

Integral role of cardiovascular magnetic resonance imaging in the diagnostic workup of suspected takotsubo cardiomyopathy: Avoiding misdiagnosis

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Abstract

Takotsubo cardiomyopathy (TC), or stress cardiomyopathy, is an increasingly recognized acute but reversible myopathic process affecting the ventricle. Although specific criteria have been published to diagnose this entity, traditionally, coronary angiography has been used to exclude coronary artery disease in this condition. We present a case initially suspected to be TC based on clinical and angiographic data. However, cardiovascular magnetic resonance with delayed enhancement imaging with gadolinium identified occult coronary artery disease and refuted the original diagnosis of TC. Cardiovascular magnetic resonance should be an integral part of the diagnostic workup of suspected Takotsubo cardiomyopathy. (Cardiol J 2007; 14: 592–594)

Key words: takotsubo cardiomyopathy, cardiac magnetic resonance imaging

Introduction

Takotsubo cardiomyopathy (TC) or stress cardiomyopathy (also known as transient left ventricular apical ballooning, and broken heart syndrome) is an increasingly recognized acute but reversible myopathic process affecting the ventricle. Although originally described in Japanese populations [1, 2], it has been increasingly reported in European and North American populations [3, 4]. Most patients appear to have near normalization of left ventricular function and wall motion in the ensuing few weeks after the index event. The exact etiology is unclear, but is believed to be a result of profound sympathetic excess leading to a diffuse microvascular spasm or dysfunction leading to acute stunning [5]. This syndrome necessitates coronary angiography to rule out obstructive coronary artery

disease (CAD). Contrast-enhanced cardiovascular magnetic resonance (CMR) is a powerful, non-invasive technique for the assessment of cardiac morphology and function. First-pass perfusion and delayed enhancement imaging after administration of gadolinium contrast enables the distinction between reversible and irreversible injury to the myocardium, regardless of severity of wall motion abnormalities or age of infarct [6, 7]. CMR utilization in TC has to date been mainly an “adjunct” diagnostic modality. It has been used to study the features of TC and confirm the absence of myocardial necrosis by gadolinium-based delayed enhancement imaging. We report a case initially diagnosed as TC, in which CMR imaging demonstrated that myocardial infarction was in fact the true diagnosis.

Case report

A 52-year-old female with prior history of hypertension, diabetes, mild mental retardation and no known coronary artery disease was admitted with urosepsis. Initial ECG revealed T wave inversion in I, II, aVL, aVF and V3–V6 with poor R wave progression. A troponin test, carried out at presentation due to EKG abnormalities, was normal.

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Received: 23.04.2007

Accepted: 17.07.2007

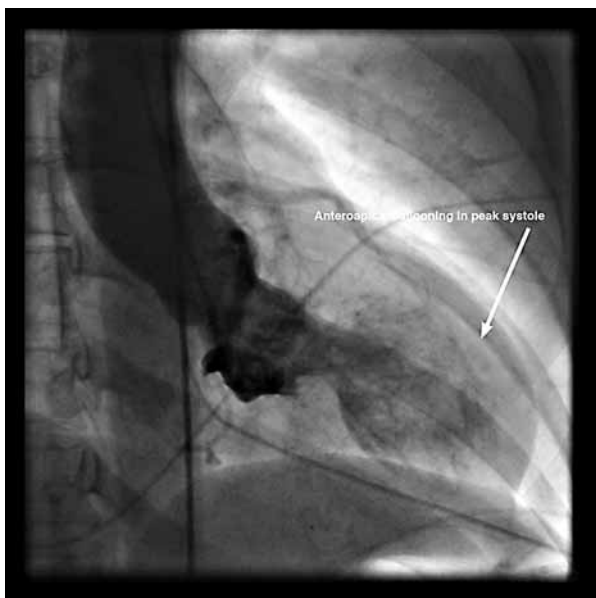


Figure 1. Coronary angiography revealed severe apical and distal septal dyskinesia consistent with apical ballooning.

