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CASE REPORT

Takotsubo syndrome or acute myocarditis? The role of cardiac magnetic resonance imaging☆

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Abstract Acute myocarditis is often misdiagnosed, and its evolution is not always benign; correct and prompt diagnosis is therefore essential.

We report the case of a 62-year-old woman with chest pain after a stressful event and ST-segment elevation on the electrocardiogram, in whom urgent cardiac catheterization showed normal coronary arteries and left ventricular apical ballooning, suggesting takotsubo syndrome. However, cardiac magnetic resonance imaging showed lesions typical of acute myocarditis, thus suggesting this diagnosis.

We highlight the diagnostic difficulty in patients with chest pain, elevated troponin and normal coronary arteries, and the key role of cardiac magnetic resonance in differential diagnosis.

PALAVRAS-CHAVE
Miocardite aguda;
Síndroma de *takotsubo*;
Ressonância magnética cardíaca

Síndroma de takotsubo ou miocardite aguda? O papel da ressonância magnética cardíaca

Resumo A miocardite aguda é uma entidade frequentemente subdiagnosticada e com uma evolução que nem sempre é benigna, pelo que o seu diagnóstico se torna mandatório.

Apresenta-se o caso de uma mulher de 62 anos, com dor precordial anginosa após evento de *stress* emocional, associada a elevação do segmento ST no eletrocardiograma, cujo cateterismo cardíaco de urgência mostrou artérias coronárias normais e balonização apical do ventrículo esquerdo, sugestivo da síndroma de *takotsubo*. Contudo, a ressonância magnética cardíaca mostrou lesões compatíveis com miocardite aguda, revelando, assim, este diagnóstico.

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Destacamos a dificuldade diagnóstica no doente com dor torácica, elevação de troponina e artérias coronárias normais, em que a ressonância magnética cardíaca assume um papel fundamental no diagnóstico diferencial.

Introduction

In patients admitted to the emergency department (ED) with chest pain, distinguishing between ischemic and nonischemic etiology can be a challenge. Differential diagnosis is based on clinical history and diagnostic exams.[1]

Patients with hemodynamically significant coronary lesions are identified by cardiac catheterization, confirming ischemic etiology. However, a definitive diagnosis in patients with acute coronary syndrome and angiographically normal coronary arteries is more difficult: it could be acute myocarditis, takotsubo cardiomyopathy (TC) or myocardial ischemia with normal coronary arteries, each of which has distinct features on cardiac magnetic resonance imaging (CMRI).[2]

CMRI is a noninvasive exam that provides a unique morphological characterization, including at tissue level. It identifies, localizes and determines the etiology of myocardial lesions, distinguishing inflammation from ischemia.[1]

The first use of CMRI in acute myocarditis was described in 1991 by Gagliardi et al., for diagnosing the condition in children.[3] CMRI assesses the myocardium through T1- and T2-weighted sequences, on which areas with the extracellular and interstitial edema found in acute myocarditis show up as a hyperintense signal on T2 sequences.[4] Furthermore, using a paramagnetic contrast agent, irreversible myocardial lesions can be identified by late enhancement sequences, which distinguishe myocarditis from TC. Biopsy of such lesions may reveal foci of active myocarditis, and so, as well as determining the type of lesion, CMRI can identify the best site for endomyocardial biopsy.[5]

There has also been considerable research in the use of CMRI in TC. Eitel et al.[6] and Koeth et al.[7] demonstrated focal edema in T2-weighted sequences in patients with TC in segments showing wall motion abnormalities, but no myocardial lesions on late enhancement studies.

In myocardial ischemia with angiographically normal coronary arteries, CMRI shows a perfusion defect in inversion-recovery gradient-echo or T1-weighted steady-state free precession sequences, with evidence of late enhancement in the same area, transmural or subendocardial, if there is necrosis. The location of this necrosis correlates with wall motion abnormalities, enabling a diagnosis of myocardial infarction (MI) in the absence of coronary lesions.[2]

Case report

We report the case of a 62-year-old woman with a first episode of chest pain after a family argument. She had no cardiovascular risk factors but had been diagnosed with age-related macular degeneration. She reported no recent illness or therapy.

When she went to the ED with chest pain of eight hours' duration she was hemodynamically stable with no signs of heart failure. The ECG showed sinus rhythm with ST-segment elevation in DI and aVL and ST-segment depression in DIII and aVF (Figure 1). Transthoracic echocardiography performed in the ED showed akinesia with dilatation of the apex and the mid-apical segments of the left ventricular (LV) wall, with no pericardial effusion (Figure 2).

Urgent cardiac catheterization, performed under anticoagulation and dual antiplatelet therapy with aspirin and clopidogrel showed normal coronary arteries and no vasospasm. Ventriculography showed akinesia with apical ballooning and moderate global systolic dysfunction (ejection fraction 42%) (Figure 3). Biomarkers of myocardial necrosis were elevated (peak troponin I 6.45 ng/ml and CK-MB mass 27.1 μg/dl, with total CK 248 μg/dl). C-reactive protein (CRP) was 0.2 mg/dl. During hospital stay she was free of chest pain and developed generalized T-wave inversion on the ECG (Figure 4), accompanied by progressive recovery of the LV wall motion abnormalities. Therapy was maintained with dual antiplatelets, anticoagulation, angiotensin-converting enzyme inhibitors (captopril 6.25 mg every 8 h) and statins (atorvastatin 10 mg/day). Beta-blockers were not tolerated due to symptomatic bradycardia.

In view of the clinical setting and results of the diagnostic exams, a provisional diagnosis of TC was made. Since this is a diagnosis of exclusion, 24-h urinary catecholamine assessment was performed, which was normal, as was computed tomography of the adrenal glands. Serology for cardiotropic viruses (adenovirus, Coxsackie, CMV, echovirus, EBV, HZV, HSV-1 and HSV-2, parvovirus B19, influenza, HCV and HIV) in two determinations, despite negative IgM, and autoimmune tests were also negative.

CMRI performed on the 7th day revealed a non-dilated and non-hypertrophied left ventricle, no longer showing wall motion abnormalities and with good global systolic function; there was a hyperintense myocardial signal in T2-weighted turbo spin-echo sequences located in the lateral wall and late gadolinium enhancement in the subepicardium of the same wall (Figure 5). The right ventricle presented no abnormalities.

These findings were compatible with acute myocarditis with involvement of the LV lateral wall, which was in agreement with the ECG changes, although the wall motion abnormalities at admission were compatible with TC.

Treatment with captopril was maintained and ibuprofen 400 mg every 8 h was initiated. There was complete recovery of wall motion and global LV systolic function and normalization of the ECG pattern, and the patient was discharged on the 10th day with a diagnosis of acute myocarditis.