Original Article

Recurrent Myocarditis—An Important Mimic of Ischaemic Myocardial Infarction

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Abstract: **Patients presenting with a syndrome of chest pain, elevated cardiac enzyme levels with or without electrocardiogram changes are a common diagnostic and management problem in cardiology. Most commonly, this is due to ischaemic myocardial infarction secondary to coronary artery disease. However, when coronary angiography does not demonstrate any obstructive coronary artery lesion, the diagnosis of myocarditis should be considered. Cardiac magnetic resonance imaging is helpful towards making this diagnosis. Here, we describe the first reported Australian cases of recurrent myocarditis presenting with ischaemic chest pain and elevated cardiac enzyme levels. These cases serve as an important reminder to clinicians that myocarditis is an important mimic of ischaemic myocardial infarction.**

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Keywords. Myocarditis; Myocardial infarction; Cardiac magnetic resonance imaging; Endomyocardial biopsy

Clinical record

Case 1

A 39 year-old male had been diagnosed as having had two non-ST elevation myocardial infarctions. On the first occasion, at the age of 33, following a gastroenteritis illness, he developed episodes of chest pain which radiated down his arm. There were progressive T wave changes on his electrocardiographs (ECGs). His serum troponin was elevated at 5.7 μg/L (normal < 0.03 μg/L). A clinical diagnosis of non-ST elevation myocardial function was made and coronary angiography was performed. This demonstrated normal coronary arteries with a localised area of hypokinesis in the mid inferior/inferobasal segment of the left ventricle. It was assumed that the event was due to thrombotic occlusion of a coronary artery, which had re-canalised spontaneously. Interestingly, a transthoracic echocardiogram performed soon after the coronary angiogram showed normal left ventricular function, without any regional wall motion abnormalities. At the age of 39, following a respiratory tract infection, the patient presented to another hospital with collapse and chest pain. Serum troponin level was elevated at 3.84 μg/L (normal < 0.03 μg/L). There were no acute ECG changes. The patient underwent a repeat coronary angiogram at the second institution. This demonstrated normal coronary arteries with hypokinesis of mid-inferior segment of his left ventricle. It was again assumed that the patient suffered a non-ST elevation myocardial infarction in the setting of infection, due to an underlying pro-thrombotic condition. However, thrombophilic screen including Factor V Leiden mutation, prothrombin gene mutation, anti-thrombin III deficiency, protein C and S deficiencies were unremarkable. There was no history of rheumatological or autoimmune illnesses, with a negative autoimmune screen. Following discharge, the patient was referred for specialist review at our institution. The possibility of recurrent myocarditis was considered. Therefore, cardiac magnetic resonance (CMR) imaging was performed (on day 45 after initial presentation to the referring institution) to assess for evidence of myocarditis and myocardial infarction on delayed gadolinium imaging. The CMR cine imaging demonstrated normal left ventricular size and systolic function with no regional wall motion abnormalities. There was evidence of myocardial oedema on T2-weighted short-tau inversion recovery (STIR) imaging with an increased global myocardial to skeletal muscle signal intensity ratio of 2.2 (abnormal if ≥2.0). There was evidence of myocardial hyperaemia with elevated early post-gadolinium contrast enhancement of absolute myocardial signal intensity of 66% (abnormal if ≥45%). Furthermore, there was evidence of regional non-ischaemic myocardial necrosis/fibrosis in the mid to apical inferior wall demonstrated by sub-epicardial delayed gadolinium enhancement imaging (Fig. 1). In this

