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Contribution of cardiac MRI to early evaluation and impact on the long term follow-up in myocarditis mimicking an acute coronary syndrome. A 43-cases prospective study

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Background: Acute myocarditis (AM) diagnosis is a challenge to rule out an acute coronary syndrome (ACS). AM is thought to favour the evolution towards dilated cardiomyopathy (DCM) and the occurrence of severe arrhythmias. Three months after the acute episode, re-evaluation including cardiac MRI could help to identify patients at risk for unfavourable evolution. The use of MRI has rarely been investigated in AM prognostic stratification.

Methods and results: We report a prospective series of 43 consecutive patients hospitalized for AM mimicking ACS: 36 men and 7 women, 32 years old on average, without sign of heart failure. All patients presented with troponin I elevation. Echocardiography showed moderate global left ventricular dysfunction in 6 cases and segmental wall motion abnormalities in 22. MRI performed early after admission never showed myocardial first-pass perfusion defect after gadolinium injection but subepicardial delayed-enhancement (DE) areas in 39 cases mainly located in lateral segments. Three months after the acute episode, no patient was symptomatic. Echocardiography, Holter monitoring and biological check-up were normal. MRI showed the persistence of DE in 23 cases without wall motion abnormality in the affected segments. The presence of these latter abnormalities led to effect an annually clinical examination with an ECG. One patient was lost at further follow-up. Among the other 22 patients, only one patient displayed heart failure revealing DCM with ventricular arrhythmias at 3 – year mean follow-up.

Conclusions: at the time of admission, the absence of early perfusion defect at cardiac MRI after gadolinium injection and the subepicardial localization of the DE constitute reliable criteria in favour of AM diagnosis, allowing to rule out ACS. During the follow-up the persistence of a DE does not allow any prognosis stratification. In our series after a mean 3-year follow-up, it is not associated with any clinical and para-clinical disorder except in one case.

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Diabetic cardiomyopathy: data from a series of 656 asymptomatic diabetic patients with known cardiac ischemic status

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Background: The aim of the study was to assess the prevalence of sub-clinical cardiomyopathy among patients with type 2 diabetes without hypertension or coronary artery disease (CAD). Materials and methods: 656 patients with type 2 diabetes for 14±8 yrs (359 men, 59.7±8.7 years, HbA1c 8.7±2.1%), without cardiac symptom and at least one cardiovascular risk factor (hypertension 74%; dyslipidemia 70%; smoking habits 22%; peripheral occlusive arterial disease 10%, nephropathy 39%) had a contributive cardiac echography at rest; underwent a stress cardiac scintigraphy to screen for silent myocardial ischemia (SMI), and in case of SMI, a coronary angiography to screen for silent CAD.

Results: SMI was diagnosed in 206 patients, and 71 of them had silent CAD. In the patients without hypertension or CAD (n=157), left ventricular hypertrophy (LVH: 24.1%) was the most frequent abnormality, followed by left ventricular dilation (8.6%), hypokinesia (5.3%), abnormal type 1 relaxation (4.8%) and systolic dysfunction (3.8%). No parameter was associated with LVH neither with LV dilation nor with abnormal relaxation. In multivariate analysis, the parameters associated with hypokinesia were SMI (Odds ratio 4.7 [2.7-8.17] p<0.01) and peripheral occlusive arterial disease (OR 12.2 [1.4-103.1] p<0.05); those associated with systolic dysfunction were SMI (OR 114.6 [1.7-7907], p<0.01), HbA1c (OR 1.9 [1.1-3.2] p<0.05) and BMI (OR 1.6 [1.1-2.4] p<0.05). LVH was more prevalent among hypertensive patients (without CAD 34.5%; with CAD 46.7%); and hypokinesia in the patients with CAD (without hypertension 13.3%, with hypertension 13.7%).

Conclusion: In asymptomatic type 2 diabetic patients, diabetic cardiomyopathy is highly prevalent and is characterized by LVH, SMI, obesity and poor glycemic control contribute to systolic dysfunction and/or hypokinesia. Hypertension is associated with more LVH, and CAD with more hypokinesia.