Value of cardiac MRI in peripartum cardiomyopathy

Intérêt de l’IRM cardiaque dans la cardiomyopathie du péripartum

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A 28-year-old woman was admitted for heart failure with severe dyspnoea, 15 days after her first normal delivery with a normal last trimester of pregnancy. She had no personal history, familial cardiac disease or cardiovascular risk, and her electrocardiogram was normal, but transthoracic echocardiography showed left ventricular (LV) dysfunction (LV ejection fraction [LVEF] 20%) despite normal findings on coronary angiography. C-reactive protein and N-terminal pro-brain natriuretic peptide concentrations were 46 mg/L and 2015 ng/L, respectively. Rapidly, she presented cardiogenic shock that needed mechanical support (extracorporeal life support). After a transient improvement, allowing weaning of mechanical support, cardiac magnetic resonance imaging (MRI) was performed. Cine sequences showed global hypokinesia, with LVEF 23%, LV end-diastolic volume 153 mL, and LV end-systolic volume 119 mL. LV short-axis views with T2-weighted sequences showed high signal intensity circumferential (Fig. 1, Panel A, arrow) evidence of inflammation or oedematous tissue. MRI performed 10 minutes after a bolus of gadolinium for delayed enhancement imaging showed circumferential areas of myocardial delayed enhancement (MDE) (Fig. 1, Panel B, arrow). Endomyocardial biopsy showed no evidence of acute viral myocarditis. One month later, the patient underwent heart transplant, with an uneventful postoperative course.

At gross examination of the explanted heart, while the right ventricle was nearly normal, the LV cavity was dilated, thin and had a translucent pale area, almost circumferential, suggesting a developing fibrotic process (Fig. 1, Panel C, arrow). On histological examination of the left ventricle, marked interstitial fibrosis was observed without necrosis, associated with an inflammatory cell infiltrate with a background that was still very

Abbreviations: LV, Left ventricular; LVEF, Left ventricular ejection fraction; MDE, Myocardial delayed enhancement; MRI, Magnetic resonance imaging; PPCM, Peripartum cardiomyopathy.

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Figure 1. Panel A: T2-weighted spin echo sequences; hypersignal circumferential means presence of myocardial oedema (white arrow). Panel B: T1-weighted inversion recovery sequences, realized 10 minutes after injection of gadolinium with myocardial late gadolinium enhancement circumferential (white arrow). Panel C: the explanted heart has a translucent pale area, almost circumferential, suggesting a developing fibrotic process (white arrows). Panel D: histological examination of the left ventricle showed marked interstitial fibrosis without necrosis, associated with a mild and polymorphous inflammatory cell infiltrate in an oedematous background (black arrow) and myocytes with myofibrillary loss (white arrow). LV: left ventricle; RV: right ventricle.

oedematous (Fig. 1, Panel D, black arrow). Myocytes showed frequent myofibrillary loss and vacuolization (haematoxylin-eosin-saffron, original magnification × 200) (Panel D, white arrow). These histological features are highly dependent on the time between the onset of symptoms and myocardial analysis but in our case were quite evocative. The earlier the analysis is done, the more peripartum cardiomyopathy (PPCM) is associated with an inflammatory process in the myocardium.

PPCM is a rare (one case per 4000 live births in the USA) but severe disease in 20% of cases, where patients can either die or require heart transplantation. Normally, only 30% recover normal LV function after six months of treatment but continuing improvement has been observed in the second and third years, confirming that the recovery phase is not limited to the first 6 months. The aetiology is not fully understood and several physiopathological mechanisms have been proposed, including viral myocarditis, abnormal immune response to pregnancy, abnormal response to the haemodynamic stress of pregnancy, increased myocyte apoptosis, cytokine-mediated inflammation, low selenium level, genetic factors, excessive prolactin production, abnormal hormonal function and increased adrenergic tone. Owing to the complexity of the physiopathology, the prognosis remains difficult to predict. Interestingly, MDE was correlated with histological findings showing fibrosis and necrosis, and had poor prognostic value. Cardiac MRI could improve the follow-up of PPCM with earlier detection of serious forms.

Conflicts of interest statement

None.