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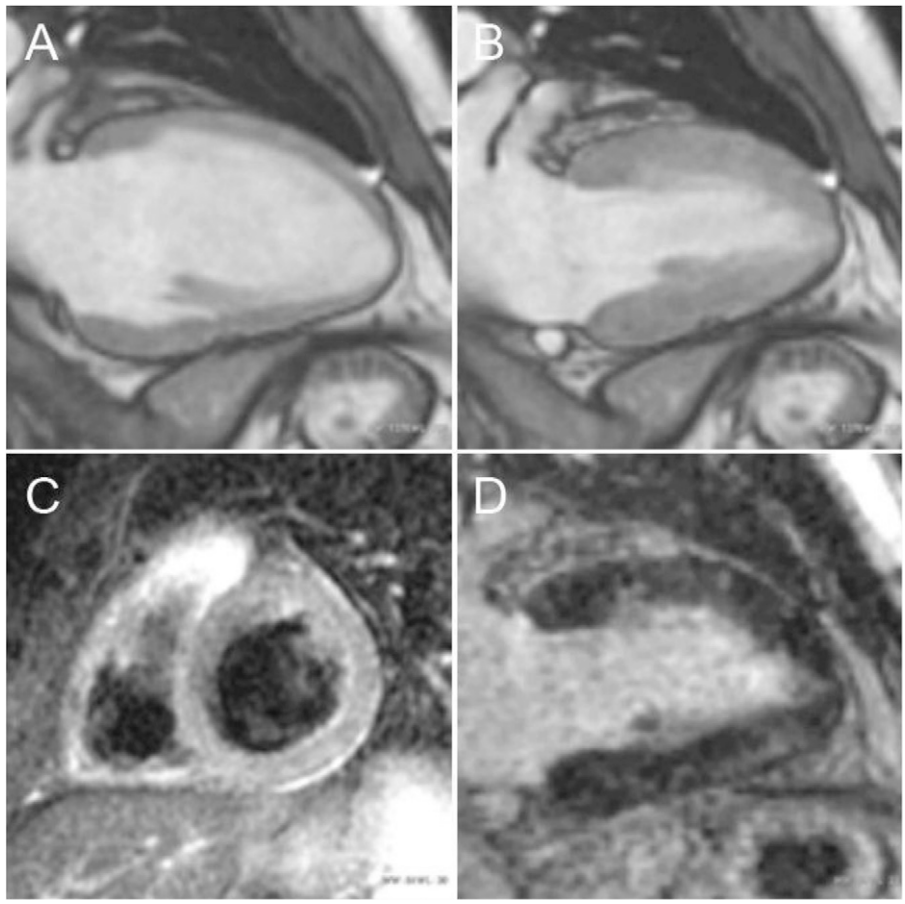

Fig. 1. *CMR images from Case 1 showing functional post-contrast steady state free precession (SSFP) images in the 2 chamber view in diastole (A) and systole (B). The short-axis short-tau inversion recovery (STIR) images showed evidence of myocardial oedema (C) and delayed post-gadolinium images revealed sub-epicardial enhancement in the mid to apical inferior wall (D).*

clinical setting, the CMR findings were consistent with the clinical diagnosis of recurrent myocarditis. The patient was treated conservatively, with ongoing follow up and has had no further episodes in the last 12 months.

Case 2

A 49 year-old male's past medical history included diagnoses of one ST-elevation myocardial infarction and one non-ST elevation myocardial infarction over the last 10 years. On both occasions, he presented with central chest pain associated with a significant troponin rise. On the first occasion, coronary angiography demonstrated normal coronary arteries, with evidence of mild left ventricular anterior wall dysfunction. It was suspected that a thrombotic ischaemic event occurred, with subsequent re-canalisation of the left anterior descending artery. On the second occasion, coronary angiography demonstrated no coronary artery stenoses, with a focal area of hypokinesis noted in the inferior wall of the left ventricle. It was again suspected that there was recent thrombotic occlusion of the right coronary artery, which has cleared. He was treated with anti-platelet agents, a statin and a beta-blocker.

On his most recent presentation, he presented to a rural hospital with a two-day history of central chest pain, fever and cough. ECG did not show any acute changes (Fig. 2). Serum troponin level rose to 18.4 μg/L (normal < 0.03 μg/L). He was transferred to our institution for further investigation. Coronary angiography again demonstrated no obstructive coronary artery disease (Fig. 3). The clinical diagnosis of recurrent myocarditis was suspected. A CMR scan was performed on day 3 following hospital admission, demonstrating a mildly dilated left ventricle with moderate segmental systolic dysfunction, and a left ventricular ejection fraction (LVEF) of 41%. Delayed gadolinium enhancement imaging revealed extensive sub-epicardial and mid-wall necrosis/fibrosis within a non-vascular distribution. There was evidence of myocardial inflammation with myocardial oedema demonstrated on STIR imaging with a global myocardial to skeletal signal intensity ratio of 2.8, as well as evidence of myocardial hyperaemia on early post-gadolinium enhancement imaging, with an increased ratio of myocardial to skeletal muscle signal intensity of 24.7 (abnormal if ≥4.0). These changes were consistent with a diagnosis of myocarditis (Fig. 4). Diagnostic endomyocardial biopsies were performed which demonstrated foci of lymphocytic