Case report

Magnetic resonance imaging findings in apical ballooning syndrome or takotsubo cardiomyopathy

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KEYWORDS

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ABSTRACT

Cardiac magnetic resonance imaging (CMRI) plays an important role in the diagnosis and follow-up of apical ballooning syndrome (takotsubo syndrome), a recently described cardiac condition characterised by transient dyskinesia of the left ventricle secondary to an acute emotional event. We present the CMRI findings in a 53-year-old female diagnosed with apical ballooning syndrome and discuss its value in the diagnosis and follow-up of this condition.

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Introduction

Transient left ventricular (LV) apical ballooning syndrome is a cardiac syndrome of unknown aetiology usually seen in postmenopausal women following severe emotional or physical stress. It is characterised by transient LV dyskinesia, electrocardiogram (ECG) that may mimic acute myocardial infarction (MI), slightly increased myocardial enzymes in the absence of obstructive coronary artery disease. Our report highlights how cardiac magnetic resonance imaging (CMRI) can be a useful non-invasive tool and can potentially avoid cardiac catheterisation in an appropriate clinical setting.

Case report

A 53-year-old white woman, with no prior cardiac problems, presented to the emergency room (ER) with chest uneasiness and orthopnoea and was found to have ST elevation of >0.5 mm in leads V2–V6 on ECG (Figure 1), deep T inversions in V2–V6, and T inversions in inferior leads. The differential considerations included acute myocardial ischaemia, acute

dilatation of the left ventricle and myocardial contusion. Elevated troponin of 2.14 was noted.

Cardiac catheterisation, revealed normal coronary arteries. A LV angiogram suggested a poor ejection fraction with basal hypercontractility and apical akinesia.

With these findings, it was considered that the patient had an anterior wall MI with spontaneous thrombolysis. An echocardiogram done at the same time also showed markedly decreased ejection fraction of 10–15%.

A cardiac MR study was ordered to rule out MI and was performed using a phased-array body matrix coil with bright blood cine (2 and 4 chamber and short axis views) and delayed enhanced (DE) sequences after gadolinium administration. Cardiac magnetic resonance imaging showed poor ejection fraction measuring <20%, LV apical ballooning and normal contractility of the ventricular base (Figure 2). No abnormal myocardial enhancement was noted on the DE sequence (Figure 3). These findings were suggestive of apical ballooning syndrome and also explained the findings on cardiac catheterisation. Specific stressors on review of the patient's history included recent deaths of her husband and sister.

The patient was managed as non-ischaemic cardiomyopathy in the hospital and was started on angiotensin-converting enzyme inhibitors (ACEI), β -blockers and Lasix. At the time of discharge the patient's vitals were stable.

A follow-up CMRI 3 months later showed completely normal LV contractility as well as marked improvement of

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Figure 1 The electrocardiogram of the patient at the time of presentation shows sinus tachycardia and ST elevation in the precordial leads V2–V6 of >0.5 mm as well as wide spread T wave abnormalities.

Figure 2 Acute stage. (A) Cine True FISP imaging steady-state precession sequence in two chamber-during diastole and four chamber-during systole. (B) Show non-contractility and ballooning of the left ventricular apex. (C) Four chamber delayed postcontrast image shows no abnormal myocardial enhancement during the acute episode.

ejection fraction which increased from 20% to 70%. The LV apical ballooning also showed complete reversal (Figure 3).

Based on the clinical presentation, the diagnostic work up and the magnetic resonance imaging (MRI) findings, the final diagnosis of apical ballooning syndrome was made.

Discussion

Takotsubo cardiomyopathy is a syndrome characterised by the acute onset of chest pain and a completely reversible

regional contractile myocardial dysfunction. First described in the Japanese literature, the apical ballooning was named after the bottle used for trapping octopus with a round bottom and a narrow neck.¹

The characteristics needed for diagnosis include:

- (a) Transient akinesis or dyskinesia of the LV apex and midventricular segments with regional wall motion abnormalities extending beyond a single epicardial vascular distribution,
- (b) Absence of significant obstructive coronary disease,
- (c) New EKG abnormalities (either ST-segment elevation or T wave inversion) and

Figure 3 Three months after the first scan. (A) Cine True fast imaging with steady-state precession sequence in the two chamber-diastolic and four chamber-systolic. (B) Shows normal contractility of the left ventricular apex with complete resolution of the apical ballooning. (C) Four chamber delayed postcontrast sequence again shows no abnormal myocardial enhancement.

