

A MISTAKEN CASE OF NON-Q WAVE ACUTE INFARCTION

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SUMMARY

Over the last few years, enormous progress has been made in Cardiovascular imaging. Myocarditis is today an underdiagnosed cause of acute sudden death, heart failure, and chronic dilated cardiomyopathy. Its diagnosis is frequently missed or delayed and it can simulate an acute coronary syndrome because of its clinical presentation and elevation of cardiac enzymes. Cardiac Magnetic Resonance has become the leading modality for its noninvasive diagnosis, and it often replaces endomyocardial biopsy in clinical practise.

Introduction

Myocarditis is clinically and pathologically defined as "inflammation of the myocardium". Despite its rather clear-cut definition, the diagnosis, classification, and treatment of myocarditis continues to be of considerable debate [1].

Patients may present with mild symptoms of chest pain, fever, sweats, chills, and dyspnea. In viral myocarditis, patients may present with a history of a recent (within 1-2 week) flulike syndrome of fevers, tonsillitis, arthralgias, and malaise or pharyngitis or upper respiratory tract infection. Adults may also present with only a few symptoms, rather than the acute toxic state of cardiogenic shock or frank heart failure (fulminant myocarditis) that can be often associated with myocarditis [2].

Myocarditis is caused by a variety of bacterial and viral infections but exposure to drug treatment, physical stimuli such as radiation, immune and metabolic disorders and pregnancy may also cause myocarditis. In the case of idiopathic myocarditis, the etiology is yet to be determined.

Diagnosing myocarditis is a challenging process because of its diverse presentation. Other complicating factors are the limited sensitivity of endomyocardial biopsies (EMB) and overlapping symptoms. Furthermore, the criteria on which the diagnosis is based are not well defined. However, early diagnosis is essential in order to achieve specific aetiology-directed therapeutic management of the myocarditis which has a direct effect on patient morbidity and mortality. Currently, EMB is the only way of confirming the presence of a viral genome and other histopathological findings which allow for appropriate treatment to be implemented in cases of myocarditis.

However, increased recognition of the role of myocardial inflammatory changes has

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given rise to interest in noninvasive imaging as a diagnostic tool, especially cardiovascular magnetic resonance imaging (CMR) [3]. CMR has become the leading modality in noninvasive imaging of myocarditis. Consensus on the use of 3 CMR criteria for myocarditis, referred to as edema, early and late enhancement, has standardized CMR protocol for assessing myocarditis. Confirming a diagnosis of myocarditis remains challenging, the diagnosis of this disease needs further investigation with the aim of providing robust noninvasive tests [4].

Case Presentation

A 45 year-old man was referred to the Emergency Department of another Hospital with chest and epigastric pain. In anamnesis, the patient reported no history of either smoking or hypertension.

On admission the patient presented these hematologic values: Troponin-I max level: 779 pg/ml, pro- BNP: 544 pg/ml, C-reactive protein: 13.40 mg/dL, TGO: 54 U/L, TGP: 56 U/L.

Admission and discharge ECG presented: sinus rhythm, rate 75 bpm, no ischemia signs, non-specific abnormalities of the ST-T wave segment in inferior derivations.

The patient had been admitted with a diagnosis of "non-q wave acute infarction".

Imaging Findings

Transthoracic echocardiogram showed: ejection fraction: 60%, mild mitral insufficiency, hypokinesia of inferior-basal wall. Chest X-ray was unremarkable. Coronary angiography showed: normal coronary arteries (right coronary artery, left anterior descending coronary artery and left circumflex coronary artery).

