Acute myocarditis simulating ST-elevation myocardial infarction: A case report

Myocardite aiguë simulant un infarctus du myocarde avec élévation du segment ST: A propos d'un cas

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Résumé

Introduction : Le diagnostic de myocardite aiguë est difficile surtout quand la présentation clinique mime un syndrome coronarien aigu avec élévation du segment ST.

Observation : Nous rapportons l'observation d'un patient âgé de 37 ans admis pour des douleurs thoraciques avec à l'électrocardiogramme des modifications électriques compatibles avec un syndrome coronarien aigu avec sus décalage persistant du segment ST en inféro latéral. Une coronarographie a été réalisée en urgence montrant un réseau coronaire angiographiquement sain. L'imagerie par résonance magnétique (IRM) a permis par la suite d'affirmer le diagnostic de myocardite.

Conclusion : Cette observation confirme le rôle primordial de l'IRM cardiaque chez les patients présentant une suspicion clinique de myocardite aiguë ou un tableau de syndrome coronarien aigu à coronaires saines

Mots-clés

Syndrome coronarien aigu avec élévation du segment ST, Myocardite, IRM cardiaque

Summary

Background: The diagnosis of acute myocarditis is difficult especially when the clinical presentation is an ST elevation myocardial infarction (STEMI).

Case report: We report a case of focal myocarditis in a 37-year-old man mimicking acute STEMI. He presented with chest pain and the electrocardiogram changes were consistent with infero-lateral STEMI. Coronary angiogram revealed smooth arteries with no obstruction. Subsequently performed, cardiac magnetic resonance imaging (CMR) confirmed the diagnosis of myocarditis.

Conclusion: This case exhibits the use of cardiac magnetic resonance imaging for diagnosis in such scenarios as often if the angiogram is normal.

Keywords

ST elevation myocardial infarction, myocarditis, cardiac magnetic resonance

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INTRODUCTION

Myocarditis is an inflammatory disease of the cardiac muscle mainly caused by viral infection. The diagnosis of acute myocarditis is one of the most challenging issues in cardiology due to the non-specific pattern of clinical presentation. It can present with various clinical manifestations ranging from mild disease with poor symptoms to heart failure and death. Myocarditis may mimic myocardial infarction (MI), since patients usually present with chest pain, and the electrocardiographic changes similar to those observed in acute ST elevation myocardial infarction (STEMI) (1,2).
Cardiac magnetic resonance (CMR) is currently the non-invasive reference standard to assess myocardial injury in patients with clinically-suspected myocarditis (3,4).
We report a case of focal myocarditis in a young man mimicking acute ST-segment elevation MI.

OBSERVATION

A 37 years old man, smoker, with no relevant medical history presented to the emergency room with 2-hour history of acute chest pain and breath shortness. There was no current or past use of cocaine, alcohol or other stimulants.
Physical examination was notable for regular heart rhythm without murmurs, gallops, or rubs. He was afebrile with a heart rate of 75 bpm, respiratory rate of 12, blood pressure of 130/80 mmHg, and oxygen saturation was 99% on room air. He had no peripheral edema or jugular venous distention. His lungs were clear to auscultation.
An electrocardiogram was performed and showed ST-segment elevation (STE) in leads II , III , aVF associated to an ST depression in aVL. [Figure 1].

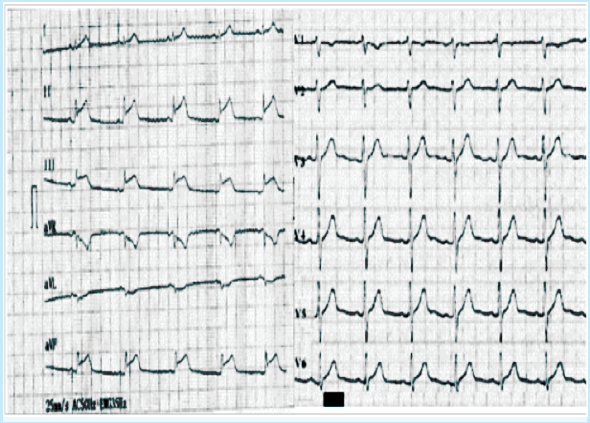

Figure1: twelve lead electrocardiogram showing ST segment elevation in leads II,III and aVF associated to an ST depression in aVL.