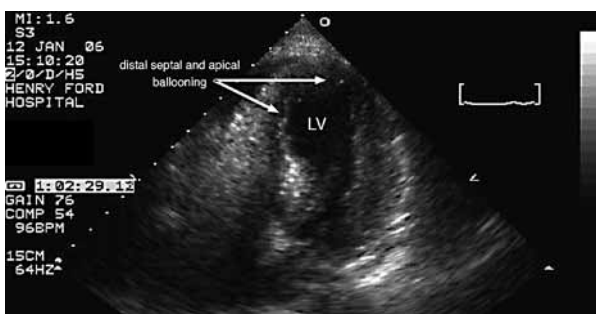


Figure 2. 2D echocardiography showed left ventricular apical ballooning at peak systole.

Within 48 hours of hospitalization, she developed profound shortness of breath and was transferred to the intensive care unit. ECG showed sinus tachycardia and ST elevation in V1–V6. Repeat troponin was 0.24 (normal: ≤ 0.2). Subsequently, serial troponins were negative. Coronary angiography was performed and revealed normal coronary arteries and moderate left ventricular dysfunction with severe apical and distal septal dyskinesia (apical ballooning) (Fig. 1). 2D echocardiography confirmed the wall motion abnormalities (Fig. 2), and left ventricular ejection fraction was 30–35%. A provisional diagnosis of TC was established, and gadolinium-enhanced CMR was performed to

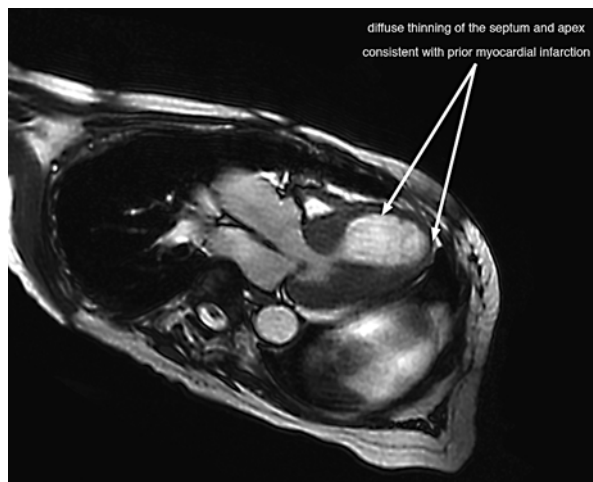


Figure 3. Cardiovascular magnetic resonance revealed thinned anteroseptal, anteroapical and periapical myocardium.

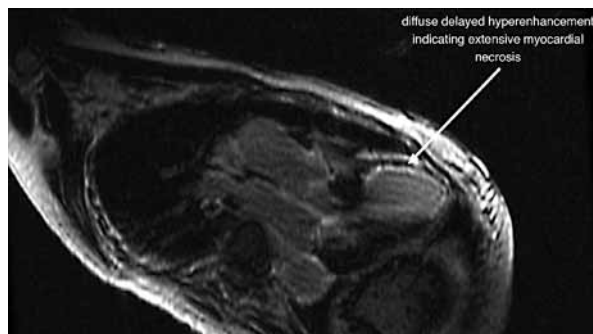


Figure 4. Cardiovascular magnetic resonance with gadolinium revealed diffuse abnormal delayed subendocardial myocardial enhancement involving approximately 50% of the periapical, mid-distal anteroseptal wall consistent with prior myocardial infarction due to coronary atherosclerosis.

highlight the features of TC. CMR revealed thin anteroseptal, anteroapical and periapical myocardium with concomitant severe hypo-akinesia and periapical dyskinesia (Fig. 3). There was diffuse, delayed subendocardial enhancement involving 50% of the periapical and mid-distal anteroseptal walls and focal subendocardial enhancement in the basal anteroseptal region (Fig. 4). These findings were compatible with ischemic etiology and established CAD as the diagnosis. Repeat 2D echocardiography 2 weeks after hospital discharge showed persistent left ventricular dysfunction and wall motion abnormalities.

