

Case Report

ACS like presentation with normal coronaries: Are they all MINOCA? Utility of cardiac MRI



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ABSTRACT

Normal or unobstructed coronaries in the setting of chest pain and troponin positivity is not uncommon. A definite diagnosis may prove elusive and long term management of such patients poses a difficult question. Cardiac Magnetic resonance (CMR) imaging may prove to be useful in such a situation by not only providing a definite diagnosis but also by excluding other possible etiologies.

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Introduction

Myocarditis is known to mimic the presentation of ACS. The long term management and prognosis is quite different for both etiologies. A definitive diagnosis goes a long way in reassuring and educating the patient regarding treatment options and duration. We herein report a case of suspected myocardial infarction with normal coronary arteries (MINOCA) wherein CMR played a crucial role in clinching the diagnosis of myocarditis.

Case report

30 year-old male patient presented with history of acute onset chest pain. He was not a known diabetic or hypertensive and is a non smoker. Clinical examination was unremarkable and he was hemodynamically stable. 12 lead ECG showed inferior and lateral wall ST-T changes (J point elevation) and 2D echocardiography showed inferolateral regional wall motion abnormalities with mild left ventricular systolic dysfunction (Fig. 1). Serial cardiac biomarkers were positive. He was diagnosed to have acute inferolateral wall myocardial infarction and was subjected for coronary angiogram

which showed normal coronary arteries. The provisional diagnosis was acute coronary syndrome with recanalised coronary artery (Fig. 2). However in view of the patient's age and absence of traditional coronary risk factors, cardiac MRI was done to rule out a nonischemic cause. Cine MRI showed global left ventricular hypokinesia with mild LV systolic dysfunction- LVEF of 43%. There was late gadolinium enhancement of subepicardial myocardial region globally with patchy areas of non-involvement. (Fig. 3 Figs. 3 and 4) Trace fluid was seen in the pericardium. All of the above features favoured a radiological diagnosis of myocarditis. He was managed with beta blockers and ACE inhibitors with which he clinically improved. He was asymptomatic and hemodynamically stable at discharge. At one month follow-up, cardiac MRI was done and it showed significant improvement in left ventricular wall motion and reduction in myocardial wall enhancement (Fig. 5)

Discussion

Myocardial infarction with non obstructive coronary arteries (MINOCA) is seen in 7–15% of patients undergoing coronary angiogram for ACS (7–10% with STEMI and 10–15% with NSTEMI).^{1–2} Patients with MINOCA are more likely to be younger and female and less likely to have hyperlipidemia, other cardiovascular risk factors being similar.² The various possibilities being a recanalised vessel, embolic phenomenon, coronary vasospasm, endothelial dysfunction, coronary artery dissection or other etiologies like myocarditis, takotsubo cardiomyopathy and acute

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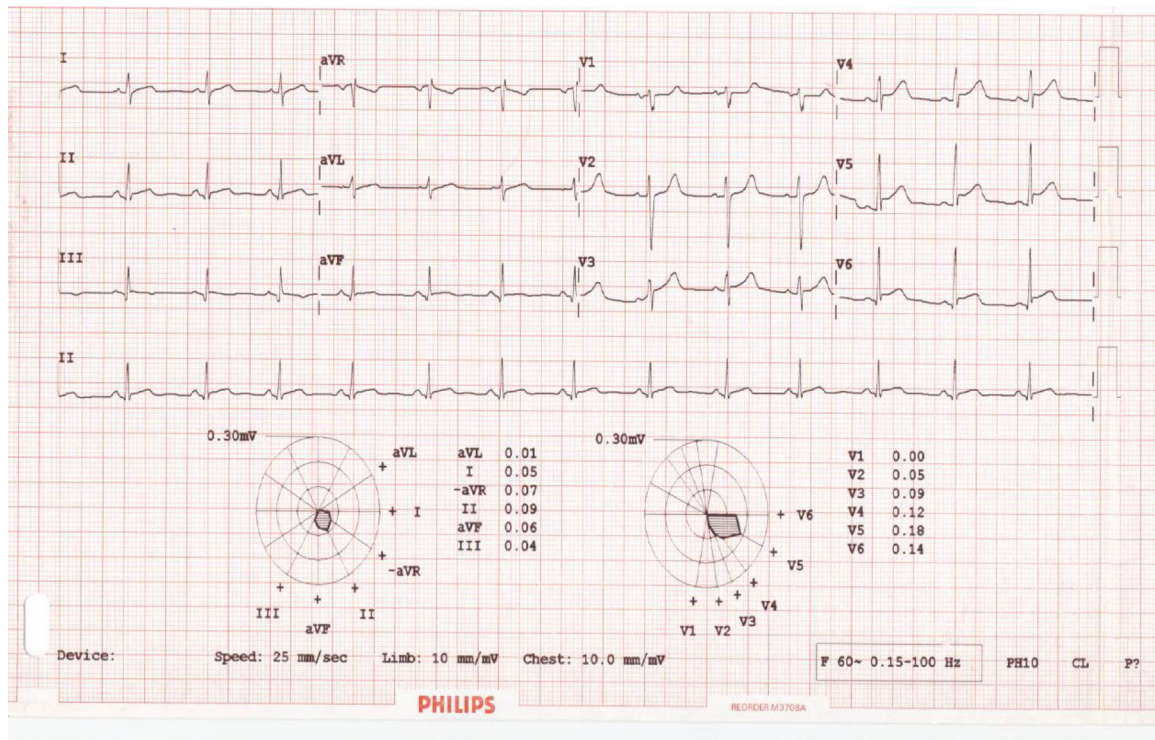


Fig. 1. 12 lead electrocardiogram at presentation.