Patient was immediately transferred to the cath lab and underwent coronary angiography, which showed smooth unobstructive epicardial coronary arteries [Figure 2].
A Series of investigations were subsequently performed. Cardiac enzyme tests showed significantly elevated troponin I (Tn I) level .Trans-thoracic echocardiography was normal: the left ventricle was not dilated and the ejection fraction was estimated at 60%. No evidence of regional wall motion abnormality was detected
A new interrogation of the patient revealed a 5-day history of acute upper respiratory infection symptoms including fever, cough, sore throat and nausea.
CMR was performed next day. It showed high signal areas on STIR sequences as well as early and late gadolinium enhancement in mid and epicardial regions of left

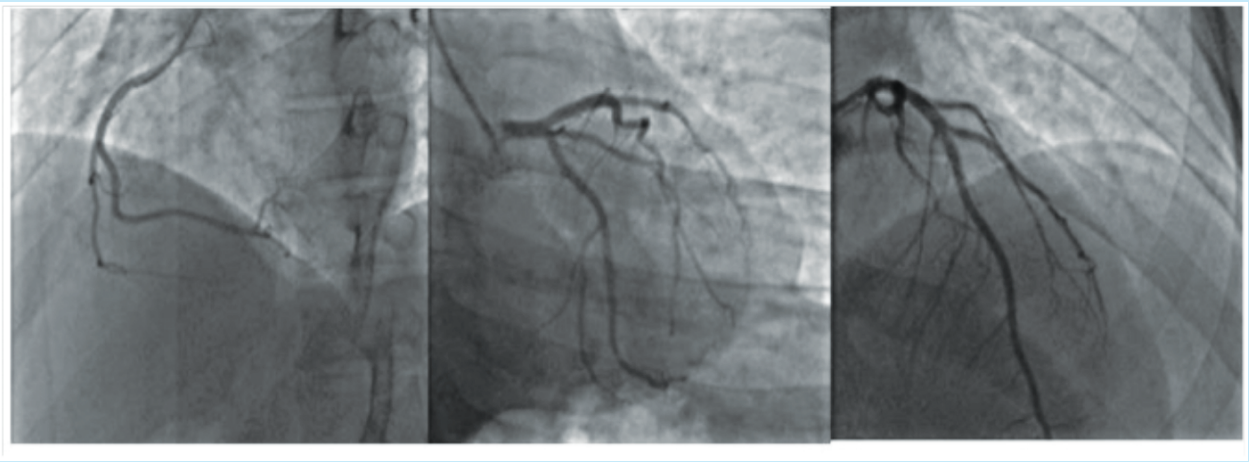

Figure2: Coronary angiography revealing normal epicardial coronary arteries

ventricle inferior and lateral walls. Thus, Lake Louise criteria were all found (Figure 3).
Patient has uneventful recovery and was discharged from hospital 48 hours later. He remained asymptomatic at 3-months' follow-up with normalization of ST segments changes.

DISCUSSION

Myocarditis is associated with a broad spectrum of clinical and electrocardiographic manifestations, ranging from completely asymptomatic courses to signs of myocardial infarction or cardiogenic shock. The clinical presentation of myocarditis may mimic acute myocardial infarction (2)
In the present case, history and clinical presentation (young age, low coronary risk profile, concomitant flu-like symptoms in the few days before admission) were consistent with myocarditis. But the localized ST-segment elevation along with the "reciprocal-like" changes and worsening chest pain raised the possibility of an acute myocardial infarction. Then, the patient underwent coronary angiography.
Several studies have previously shown that a subset of acute myocardtis patients may have an infarct-like presentation, with chest pain, elevated Tn levels, and ECG findings suggestive of STEMI [5, 6]. This pattern of clinical presentation has been related to parvovirus B19 infection of the endothelial cells of myocardial vessels, causing endothelial dysfunction and coronary vasospasm, migration of inflammatory cells into the myocardial interstitium, and subsequent myocyte damage [7, 8].
Endomyocardial biopsy (EMB) used to be the "gold standard" to make the diagnosis of myocarditis [9]. Nowadays, it is seldom performed because of its invasive nature, high rate of sampling error, and variability in diagnostic criteria and interpretation. It has been replaced by CMR imaging which is a helpful tool for the diagnosis of myocarditis.
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

