Novel diagnostic procedure

The importance of cardiac MR in diagnosis of myocarditis in a 30-year-old man presenting with inferior ST elevation, elevated cardiac troponin but unobstructed coronary arteries

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Summary

A 30-year-old man presented with chest pain, inferior ST elevation and elevated troponin T. Emergency coronary angiography showed unobstructed coronary arteries. A clinical diagnosis of myocarditis was made. This was supported by cardiac MRI (CMR) which showed a dilated left ventricle with severely impaired systolic function, and patchy delayed gadolinium enhancement (DGE). He was admitted to the coronary care unit where he had episodes of monomorphic ventricular tachycardia (VT). He was treated with ACE inhibitors, β -blockers and corticosteroids. Targeted cardiac biopsies were taken during admission, and also 6 weeks later; none showed histological evidence of myocarditis. Repeat CMR on day 11 showed improvement in left ventricular systolic function, extent of oedema and DGE. He has subsequently had episodes of nonsustained ventricular tachycardia and evidence of established myocardial scar on CMR several months later.

BACKGROUND

Acute cardiac symptoms associated with elevated cardiac biomarkers are a common presentation. Coronary angiography is normal in approximately 10% of such cases.¹ The diagnosis of myocardial infarction (MI) due to angiographically 'silent' acute coronary syndrome is often made. Cardiac MRI (CMR) provides the ability to distinguish between inflammatory and ischaemic causes of these acute myocardial syndromes, potentially avoiding inappropriate lifelong antiplatelet therapy with its associated costs and complications. In addition, acutely presenting myocarditis is associated with adverse cardiac outcomes.

CASE PRESENTATION

Introduction

A 30-year-old Caucasian man presented with a 12 h history of central chest pain radiating to his left arm. Admission 12 lead ECG showed ST elevation in leads II, III and aVF and ST depression in V1–6 (figure 1). Admission troponin T was elevated at 6.53 $\mu\text{g/l}$ (reference range <0.04). Emergency coronary angiography showed unobstructed coronary arteries. On direct questioning, he reported 'flu-like' symptoms a month prior to admission from which he had completely recovered. He was an ex-smoker of 7–10 cigarettes/day.

Figure 1 Admission ECG.

Figure 2 Day 1 cardiac MR. Long-axis and short-axis late gadolinium enhancement (LGE) sequences (left) showing patchy myocardial enhancement (red arrows) in a subepicardial distribution suggestive of a myocarditic process. T2 STIR sequences (right) showing myocardial oedema (green arrows) with corresponding LGE images.

Clinical course

A clinical diagnosis of myocarditis was made after a CMR was performed the following day. This showed a dilated left ventricle (LV end diastolic volume 228 ml) with severe global systolic impairment (EF 38%). There was extensive patchy subepicardial and transmural delayed gadolinium enhancement (DGE), along with corresponding increased signal on T2 STIR imaging. This extended into the right ventricular diaphragmatic surface (figure 2).

OUTCOME AND FOLLOW-UP

He was admitted to the coronary care unit where he had multiple episodes of monomorphic non-sustained ventricular tachycardia (VT) and chest pain. He was treated with β -blockers, ACE inhibitors and empirically started on corticosteroids the day after admission. CT imaging of his chest was normal and an autoimmune screen was negative. Viral serology demonstrated Epstein-Barr virus and Parvovirus B19 IgG, but no IgM. Three LV cardiac biopsy samples, from the septum and inferior wall, targeted to the regions of greatest DGE, were obtained on day 7 (figure 3). These demonstrated focal vacuolisation of myocytes and occasional contraction bands, but inflammatory infiltrates and myocyte necrosis were absent. Polymerase chain reaction testing for viral

Figure 3 Day 7 myocardial biopsy. This shows focal vacuolisation, but no significant inflammation or necrosis.

Figure 4 Day 11 cardiac MR. Corresponding views to figure 2 showing improvement in both late gadolinium enhancement and oedema after 11 days of treatment with corticosteroids.

genomes was negative. Repeat CMR was performed on day 11, and showed some improvement in LV systolic function (LVEF 49%) and a reduction in the volume of DGE and oedema (figure 4). The patient was discharged on day 12. Six weeks following discharge, ambulatory ECG detected non-sustained VT and a repeat CMR detected evidence of persisting cardiac inflammation. Repeat biopsy (RV septum) also failed to show evidence of inflammatory infiltrates (figure 5). Symptomatic episodes of nonsustained VT continue to be detected by a REVEAL device up to 8 months following the initial presentation.

DISCUSSION

Approximately 3% of patients presenting with acute ST elevation myocardial infarction (STEMI), and up to 12% presenting with non-ST elevation myocardial infarction (NSTEMI) with elevated cardiac troponin have culprit-free angiograms.¹ Current European Society of Cardiology guidelines suggest treatment of all such patients with antiplatelet agents and statins.² Cardiac biopsy, considered the gold standard for the diagnosis of myocarditis, has notoriously low sensitivity,^{3 4} owing to the patchy nature of the disease, and is a serious limitation for this invasive diagnostic technique. In contrast, CMR can differentiate between MI and other causes of acute myocardial damage with a high sensitivity and specificity.^{5 6} Furthermore, interval changes in CMR findings may provide a sensitive method for disease surveillance. CMR availability is essential for patient diagnosis

Figure 5 Six-week myocardial biopsy. This shows mild oedema and occasional contraction bands, but no fibrosis or evidence of myocarditis.

and management in centres managing acute cardiac presentations.⁶

ECG appearances in myocarditis may mimic acute coronary syndromes. In this case, although the epicardium in the inferior and anterior territories appear similarly affected on CMR, the ECG showed ST elevation inferiorly, and ST depression anteriorly. ECG abnormalities in myocarditis evolve, and the time courses in the anterior and inferior territories may have been different. Putative mechanisms of damage in myocarditis include endothelial dysfunction; and this may have been of greater severity in one territory than the other.

Learning points

- ▶ Diagnoses other than acute myocardial infarction should be considered in patients presenting with chest pain and ST elevation.
- ▶ Cardiac biopsy has a low sensitivity for the diagnosis of myocarditis.
- ▶ Cardiac MRI (CMR) can differentiate between myocardial infarction and other causes of acute myocardial damage.
- ▶ In this clinical setting, the diagnosis of myocarditis is based the history, ECG findings, cardiac troponins, angiography and CMR appearances.

Competing interests None.

Patient consent Obtained.

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