(d) Absence of recent significant head trauma, intracranial bleeding, pheochromocytoma, myocarditis and hypertrophic cardiomyopathy.²

Though the cause of this syndrome is unknown, it is consistently observed after intense emotional or physical stress, with strong predominance among postmenopausal women. During the course of the acute phase, complications like pulmonary oedema, cardiogenic shock and fatal arrhythmias may be seen.³

No definite explanation exists regarding the mechanism for this syndrome. The most widely proposed theory relates to the role of catecholamine secondary to increased sympathetic activity related to endogenous (emotional) or exogenous stresses (trauma or surgical procedure) with elevated levels of catecholamine as seen in these patients.⁴ Oxidation of catecholamine results in formation of highly toxic substances and free radicals causing intracellular calcium overload and myocardial cell damage. The distinctive contractile pattern may be explained by an enhanced responsiveness of apical myocardium to sympathetic stimulation. Alternatively, a base-to-apex gradient could result in regional differences in myocardial blood flow in the setting of catecholamine-mediated epicardial or microvascular vasoconstriction.⁵

Another theory suggests that microvascular dysfunction contributes substantially towards the development of this syndrome though it is unclear whether this is the primary or secondary mechanism involved in the development of this syndrome. Furthermore, the underlying cause of the microvascular dysfunction is unknown.⁶

Other mechanisms thought responsible for this condition include epimyocardial vasospasm, though the wall motion abnormality does not correspond to a single vascular territory.⁴

Though the typical appearance of this syndrome is well visualised by echocardiography and ventriculogram during coronary angiography, coronary angiograms can exclude significant coronary artery disease.⁷ The CMRI definitively rules out the presence of an underlying infarction which is the most important differential diagnostic consideration in such

patients. This is because it has been conclusively shown that delayed hyper-enhancement seen on MRI (DE-MRI) is exclusively associated with myocyte necrosis.^{8,9} The utility of CMRI in apical ballooning was recently investigated by Mitchell et al.¹⁰ Though, there were concerns about resolution of CMRI to detect changes at microscopic level, excellent correlation has been shown between DE-MRI and histopathology even for small infarcts.^{8,9,11,12}

It is important to note that regions subjected to severe but reversible ischaemic injuries do not hyper-enhance on DE-MRI, even in the presence of myocardial stunning. Similarly, regions within areas at risk but outside of areas of infarction do not exhibit hyper-enhancement.¹³ This explains why in patients with apical ballooning there is an absolute lack of hyper-enhancement of the myocardium because it essentially is a reversible stunned myocardium. The DE-MRI is by far the most accurate modality which can make the distinction between infarcted and ischaemic myocardium. It can suggest presence of other conditions like myocarditis which can mimic apical ballooning syndrome in terms of clinical presentation.¹³

Cardiac magnetic resonance imaging by demonstrating the characteristic wall motion abnormality extending beyond a single vascular territory also argues against MI as an underlying aetiological factor.

Moreover, the combined use of DE-MRI and morphological assessment can predict those patients who are most likely to experience improvement in LV ejection fraction.^{13–15} Since, DE-MRI showed no hyper-enhancement of the myocardium, it can be predicted that on follow-up complete reversal of the morphological abnormalities should be observed, as was seen in our case (Figure 3).

There are no established guidelines for treatment of this condition. However, patients are evaluated and treated initially in a manner similar to an acute MI.

In conclusion, apical ballooning syndrome is a unique cardiac syndrome which has characteristic clinical and imaging findings and in the appropriate clinical setting, performing

CMRI early in the work up may prove valuable in the patient management and may avoid invasive procedures.

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