

CASE REPORT

Delayed hyperenhancement in a case of Takotsubo cardiomyopathy

DARIUSCH HAGHI, M.D.,* STEPHAN FLUECHTER, M.D., TIM SUSELBECK, M.D., MARTIN BORGGREFE, M.D., and THEANO PAPAVALASSILIU, M.D.

I. Medical Department, University Hospital Mannheim, Mannheim, Germany

Takotsubo cardiomyopathy (TTC) consists of an acute onset of transient akinesia of various parts of the left ventricle (apex and mid in classical TTC, mid and base in the variant form), without significant coronary artery stenosis, often accompanied by chest pain, dynamic reversible ST-T segment abnormalities and elevation of cardiac enzymes disproportionate to the extent of akinesia. Contrast-enhanced cardiovascular magnetic resonance (CMR) is a useful adjunct in the diagnostic work up of patients with TTC. Delayed hyperenhancement on gadolinium-enhanced CMR, which is indicative of active inflammation (e.g. myocarditis) or myocardial fibrosis (e.g. myocardial infarction), is usually absent in patients with TTC. In this report we present the case of a 46-years old women with TTC who had an extensive area of apical and midventricular akinesia and in whom gadolinium-enhanced CMR demonstrated a small area of subendocardial delayed hyperenhancement. A gadolinium-enhanced CMR performed 6 weeks later exhibited complete reversal of all wall motion abnormalities and an identical area of subendocardial delayed hyperenhancement.

Key Words: Takotsubo cardiomyopathy; Left ventricular apical ballooning syndrome; Cardiovascular magnetic resonance

1. Introduction

The syndrome of transient left ventricular (LV) apical ballooning (takotsubo cardiomyopathy) consists of an acute onset of transient akinesia of the apical and mid portions of the left ventricle, without significant coronary artery stenosis, often accompanied by chest pain, dynamic reversible ST-T segment abnormalities and elevation of cardiac enzymes disproportionate to the extent of akinesia (1). The vast majority of patients are women, and an acute emotional or physiologic stressor triggering the event can often be identified.

Contrast-enhanced cardiovascular magnetic resonance (CMR) provides excellent information on LV morphology and function in affected patients and may be a valuable tool in differentiating takotsubo cardiomyopathy from conditions with similar clinical presentations such as acute myocardial infarction or myocarditis. Delayed hyperenhancement on gadolinium-enhanced CMR, which is indicative of active inflammation (2) (e.g., myocarditis) or myocardial fibrosis (3) (e.g., myocardial infarction), has been absent in patients with takotsubo cardiomyopathy (4–6).

2. Case

A 46-year-old women presented to the emergency department with a 2-hour history of sudden chest pain after a heavy argument with her teenage children. Her past medical history was unremarkable. Her electrocardiogram showed T wave inversions in leads I, II, III, aVL, aVF, V2 to V6 and prolongation of the QT interval (QTc = 502 ms). The troponin I and creatine kinase concentrations were slightly elevated. Left ventriculography demonstrated apical and mid-ventricular akinesia (Figure 1, panel A and B). Coronary angiography demonstrated normal coronary arteries. A gadolinium-enhanced CMR was performed on the sixth hospital day. CMR images were acquired on a 1.5-Tesla Siemens Sonata (Erlangen, Germany) using a phased-array coil during repeated breath-holds. Steady-state free precession images were acquired in multiple short-axis and 2 to 3 long-axis planes. Gadolinium was administered intravenously, and contrast-enhanced images were acquired after 10 minutes with a segmented inversion-recovery technique in identical planes. CMR demonstrated only mild apical and mid-ventricular hypokinesia and a small area of subendocardial delayed hyperenhancement (Figure 1, panel C and D). The patient had an uneventful recovery. A gadolinium-enhanced CMR performed 6 weeks later demonstrated absence of any wall motion abnormalities (WMA) and an identical area of subendocardial delayed hyperenhancement.