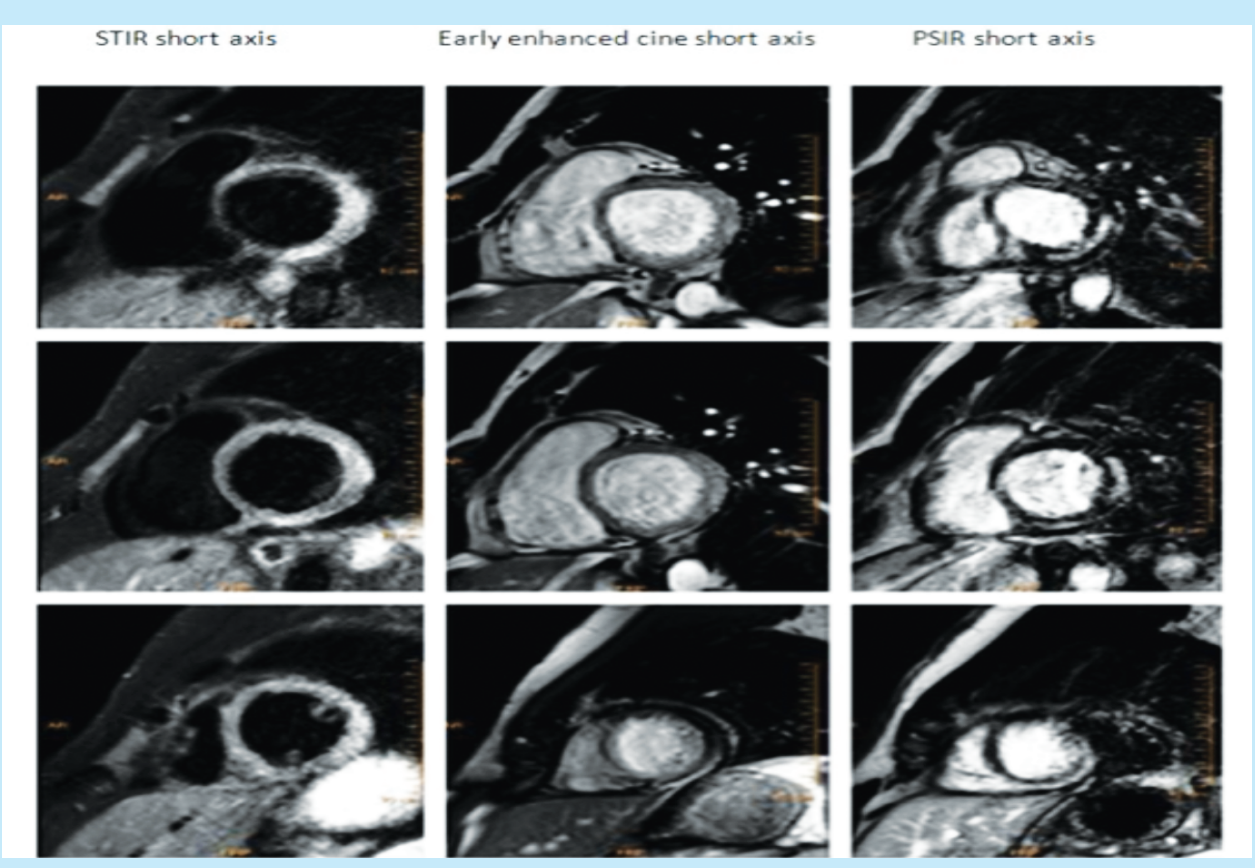

Figure 3: STIR (Short Tau Inversion-Recuperation) sequence revealed high signal areas (arrows) in inferior and lateral left ventricule wall that showed a linear mid and sub-epicardial early (dashed arrows) and late (thick arrows) gadolinium enhancement

either in ischemic or non-ischemic heart diseases (4, 10). The zonal distribution of late gadolinium enhancement enables confident distinction of infarct from inflammatory related enhancement: subendocardial extending transmurally in a defined coronary artery territory in the former and mid-epi myocardial distribution in the latter.

An international Consensus Group established the proposed CMR criteria for diagnosis of acute myocarditis (Lake Louise Criteria) in 2009, consisting of 3 tissue markers: irreversible tissue injury on LGE images, edema on T2 STIR images and hyperemia on early enhanced (EE) images (11). The presence of 2 of the 3 CMR characteristics described in the Lake Louise criteria results in a sensitivity of 67%, specificity of 91%, and negative predictive value of 69% for the diagnosis of biopsy-proven myocarditis (12).

In our case all Lacke Louise criteria were found.

The correlation between ECG findings and LGE in CMR is limited. Deluigi et al (13) and Di Bella et al (14) evaluated the relation between the site of repolarization abnormalities and location of myocardial injury assessed by cardiac MRI in patients with acute myocarditis (including 20 and 46 patients with infarct-like myocarditis, respectively); both groups found a weak correlation between ECG leads showing repolarization abnormalities and location of LGE.

In addition, Nucifora et al (15), concluded that the site of STE is not a perfect predictor of the region of myocardial injury among both groups of patients presenting with anterolateral or inferolateral STE; topographic agreement between the site of LGE and the site of STE was only 59% and 46%, respectively.

CONCLUSION

Despite considerable progress, it remains a daunting challenge for physicians to discriminate between acute myocarditis and myocardial infarction, particularly in the early phase. An integrated assessment and evaluation of evidence, including medical history, clinical presentation and results of other auxiliary tests, are necessary for the accurate diagnosis of myocarditis and can guide treatment accordingly.

Our case exhibits the use of cardiac magnetic resonance imaging for diagnosis in such scenarios especially when the angiogram is normal.

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