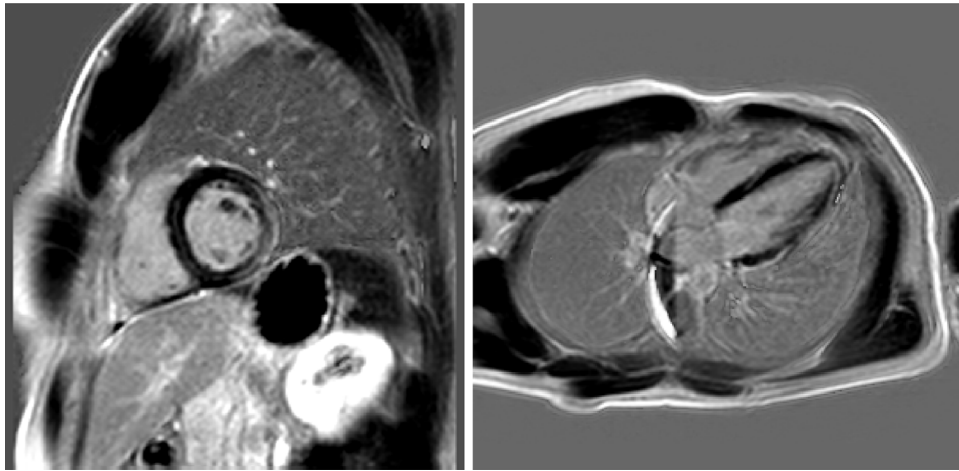
Fig. 2. Left and right coronary artery images.

pulmonary embolism. A systematic review by Pasupathy et al showed an overall 12 month mortality of 4.7% in patients with MINOCA, which is similar to patients with myocardial infarction with a single/double vessel disease.²

Diagnostic imaging modalities that could be considered in patients with MINOCA include fractional flow reserve for suspected endothelial dysfunction, provocative testing for vasospastic angina, intravascular ultrasound or optical coherence tomography (to exclude obstructive lesions, dissections, plaque rupture), MDCT for aortic dissection/pulmonary embolism and CMR imaging. CMR being radiation free, non invasive and with its ability to evaluate multiple etiologies in a single session provides a wealth of information within a reasonable total scan duration time of approximately 45 minutes. Myocarditis is diagnosed by CMR using Lake Louise criteria,³ when CMR is considered indicative of active myocardial inflammation if two out of the three criteria demonstrating myocardial edema, hyperemia or fibrosis are positive.

CMR is the only imaging modality for detection of myocardial edema/inflammation and this helps in distinguishing acute from chronic myocarditis. The sensitivity of detection of edema can be improved if T2-weighted imaging is done early (<2 weeks) in the course of illness as edema has been shown to be reversible and to resolve with time. LGE commonly reveals 2 patterns of myocardial necrosis – an intramural, rim-like pattern in the septal wall or a sub-epicardial patchy distribution in the free lateral wall of left ventricle. Subepicardial predominance of LGE is a highly specific for myocarditis. Myocardial damage in myocarditis is usually patchy, non contiguous but can also be diffuse not confined to any coronary artery territory. Conversely, myocardial involvement in myocardial infarction is subendocardial and can be transmural.

In patients with ACS with unobstructed CAD, a definitive etiology could be arrived at in more than 2/3 rd of patients using CMR. The most common underlying etiology was found to be myocarditis ranging from 25–50% in most of the studies.^{2,4,5} In a



Figs. 3 and 4. Short axis and long axis delayed enhancement imaging showing subepicardial involvement globally with patchy areas of non involvement.

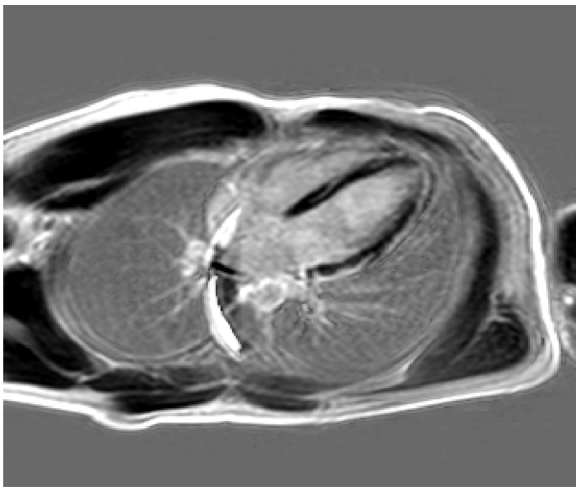


Fig. 5. Repeat CMR after one month showing partial resolution of inflammation by reduction in areas with myocardial enhancement.

systematic review of patients with suspected myocardial infarction and normal coronaries (MINOCA), CMR identified the underlying pathophysiology as myocarditis in 33%, MI in 24% and no significant abnormality in 26%.² In a study of 61 patients, CMR was able to provide a diagnosis in 65% of the cases, the commonest being myocarditis (50%), followed by myocardial infarction (11.6%) and

cardiomyopathy (3.4%).⁴ In another study of 27 patients with ACS with normal coronaries, LGE typical of myocarditis was seen in 44% and subendocardial or transmural enhancement suggestive of MI was evident in the another 44%.⁵

Our case mimicked the presentation of acute myocardial infarction with ECG changes and troponin positivity but showed normal coronaries. CMR revealed global wall motion involvement and subepicardial, patchy and non contiguous LGE suggestive of myocarditis. Without a CMR, he would have been labelled as ACS with recanalised coronaries and might have been put on indefinite antiplatelet and anti ischaemic therapy warranting long term follow up. Extending its indication to patients with suspected ACS and normal coronaries, CMR would provide a definite diagnosis and also help in prognostication and long term management.

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