CASE REPORT

Myocardial infarction masquerading as myocarditis in a patient with factor V Leiden: unmasked with MR

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SUMMARY

We present a case of a 21-year-old man presenting with sharp left-sided chest pain. A CT pulmonary angiogram was negative, ECG was unremarkable and a mild troponin rise was observed. Myocarditis was suspected as the most likely diagnosis, particularly in view of the patient's previous diagnosis of myocarditis 3 years prior. A cardiac MRI was indicative of an acute mid-anterior myocardial infarction (MI) and an old inferior MI with an associated aneurysm. A subsequent angiogram revealed a subtotal occlusion in the second diagonal artery, likely precipitated by homozygous factor V Leiden. This case illustrates the value of MRI in differentiating acute MI from myocarditis when clinical suspicion is low, as in this young patient with atypical chest pain. Further, it demonstrates the value of MRI in detecting previous MIs and reinforces the importance of searching for precipitants of MI in young patients.

BACKGROUND

Atypical chest pain and younger age reduces the likelihood of coronary artery disease considerably and therefore suspicion of myocardial infarction (MI).¹ However, factors such as familial hypercholesterolaemia, illicit drug use, congenital coronary artery abnormalities and hypercoagulable states may precipitate MI in young patients.² Complications from MI are significantly increased with delay in treatment,³ therefore accurate and prompt diagnosis is essential to reduce the morbidity associated with MI, which can be particularly marked in younger patients.^{2,4}

Here, we present the case of a 21-year-old man presenting with atypical chest pain with a recent diagnosis of homozygous factor V Leiden. Cardiac MRI identified an acute anterior MI and an old inferior MI resulting in aneurysm formation, previously misdiagnosed as myocarditis.

CASE PRESENTATION

A 21-year-old man presented with left-sided stabbing chest pain and mild dyspnoea. His symptoms had been present for 2 hours. His medical history was remarkable for an episode of myocarditis 3 years previously, a pulmonary embolism 2 years prior with a subsequent diagnosis of homozygous factor V Leiden. He takes 4 mg of acenocoumarol once daily. The patient denied smoking and illicit substance abuse. He had no significant family history of cardiovascular disease. Examination was

unremarkable. Serial ECGs showed normal sinus rhythm. Initial troponin T (2 hours since onset of pain) was 0.39 ng/mL (0.000–0.030) and D-dimer was 310 ng/mL (<255). A pulmonary embolism was suspected and a CT pulmonary angiography performed, which was negative. Four-hour troponin was 0.66 ng/mL. Differential diagnoses of recurrent myocarditis and MI were considered. Echocardiography showed an estimated left ventricular ejection fraction of 55%–59% and no abnormality of segmental or global contractility was detected.

Cardiac MRI showed delayed enhancement in the mid-anterior segment associated with tissue oedema in the subendocardial territory by T2 short-tau inversion recovery sequence, in keeping with acute ischaemia in a diagonal territory (figure 1). Furthermore, there was transmural delayed enhancement with a 2×3 mm clot in the apical inferior/apical lateral segments in the setting of aneurysmal formation, in keeping with an old infarction (figure 2). Left ventricular function was low normal with a left ventricular ejection fraction of 55%. The apical inferior segment of the left ventricle was dyskinetic, all other segments were contracting normally.

Subsequent angiography revealed an 80% inlet stenosis of the second diagonal branch of the left anterior descending artery. A diagnosis of non-ST segment elevation MI was made, likely precipitated by homozygous factor V Leiden. The patient was started on medical therapy alone, due to size of the vessel and proximal location of the lesion the risk of damaging the left anterior descending artery was too high to justify percutaneous coronary intervention.

OUTCOME AND FOLLOW-UP

This case demonstrates that the patient's previous diagnosis of myocarditis was likely to be a misdiagnosis, the cardiac MRI suggesting a missed inferior MI. The efficacy of cardiac MRI in differentiating acute and old MIs has been well demonstrated.⁵ Further, cardiac MRI has been shown to effectively differentiate MI from other aetiologies (especially myocarditis) in patients with troponin positive chest pain and unobstructed coronary arteries on X-ray angiography.⁶

DISCUSSION

This case highlights the value of cardiac MRI for identifying or ruling out MI, particularly in the

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Figure 1 Cardiac MRI with T2 short-tau inversion recovery demonstrating increased signal intensity in the subendocardial and mid-myocardial layer in the mid-anterior segment of the left ventricle, in keeping with tissue oedema (suggestive of an acute myocardial infarction).

Figure 2 Cardiac MRI with delayed enhancement in the mid-anterior and apical inferior segments of the left ventricle.

setting of chest pain and a hypercoagulable state. However, hypercoagulable states may go undiagnosed, therefore, considering MRI in a young patient with atypical chest pain, a normal ECG and a troponin rise may be an appropriate early investigation to identify or rule out MI.

Learning points

- ▶ A high clinical suspicion of myocardial infarction (MI) should be maintained in young patients presenting with suspected myocarditis.
- ▶ Consider underlying precipitants of MI in young patients, such as hypercoagulable states.
- ▶ Cardiac MRI can be used as non-invasive method for differentiating acute MI from myocarditis and detecting previous MIs.

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