllmann H, Hilpert P, Masseli F, Troidl C, Rolf A, et al. Severe mitral regurgitation in Tako-Tsubo cardiomyopathy. Int J Cardiol 2009;132:e77-9.
13. Syed FF, Asirvatham SJ, Francis J. Arrhythmia occurrence with takotsubo cardiomyopathy: A literature review. Europace 2011;13:780-8.
14. Kato Y, Takeda H, Furuya D, Deguchi I, Tanahashi N. Takotsubo cardiomyopathy and cerebral infarction. Rinsho Shinkeigaku 2009;49:158-66.
15. Gogas BD, Antoniadis AG, Zacharoulis AA, Kolokathis F, Lekakis J, Kremastinos DT. Recurrent apical ballooning syndrome "The masquerading acute cardiac syndrome". Int J Cardiol 2011;150:e17-9.