Case Report

Acute Myocarditis from Coxsackie Infection, Mimicking Subendocardial Ischaemia

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Viral myocarditis may have various clinical presentations, sometimes mimicking acute myocardial infarction or ischaemia. We describe the case of a young man presenting with acute heart failure, who had electrocardiographic changes suggesting myocardial ischaemia and an episode of sustained ventricular tachycardia. The diagnosis of acute myocarditis was confirmed using cardiac magnetic resonance imaging.

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Viral myocarditis may have various clinical presentations, sometimes mimicking acute myocardial infarction or ischaemia. The diagnosis of acute myocarditis may be based on clinical presentation, physical examination, laboratory testing, the course of electrocardiographic changes, and the lack of epicardial coronary disease revealed by cardiac angiography. Here we describe the case of a young man presenting with acute heart failure, who had electrocardiographic changes suggesting myocardial ischaemia and an episode of sustained ventricular tachycardia. The diagnosis of acute myocarditis was confirmed using cardiac magnetic resonance imaging (CMR).

Case presentation

This is the case of previously asymptomatic 34-year-old man who was referred to us from another hospital, where he had been admitted three days previously for new-onset chest pain, dyspnoea and fatigue. The patient's previous medical history showed no cardiovascular risk factors or exposure to any drug. One week before this initial hospitalisation, he was diagnosed by a community physician as having a gastrointestinal infection with fever and vomiting. During his initial hospitalisation the patient presented acute renal failure and ischaemic hepatitis. Laboratory examinations showed renal failure (serum urea 209 mg/dl, serum creatinine 1.8 mg/dL, and hyperkalaemia at 5.7 mmol/L), an elevation of liver enzymes (ASAT/ALAT 22311/965 UI/L, normal value 10-40 UI/L), an increase of serum lactate dehydrogenase (LDH: 763 UI/L, normal value 135-220 UI/L), creatine phosphokinase (CPK: 406 UI/L), and troponin I (0.12 ng/ml, normal value <0.04 ng/L). The ECG in the emergency department was described as having ST-segment depression in the inferior leads with reciprocal ST-segment depression in V_1-V_2, suggesting inferoposterior or submural ischaemia, while the echocardiographic assessment revealed a left ventricular ejection fraction around 30%.

On his admission to our department the patient was stable, with normal vital signs, while the laboratory findings showed a rapid improvement. Chest X-ray revealed a slightly enlarged cardiothoracic index with a small right pleural effusion. The ECG showed sinus rhythm with negative T waves in the inferolateral leads (Figure 1). Echocardiographic evaluation revealed normal cardiac chamber dimensions, with mild hy-

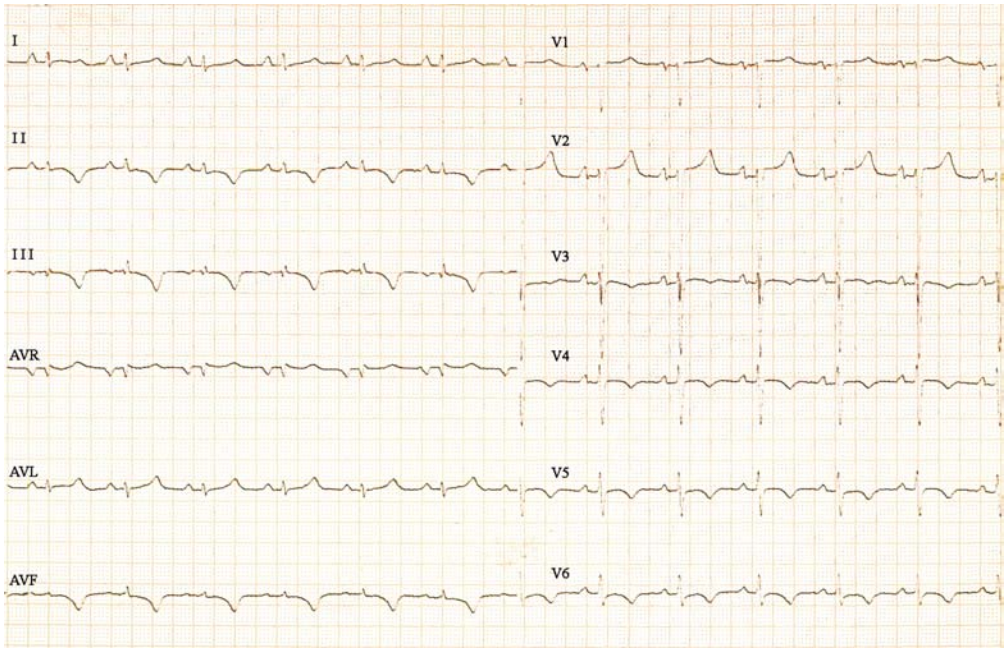


Figure 1. The ECG on admission to our department reveals sinus rhythm with inverse, symmetric T waves in leads II, III, aVF, V_3-V_6.

