CASE REPORT

STEMI mimicker in a 26-year-old man

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SUMMARY

We herein describe a case of acute myocarditis which may mimic myocardial infarction, since affected patients experience 'typical' chest pain, the ECG changes are identical to those observed in acute coronary syndromes, and serum markers are increased. This case emphasises the importance of performing appropriate cardiac MRI to help in the differential and definitive diagnosis as well as the extent of myocardial involvement. ST elevation myocardial infarction is rare in young adults and when it is encountered, it should raise the differential diagnosis of its mimickers.

BACKGROUND

Cardiac MRI might have a greater evaluation value for diagnosis of acute myocarditis and its extent.¹ Cell-level change such as cell inflammation and tissue damage have all been involved in the setting of myocarditis.² Distinction between acute myocardial infarction and myocarditis is based on the segment of wall-motion abnormalities.³ Prior clinical and pathological studies already have established the specificity of myocardial delayed enhancement on cardiac MRI in diagnosing acute myocarditis.⁴ Differential diagnosis of ST elevation myocardial infarction (STEMI) mimickers in our case should be entertained in a young patient presenting with STEMI. The MRI findings in our study were also inconsistent with myocardial infarction because the MRI did not demonstrate endomyocardial enhancement the pattern found in myocardial infarction.⁵ Myocarditis usually shows a characteristic pattern of contrast enhancement, which originates primarily from the epicardium, sparing the subendocardial layer.⁵ There was no significant relationship between the findings on transthoracic echocardiogram (TTE), cardiac MRI and the biochemical markers of myocardial damage.¹ The meaningful relationship between ECG's ischaemic change and transmural myocardial oedema found in cardiac magnetic resonance in patients with clinically suspected acute

myocarditis was reported,⁶ which could be applicable to our study (figures 1 and 2).

CASE PRESENTATION

A 26-year-old man with no medical history presented to the emergency room with acute central chest pain of 4 hours duration, waking him up from sleep. The ECG showed 3 mm ST elevation in the inferior leads. His physical examination was normal, and he was haemodynamically stable. The initial cardiac enzymes were elevated with troponin-I of 27.8 ug/mL, Creatine Kinase (CK) 3265 units/L, and CK-MB (Creatine Kinase-muscle/brain) 67.1 ng/dL. Though young, with no cardiac risk factors, he met the criteria for STEMI activation. He received aspirin, nitroglycerin, beta-blocker, high dose statin and intravenous heparin infusion in the emergency room and was taken to the cardiac catheterisation laboratory. Cardiac catheterisation showed normal coronary arteries with no obstruction or dissection. Transthoracic echocardiogram showed normal wall motion with left ejection fraction 60%. Suspicion of myocarditis was entertained, and hence cardiac MRI was ordered. It showed left ventricle inferior base hypokinesis with transmural delayed enhancement, mild enhancement of distal lateral wall, proximal septum and anterior wall and pericardial enhancement in the inferior base and no pericardial effusion or thickening confirming a diagnosis of myopericarditis with transmural inflammation of the inferior base resulting in ST elevation and chest pain possibly because of intense regional pericarditis. Serological markers for coxsackievirus, adenoviruses, herpes virus, influenza virus, hepatitis C, parvovirus B 19, *B. burgdorferi* and antinuclear antibody came back negative subsequently. He responded rapidly to colchicine and high dose aspirin. Left ventricular inferior wall-motion abnormalities was moderately hypokinetic. Cardiac MRI is unique in that it provides non-invasive myocardial tissue characterisation based on the local chemical microenvironment.⁷ Delayed transmural enhancement was found predominantly in the inferior wall (figures 3 and 4). And it also showed in the mid-lateral wall and proximal septum and anterior wall. TTE findings was not correlated with cardiac MRI findings. The MRI in our patient was classic for myocarditis, with mid-myocardial enhancement of the septum, which spared the subendocardium and corresponded to the ECG changes, and wall-motion abnormality.