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*Address correspondence to Dr. Dariusch Haghi, M.D., I. Medizinische Klinik, Universitätsklinikum Mannheim, Mannheim 68167, Germany; Fax: 0049-621-3832172; E-mail: dariush.haghi@med.ma.uni-heidelberg.de

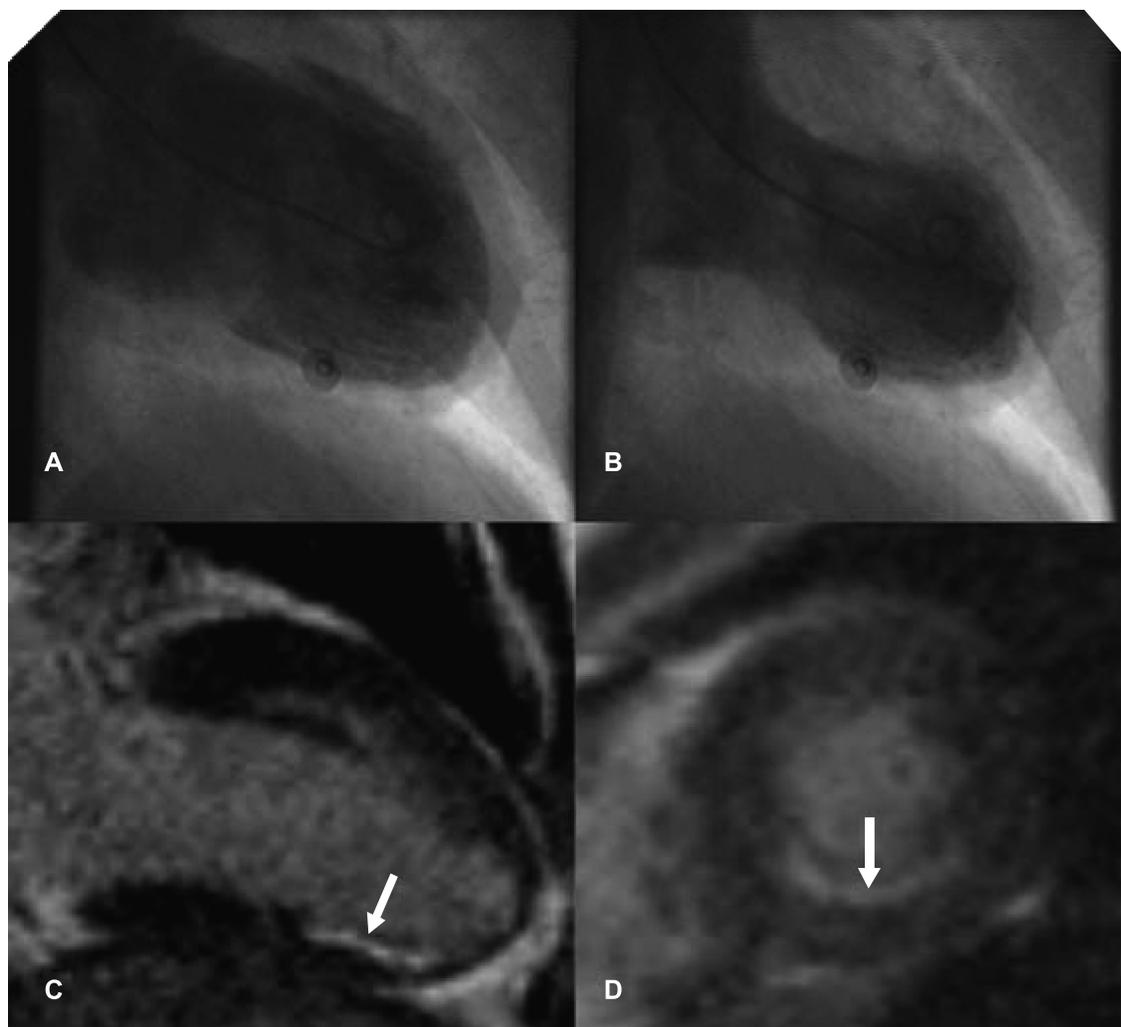


Figure 1. Left ventriculography during diastole (panel A) and systole (panel B) demonstrating apical and mid-ventricular akinesis. Contrast-enhanced cardiovascular magnetic resonance demonstrating a small area of subendocardial delayed hyperenhancement (arrows) in the long axis view (panel C) and in the short axis view (panel D).