pokinesis of the inferolateral segments, and an improved left ventricular ejection fraction of 40%, compared to the previous evaluation. During the first hours of his admission he presented an episode of ventricular tachycardia, which was restored to sinus rhythm using electrical cardioversion. After this event the patient was started on amiodarone at 1100 mg for the first 24 hours, then 600 mg daily for the next seven days. The remaining medication included oral metoprolol, magnesium, furosemide and ramipril.

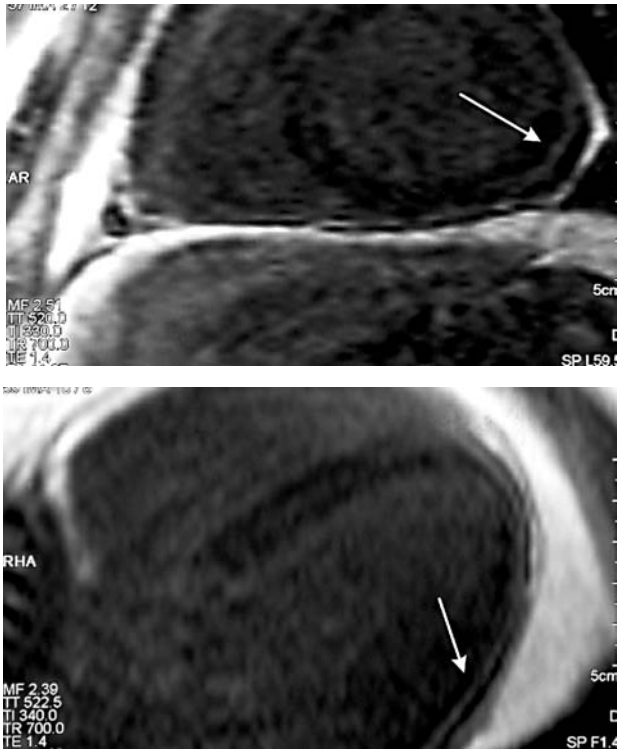
Cardiac catheterisation was performed four days later and demonstrated normal coronary arteries. No coronary vasospasm was shown and vasodilator drugs were not used during the procedure. The serological tests for connective tissue disease were found to be normal. Serum antibodies for cytomegalovirus, IgG, IgM and adenovirus were also negative, whereas positive results were found for EBNA-G (600 u/ml), VCA EBV-G (229 u/ml), HSV 1.2 G (14.4 index) and toxo G2 (18.7 IU/ml), indicative of past exposure. The remaining immunological evaluation of blood species, using ELISA, revealed recent Coxsackie infection, type B.

The patient was discharged on amiodarone, angiotensin-1 converting enzyme inhibitor, and b-blocker, in good condition. Two months later, the patient remained asymptomatic and all medication was discontinued.

The patient underwent CMR one month after his hospital discharge. CMR showed hypokinesis of the inferolateral wall and a corresponding area of myocardial delayed enhancement with sparing of the sub-endocardial tissue. Contrast-enhanced CMR included black-blood T2-weighted (BBT2w) images, without and with fat saturation, and delayed three-dimensional T1 turbo field-echo inversion-recovery sequences obtained 15 min after gadolinium injection (Figures 2, 3).

Discussion

The clinical presentation of patients with myocarditis includes fatigue, signs of congestive heart failure or arrhythmia and sudden death. Pericardial infusion often causes chest discomfort, while diffuse electrocardiographic changes can mimic an acute coronary syndrome.[1] In patients with chest pain, elevated myocardial enzymes and absence of coronary artery disease, myocarditis is the leading diagnosis. The only available method for a definite diagnosis of myocarditis is endomyocardial biopsy, which shows a sensitivity from 43% to 64%, an overall complication rate of 6%, and a 0.4% incidence of death due to perforation.[2] However, endomyocardial biopsy is not indicated in the routine evaluation of cardiomyopathy, except when there is a strong reason to believe that the results will have a meaningful effect on subsequent therapeutic decisions or prognosis, and then only by op-



Figures 2, 3. Contrast-enhanced cardiac magnetic resonance identified areas of myocardial inflammation (arrows) due to focal myocarditis in the inferolateral wall.