INVESTIGATIONS

Cardiac MRI is unique in that it provides non-invasive myocardial tissue characterisation based on the local chemical microenvironment.⁷ Delayed

Figure 1 ST-segment elevation in leads II, III and aVF with V4-6 ST elevation and reciprocal change in V1, 2 suggests inferior myocardial infarction.

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Figure 2 Normal sinus rhythm, 1 day after the first day of admission.

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DIFFERENTIAL DIAGNOSIS

Serological markers for coxsackievirus, adenoviruses, herpes virus, influenza virus, hepatitis C, Parvovirus B 19, *B. burgdorferi* and antinuclear antibody came back negative subsequently.

TREATMENT

He responded rapidly to colchicine and high dose aspirin for treatment of myocarditis.

OUTCOME AND FOLLOW-UP

No follow-up so far. But No more emergency department visit so far.

DISCUSSION

Focal myocarditis, cardiac sarcoid, coronary or aortic dissection, coronary arteritis and coronary embolism can produce

Figure 3 Late gadolinium cardiac MRIs in two-chamber and long axis planes demonstrating transmural myocardial gadolinium enhancement in the inferior wall; classic MRI features of myocarditis.

Figure 4 Late gadolinium cardiac MRIs in short axis planes demonstrating transmural myocardial gadolinium enhancement in the inferior wall; classic MRI features of myocarditis.

STEMI or STEMI-like syndrome, and appropriate multimodality imaging may help in the differential and definitive diagnosis. We conclude that cardiac MRI provides non-invasive imaging that may obviate invasive procedures such as coronary catheter angiography or endomyocardial biopsy.

Learning points

- ▶ ST elevation myocardial infarction is rare in young adults and when it is encountered, it should raise the differential diagnosis of its mimickers.
- ▶ No correlation was found between the degree or extent of imaging findings on transthoracic echocardiogram, cardiac MRI.
- ▶ The meaningful relationship between ECG's ischaemic change and transmural myocardial oedema as evidenced by cardiac magnetic resonance in patients with clinically suspected acute myocarditis should be considered.
- ▶ MRI could be one of the modalities to make diagnosis and lead to better management.

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REFERENCES

- 1 De Lazzari M, Zorzi A, Baritussio A, *et al.* Relationship between T-wave inversion and transmural myocardial edema as evidenced by cardiac magnetic resonance in patients with clinically suspected acute myocarditis: clinical and prognostic implications. *J Electrocardiol* 2016;49:587–95.
- 2 Franco A, Javidi S, Ruehm SG, *et al.* Delayed Myocardial Enhancement in Cardiac Magnetic Resonance Imaging. *J Radiol Case Rep* 2015;9:6–18.
- 3 Goitein O, Matetzky S, Beinart R, *et al.* Acute myocarditis: noninvasive evaluation with cardiac MRI and transthoracic echocardiography. *AJR Am J Roentgenol* 2009;192:254–8.

- 4 Hiramitsu S, Morimoto S, Kato S, *et al*. Significance of transient left ventricular wall thickening in acute lymphocytic myocarditis. *Heart Vessels* 2007; 22:25–9.
- 5 Hoey ET, Shahid M, Ganeshan A, *et al*. MRI assessment of cardiac tumours: part 1, multiparametric imaging protocols and spectrum of appearances of histologically benign lesions. *Quant Imaging Med Surg* 2014;4:478–88.
- 6 Khan TA, Chowdhury AW, Khan HILR, *et al*. Echocardiographic comparison of regional wall motion abnormality between patients with acute anteroseptal and acute extensive anterior ST segment elevation myocardial infarction. *Bangladesh Med Res Counc Bull* 2015;41:35–40.
- 7 Laissy JP, Messin B, Varenne O, *et al*. MRI of acute myocarditis: a comprehensive approach based on various imaging sequences. *Chest* 2002;122:1638–48.

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