3. Discussion

The syndrome of transient LV apical ballooning has been reported predominantly in Japan (7–9), but recently, the syndrome has also been increasingly recognized in European and North American populations (1, 4, 5, 10). The clinical presentation mimics acute coronary syndrome, but significant coronary artery disease has always been absent on coronary angiography. Striking hallmarks of the disease are: 1) WMA beyond a single major coronary artery vascular distribution (1); 2) mild or minimal elevation of cardiac enzymes disproportionate to the extent of akinesia (1); and 3) rapid resolution after sudden onset (11).

Clinical experience with gadolinium-enhanced CMR in takotsubo cardiomyopathy is still limited (4–6). The largest series has been published by Sharkey et al. (5) who were able to demonstrate WMA beyond the vascular distribution of a single coronary artery in all 22 patients. Delayed gadolinium

hyperenhancement was absent in 21 of 22 patients, a finding consistent with viable myocardium and the absence of myocardial inflammation (2) or myocardial fibrosis (3, 5). Only one patient who presented in cardiac arrest showed delayed hyperenhancement confined to the LV apex. To the best of our knowledge, this is the second reported case on a patient with takotsubo cardiomyopathy demonstrating delayed hyperenhancement on CMR. The affected area was small, involving subendocardial myocardium of the midventricular and apical segments of the left ventricle. The pattern of subendocardial hyperenhancement is typically seen in myocardial infarction. This pattern is quite different from that seen in active myocarditis where patchy regions of hyperenhancement originate from the epicardium but never from the endocardium (2).

In conclusion, the vast majority of patients with takotsubo cardiomyopathy demonstrate preserved myocardial viability as assessed by contrast-enhanced CMR. However, small areas

of delayed hyperenhancement, which is indicative of necrosis and decreased viability, may occur in a minority of patients.

References

1. Desmet WJ, Adriaenssens BF, Dens JA. Apical ballooning of the left ventricle: first series in white patients. *Heart* 2003; 89:1027–1031.
2. Mahrholdt H, Goedecke C, Wagner A, Meinhardt G, Athanasiadis A, Vogelsberg H, Fritz P, Klingel K, Kandolf R, Sechtem U. Cardiovascular magnetic resonance assessment of human myocarditis: a comparison to histology and molecular pathology. *Circulation* 2004; 109:1250–1258.
3. Simonetti OP, Kim RJ, Fieno DS, Hillenbrand HB, Wu E, Bundy JM, Finn JP, Judd RM. An improved MR imaging technique for the visualization of myocardial infarction. *Radiology* 2001; 218:215–223.
4. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, Wu KC, Rade JJ, Bivalacqua TJ, Champion HC. Neurohumoral features of myocardial stunning due to sudden emotional stress. *N Engl J Med* 2005; 352:539–548.
5. Sharkey SW, Lesser JR, Zenovich AG, Maron MS, Lindberg J, Longe TF, Maron BJ. Acute and reversible cardiomyopathy provoked by stress in women from the United States. *Circulation* 2005; 111:472–479.
6. 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.
7. Dote K, Sato H, Tateishi H, Uchida T, Ishihara M. Myocardial stunning due to simultaneous multivessel coronary spasms: a review of 5 cases. *J Cardiol* 1991; 21:203–214.
8. Tsuchihashi K, Ueshima K, Uchida T, Oh-mura N, Kimura K, Owa M, Yoshiyama M, Miyazaki S, Haze K, Ogawa H, Honda T, Hase M, Kai R, Morii I. 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; 38:11–18.
9. Abe Y, Kondo M, Matsuoka R, Araki M, Dohyama K, Tanio H. Assessment of clinical features in transient left ventricular apical ballooning. *J Am Coll Cardiol* 2003; 41:737–742.
10. Bybee KA, Prasad A, Barsness GW, Lerman A, Jaffe AS, Murphy JG, Wright RS, Rihal CS. Clinical characteristics and thrombolysis in myocardial infarction frame counts in women with transient left ventricular apical ballooning syndrome. *Am J Cardiol* 2004; 94:343–346.
11. Dec GW. Recognition of the apical ballooning syndrome in the United States. *Circulation* 2005; 111:388–390.