erators experienced in its performance.[3] Radiolabeled antimyosin antibody has been used to identify myocarditis, but the method is non-specific as it detects myocardial necrosis from any cause. Furthermore, in one report the sensitivity of this technique in the differentiation of myocarditis from acute myocardial infarction was found to be 67% and the specificity 63%.[4]

CMR can detect myocardial oedema and myocyte damage non-invasively. CMR in myocarditis shows a characteristic pattern of contrast enhancement, which originates primarily from the epicardium, sparing the sub-endocardial layer.[5-8] In contrast, myocardial infarction typically shows sub-endocardial enhancement. In our case CMR showed the classical features of myocarditis, with mid-myocardial enhancement of the inferior-posterior wall, which spared the sub-endocardium and corresponded to the ECG changes and the wall-motion abnormalities detected by echocardiography. Furthermore, the location of enhancement within the wall and throughout the ventricles seems to be fairly specific for myocarditis. The presence of sub-epicardial and mid-wall enhancement is highly suggestive of myocarditis in cases where there is clinical suspicion of such a condition, and always enables the exclusion of ischaemia-related myocardial damage, since this disease starts in the sub-endocardium and spreads like a wave front in the transmural direction. In addition, the lateral wall, and less frequently the basal ventricular septum, are most commonly involved.[7,8] In a study by Abdel-Aty and co-workers[9] the sensitivity, specificity and accuracy of delayed contrast enhancement for the detection of acute myocarditis were 44%, 100% and 71%, respectively, while in a study by Mahrholdt et al[5] 88% of patients showed contrast enhancement. This discrepancy in the results can be explained by the fact that in borderline myocarditis myocyte injury is not present, and for that reason delayed contrast enhancement may not be visible.

The other possible diagnosis in the case of coronary artery spasm in the context of acute myocarditis is difficult to exclude. However, CMR was inconsistent with endomyocardial enhancement, although ergonovin stimulation was not performed. Furthermore, the detection of Coxsackie virus antibodies was based on the ELISA method, while the presence of the Coxsackie virus gene in myocardium can be pathogenic for the infection.[10] In our case the latter examination was not feasible, although the titre of IgM antibodies detected in the peripheral blood was indicative of recent infection.

To summarise, this patient with acute myocarditis presented with pain and ECG changes mimicking myocardial ischaemia, having also a history of preceding viral infection. In this case CMR was useful for the confirmation of the final diagnosis.

References

1. Dec GW Jr, Waldman H, Southern J, Fallon JT, Hutter AM Jr, Palacios I. Viral myocarditis mimicking acute myocardial infarction. J Am Coll Cardiol. 1992; 20: 85-89.
2. Deckers JW, Hare JM, Baughman KL. Complications of transvenous right ventricular endomyocardial biopsy in adult patients with cardiomyopathy: a seven-year survey of 546 consecutive diagnostic procedures in a tertiary referral centre. J Am Coll Cardiol. 2001; 37: 786-792.
3. ACC/AHA 2005 Guideline Update for the Diagnosis and Management of Chronic Heart Failure in the Adult. J Am Coll Cardiol. 2005; 46; 1-82.
4. Sarda L, Colin P, Boccara F, et al. Myocarditis in patients with clinical presentation of myocardial infarction and normal coronary angiograms. J Am Coll Cardiol. 2001; 37: 786-792.
5. Mahrholdt H, Goedecke C, Wagner A, et al. Cardiovascular magnetic resonance assessment of human myocarditis: a comparison to histology and molecular pathology. Circulation. 2004; 109: 1250-1258.

6. Laissy JP, Messin B, Varenne O, et al. MRI of acute myocarditis: a comprehensive approach based on various imaging sequences. Chest. 2002; 122: 1638-1648.
7. White J, Patel MR. The role of cardiovascular MRI and the cardiomyopathies. Cardiol Clin. 2007; 25: 71-95.
8. Sourides BE, Theofilogiannakos EK, Theofilogiannakos G, et al. Clinical experience from 1000 consecutive cardiovascular MRI cases at a tertiary referral medical center. Hellenic J Cardiol. 2007; 48: 192-197.
9. Abdel-Aty H, Boye P, Zagrosek A, et al. Diagnostic performance of cardiovascular magnetic resonance in patients with suspected acute myocarditis: comparison of different approaches. J Am Coll Cardiol. 2005; 7: 1815-1822.
10. Triantafilou K, Orthopoulos G, Vakakis E, et al. Human cardiac inflammatory responses triggered by Coxsackie B viruses are mainly Toll like receptor 8 dependent. Cell Microbiol. 2005; 7: 1117-1126.