Discussion

This is a case of prior silent CAD with transient peri-infarct ischemia masquerading as TC. Tako-tsubo cardiomyopathy usually presents similarly to an acute coronary syndrome and consists of an acute onset of transient akinesis or dyskinesis of the apical and mid-ventricular segments of the left ventricle. It is often accompanied by dynamic, reversible ST segment elevation or T wave inversion and may have troponin release [3]. Normalization of wall motion abnormalities after the acute event have been documented to start as early as 48 hours after diagnosis of TC. The vast majority of patients are postmenopausal women. An acute emotional or physiologic stressor triggering the event can often be identified [4, 5]. Our patient had a mildly elevated troponin I (0.24, normal: < 0.20) in setting of sepsis. Coronary angiography showed normal coronary arteries and apical ballooning. According to traditional criteria, this patient would be misdiagnosed as TC. In our case, the diffuse subendocardial hyperenhancement in left anterior descending artery distribution with thinned walls was diagnostic for myocardial infarction. CMR features of TC include periapical left ventricular dilation with wall motion preserved in the basal left ventricular segments and a lack of gadolinium enhancement indicating an absence of myocardial necrosis [8]. A large case series of TC patients reported an absence of delayed hyperenhancement in 21/22 patients [6, 7]. Isolated case reports have described patchy, delayed hyperenhancement in TC [9, 10]. Thus the CMR findings, along with persistent echocardiographic ventricular dysfunction 2 weeks after the event, suggested CAD as the more likely diagnosis. We hypothesize that the patient had had a prior silent myocardial infarction (history of diabetes and abnormal EKG). In the setting of sepsis, she manifested transient peri-infarct ischemic changes in the left anterior descending territory.

Conclusions

This case illustrates the importance of cardiovascular magnetic resonance in the diagnostic workup of suspected TC. Cardiovascular magnetic resonance currently serves as the gold standard for diagnosing myocardial infarctions not appreciated on echocardiography or nuclear imaging. The implications of diagnosing CAD are completely

different from TC given the benign long-term outcomes of the latter after the acute stage. We recommend that cardiovascular magnetic resonance with delayed enhancement with gadolinium be performed in all patients with suspected TC in order to exclude definitively CAD, particularly if wall motion abnormalities fail to resolve.

References

1. Kawai S, Suzuki H, Yamaguchi H et al. Ampulla cardiomyopathy ('Takotsubo' cardiomyopathy): Reversible left ventricular dysfunction: with ST segment elevation. *Jpn Circ J*, 2000; 64: 156–159.
2. Kurisu S, Sato H, Kawagoe T et al. Tako-tsubo-like left ventricular dysfunction with ST-segment elevation: a novel cardiac syndrome mimicking acute myocardial infarction. *Am Heart J*, 2002; 143: 448–455.
3. Desmet WJ, Adriaenssens BF, Dens JA. Apical ballooning of the left ventricle: First series in white patients. *Heart*, 2003; 89: 1027–1031.
4. Seth PS, Aurigemma GP, Krasnow JM, Tighe DA, Untereker WJ, Meyer TE. A syndrome of transient left ventricular apical wall motion abnormality in the absence of coronary disease: a perspective from the United States. *Cardiology*, 2003; 100: 61–66.
5. Bybee KA, Kara T, Prasad A et al. Systematic review: Transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. *Ann Intern Med*, 2004; 141: 858–865.
6. Kim RJ, Wu E, Rafael A et al. The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. *N Engl J Med*, 2000; 43: 1445–1453.
7. Wagner A, Mahrholdt H, Sechtem U, Kim RJ, Judd RM. MR imaging of myocardial perfusion and viability. *Magn Reson Imag Clin N Am*, 2003; 11: 49–66.
8. Sharkey SW, Lesser JR, Zenovich AG et al. Acute and reversible cardiomyopathy provoked by stress in women from the United States. *Circulation*, 2005; 111: 472–479.
9. Fritz J, Wittstein IS, Lima JA, Bluemke DA. Transient left ventricular apical ballooning: Magnetic resonance imaging evaluation. *J Comput Assist Tomogr*, 2005; 29: 34–36.
10. Haghi D, Fluechter S, Suselbeck T, Borggrefe M, Papavassiliu T. Delayed hyperenhancement in a case of Takotsubo cardiomyopathy. *J Cardiovasc Magn Reson*, 2005; 7: 845–847.