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Myocarditis mimicking acute coronary syndrome — the role of cardiac magnetic resonance imaging in the diagnosis

Fateh Ali Tipoo Sultan, Ghufran Adnan

Abstract

Myocarditis is an inflammatory disease of myocardium with a wide range of clinical presentations. Myocarditis may mimic acute coronary syndrome (ACS) and adequate differential diagnosis is not possible by conventional tests. Cardiac magnetic resonance (CMR) has emerged as a leading imaging modality in the diagnosis of myocarditis in such patients. Here, we report three cases of myocarditis mimicking ACS with normal coronary arteries. CMR was used for confirming the diagnosis of myocarditis in all three patients presented here.

Keywords: Myocarditis, Troponin levels, Cardiac magnetic resonance imaging (CMR).

Introduction

Myocarditis is defined as "inflammation of the myocardium." The clinical presentations of myocarditis range from nonspecific symptoms to fulminant heart failure.¹ Myocarditis may resemble an ACS² and should be considered as a differential, especially in younger patients presenting with ACS. Endomyocardial biopsy is the gold standard for the diagnosis of myocarditis.³ However, the invasive character and poor sensitivity limit, the use of endomyocardial biopsy is not very popular. CMR plays an important role in the diagnosis of acute myocarditis and its differentiation from ACS.

After obtaining written consent from patients to utilize their findings, we report three cases seen at Aga Khan University Hospital, Karachi, with myocarditis. The presentation was mimicking ACS and the diagnosis of myocarditis was confirmed on CMR. CMR use is limited in countries like Pakistan, due to lack of awareness on the importance of this imaging modality and also less availability. This is the first case series of myocarditis from Pakistan in which the diagnosis was confirmed non-invasively with the help of CMR.

Case Presentation

Case-1

A 31 years old previously healthy male, presented to the

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Figure-1: Cardiac MRI T2 weighted image showing myocardial oedema.

emergency department on 16th March 2017 with worsening shortness of breath and chest pain. There was history of fever and cough for the last 3-4 days. Electrocardiogram (ECG) revealed complete atrioventricular block. On presentation, he was severely hypoxic and required intubation with ventilatory support. He developed cardiac arrest soon after intubation, but fortunately revived after two minutes of cardiopulmoray resuscitation (CPR). Temporary pace maker (TPM) was inserted for complete A-V block. Transthoracic echocardiogram (TTE) revealed an ejection fraction (EF) of 25-30% with global hypokinesia. Troponin I levels were elevated. Coronary angiography was done which revealed normal coronary arteries. He was managed with anti-platelets, anti-failure medications and antibiotics. CMR was performed which confirmed the diagnosis of myocarditis by showing evidence of myocardial oedema on T2 weighted images (Figure-1). His further hospital course was uneventful with gradual recovery. Repeat TTE after couple of weeks showed significant improvement in left ventricular systolic function.

Case-2

A25 years old previously healthy female, presented to the

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Figure-2: Cardiac MRI late gadolinium image showing subepicardial hyperenhancement.

emergency department on 6th February 2015 with acute chest discomfort. Clinical examination was unremarkable and ECG showed non-specific ST-T changes. First troponin I was 5.7 which was increased to 27, on repeating after couple of hours. TTE revealed normal left ventricular systolic function with no wall motion abnormality. In view of acute symptoms and positive troponin I, cardiac computed tomographic angiography (CTA) was performed which showed normal coronary arteries. CMR was done which confirmed the presence of myocarditis (Figure-2). Her further course was uneventful.

Case-3

A young male of 24 years age with no prior history of any significant illness, presented to the emergency



Figure-3: ECG showing ST elevation in inferolateral leads.



Figure-4: Cardiac MRI late gadolinium showing patchy subepicardial hyperenhancement.

department on 10th February 2015 with acute chest pain. ECG revealed ST elevation in infero-lateral leads (Figure-3). Troponin I was 14.08. In view of chest pain, ECG changes and positive troponin, invasive coronary angiography was performed, which revealed normal coronary arteries. CMR was done which confirmed the diagnosis of myocarditis (Figure-4). His further course was free of significant issues.

Discussion

Myocarditis may mimic ACS. Establishing a correct diagnosis is important for such patients, both from prognostic and therapeutic point of view. Conventional

tests lack enough specificity for adequate differential diagnosis.

ECG changes are neither specific nor sensitive for diagnosing myocarditis. They can vary from non-specific ST-T changes to even ST segment elevation. Two of our patients showed non-specific ST-T changes while one patient had ST elevation at the time of presentation.

Serum markers of myocardial damage may or may not be elevated in patients with myocarditis, depending on the extent of damage. All three patients presented here had positive troponins, suggesting myocardial necrosis. In the literature, around 10% of the patients, initially

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diagnosed as ACS show normal coronary arteries on angiography.⁴

The role of echocardiography is also limited as the findings are not specific for myocarditis and segmental wall motion abnormalities similar to ACS may be seen.⁵

Endomyocardial biopsy is considered the gold standard for the diagnosis of myocarditis.⁶ However, the biopsy has low sensitivity due to focal nature of the disease and being an invasive procedure it carries potential risk of complications. Due to these limitations, we did not consider the endomyocardial biopsy in our patients

CMR is especially useful in such settings.⁷ A number of studies,^{5,8,9} have demonstrated the ability of CMR to differentiate ACS from acute myocarditis. T2 weighted images can be used to detect the presence of myocardial oedema both in inflammatory and ischaemic diseases. In the case of ACS, oedema is localized to the culprit vessel territory, while in myocarditis, it is either diffuse or segmental.¹⁰

There is strong evidence in the literature that contrast CMR with late gadolinium images, is a good diagnostic modality to detect myocardial necrosis and fibrosis both in ischaemic heart disease and in other types of non-ischaemic lesions.⁷ In myocarditis, the late gadolinium enhancement (LGE) is patchy or multifocal in a sub-epicardial or intra-myocardial distribution, usually involving the lateral wall. In patients with ischaemic heart disease, LGE is either sub-endocardial or transmural in a coronary artery distribution.

Due to these features, CMR is helpful in differentiating myocarditis from ACS in patients with acute chest pain, as was the case in our patients.

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