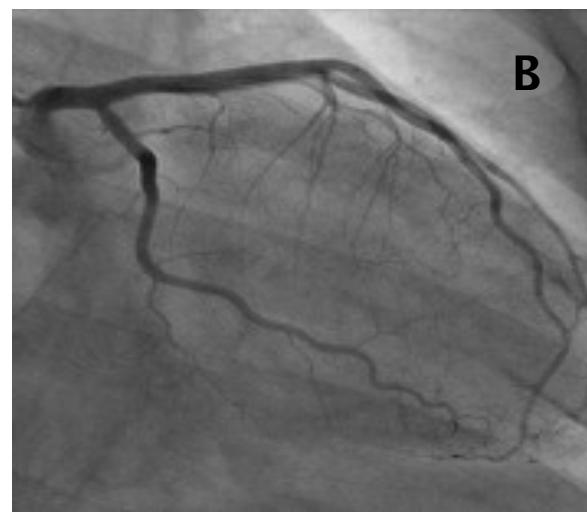
(Figure 1A-1B-1C)

Cardiovascular magnetic resonance (CMR) imaging, performed in our Department 10 days after patient discharge, showed signs of focal acute myocarditis: T2-weighted short time inversion recovery (STIR) showed a homogeneous subepicardial hyperintense area in the mid antero-left ventricle wall indicating edema; the same area of hyperintensity was seen on contrast-enhanced CINE MRI using SSFP sequences; strong enhancement was seen in subepicardial layers of the mid anterior wall on CE-IR MRI.

(Figure 2A-2B-2C-2D)



A



B



C

Figure 1A-1B-1C: Right coronary artery (A), Left circumflex coronary artery (B), Left anterior descending coronary artery (C): Normal coronary angiogram.

Discussion

Myocarditis is today an underdiagnosed cause of acute heart failure, chronic dilated cardiomyopathy and sudden death. The short-term prognosis for acute myocarditis is usually good; however, occasionally after some years, patients can develop recurrent dilated cardiomyopathy and heart failure. Myocarditis presents with non-specific symptoms including chest pain, dyspnoea and palpitations and

it often mimics more common disorders such as coronary artery disease [5]. CMR is today considered the gold standard to assess left and right ventricular mass and systolic function as well as to depict myocardial infarction scars. CMR images are most often acquired during a breath-hold of 8–20 s triggered by an ECG signal but CMR study protocol is not uniform and may vary depending on

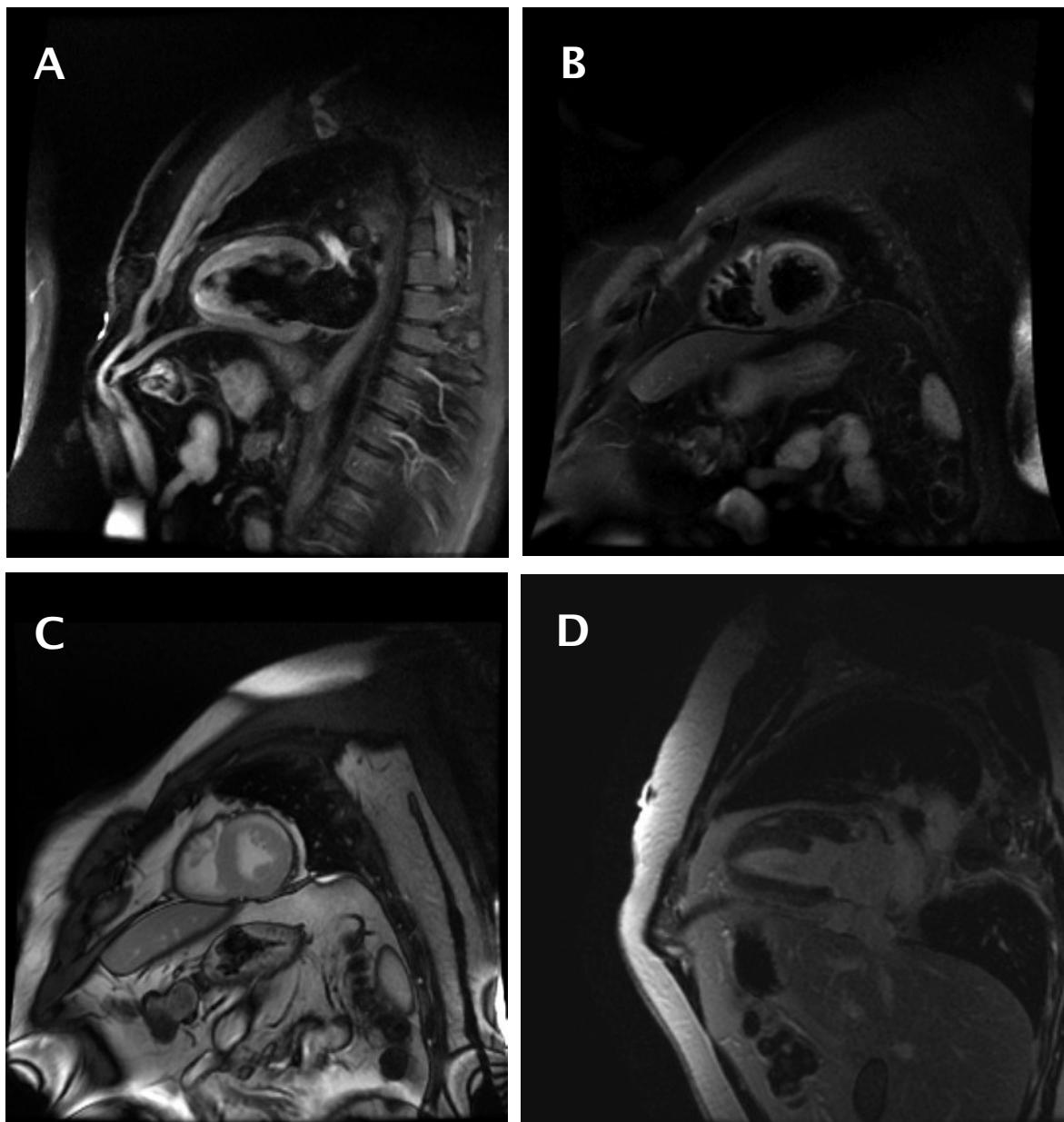


Figure 2A-2B-2C-2D: Focal acute myocarditis. T2-weighted short-tau inversion-recovery (STIR) on vertical long axis of the left ventricle (A) and short axis of the mid-left ventricle (B). Cine MRI, using the post contrast b-SSFP technique (C) and CE-IR MRI with late imaging on vertical long axis (D). T2-w STIR images show homogeneous subepicardially hyperintense area in the mid antero-Left Ventricle wall (A,B) expression of edema; the same area of hyperintensity is seen on the SSFP images (C). Strong enhancement is seen in the subepicardial layers of the mid anterior wall (D).

local availability and expertise [6]. A scientific statement by the AHA/ACC/ESC suggested in 2007 that among 14 different clinical scenarios myocardial biopsy appears appropriate in only two of them; firstly in “unexplained new-onset heart failure with hemodynamic compromise within 2 weeks” and secondly in “unexplained new-onset heart failure for 2 weeks to 3 months with dilated left ventricle and new ventricular arrhythmias or second or third-degree atrioventricular block or failure to respond to usual care within 1 to 2 weeks” [7]. The case herein described is peculiar. Indeed the patient simulated an acute infarction because of chest pain and a high level of troponin-I. We diagnosed myocarditis about ten days after discharge with the help of cardiac magnetic resonance and without endomyocardial biopsy. According to ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation, the cardiac and non-cardiac conditions that can mimic non-ST-elevation acute coronary syndromes are: pericarditis, myocarditis, valvular disease, tako-tsubo cardiomyopathy, pulmonary embolism and pulmonary infarction, pneumothorax, haematological vascular aortic dissection, aortic aneurysm, cerebrovascular disease, gastro-intestinal/oesophageal spasm, oesophagitis, peptic ulcer and pancreatitis cholecystitis, muscle injury/ inflammation, costochondritis herpes zoster etc [8]. Clinically, biopsy does not appear to be helpful in cases where CMR has confirmed a ‘typical’ pattern of myocarditis or excluded any myocardial abnormality. Therefore, CMR may offer therapeutic guidance for those patients with an acute cardiovascular event, but with normal coronary arteries [9-10-11].

Cardiovascular magnetic resonance imaging is widely established, free of radioactive material or ionizing radiation, and is considered the accepted noninvasive gold standard for numerous noninvasive cardiac markers [12].

CMR can be used to assess myocardial edema as a reliable marker for acute, potentially reversible myocardial injury. The detection of myocardial edema is clinically relevant in many acute diseases and may be further helpful to better understand the pathophysiology of many non-acute clinical diseases [12].

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