



Case Report

Cardiac Magnetic Resonance Imaging Myocardial Viability Assessment After Myocardial Infarction: A Case Report

Évaluation de la Viabilité du Myocarde par Imagerie par Résonance Magnétique Cardiaque Après un Infarctus du Myocarde : À Propos d'un Cas

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ABSTRACT

Cardiac Magnetic Resonance Imaging (MRI) represents the gold standard and valuable reference tool for the assessment of myocardial viability in the post-infarction period. We report the case of a 64-year-old man with a 3-month history of non-revascularized STEMI who underwent a cardiac MRI for a myocardial viability study. His other medical history included diabetes, hypertensive and bilateral Peripheral Arterial Disease (PAD) without stroke. His clinical presentation included a heart failure and low cardiac output signs, electrocardiographic and echocardiographic signs of ischemic heart disease with severe myocardial performance decreasing. Coronary angiography revealed triple-vessel disease (TVD). Cardiac MRI revealed nonviable myocardium with the presence of an apical thrombus in the left ventricle. Given the contraindication of myocardial revascularization due to the myocardial nonviability, the patient was put on medical treatment. He died in the intensive care unit from cardiocirculatory failure.

RÉSUMÉ

L'imagerie par résonance magnétique (IRM) cardiaque représente l'étalon-or et un outil de référence précieux pour l'évaluation de la viabilité du myocarde dans la période post-infarctus. Nous rapportons le cas d'un homme de 64 ans, ayant des antécédents de STEMI non revascularisé depuis 3 mois, qui a subi une IRM cardiaque pour une étude de la viabilité du myocarde. Ses autres antécédents médicaux comprenaient un diabète, une hypertension et une maladie artérielle périphérique bilatérale sans accident vasculaire cérébral. Sa présentation clinique comprenait une insuffisance cardiaque et des signes de faible débit cardiaque, des signes électrocardiographiques et échocardiographiques de cardiopathie ischémique avec une diminution sévère de la performance du myocarde. La coronarographie a révélé une maladie des trois vaisseaux. L'IRM cardiaque a révélé un myocarde non viable avec la présence d'un thrombus apical dans le ventricule gauche. Étant donné la contre-indication d'une revascularisation myocardique en raison de la non-viabilité du myocarde, le patient a été mis sous traitement médical. Il est décédé dans l'unité de soins intensifs d'une défaillance cardiocirculatoire.

INTRODUCTION

Cardiac magnetic resonance imaging represents the gold standard in the non-invasive exploration of coronary lesions and constitutes an excellent and valuable reference tool for the assessment of myocardial viability in the post-infarction period [1-4]. The search for myocardial viability is a prerequisite for functional recovery after any attempt at myocardial revascularization in patients in the post-infarction period with left ventricular systolic dysfunction [5-9], because myocardial viability has a

clinical, therapeutic and prognostic impact in the post-infarction period [5,10,11]. Acute or chronic coronary syndromes constitute a major public health problem because they are responsible for 60% to 80% of sudden deaths [12-13]. These coronary diseases are frequently responsible for systolic dysfunction of the left ventricle (LV), which increases cardiovascular morbidity and mortality [6, 11, 14-19]. This subsequent LV dysfunction is a major prognostic factor, responsible for complications such as heart failure [6-20, 21]. According to the literature, in case of viability only, myocardial

revascularization has a significant clinical impact by improving the hemodynamic state, patient survival [2, 11] and the segmental and global function of the left ventricle [6, 22-24]. Among the many areas of clinical applications in cardiac imaging, cardiac MRI, due to its very high temporal and spatial resolution, is a relevant tool in the morphological, metabolic and functional evaluation of the myocardium with a view to revascularization [1, 6, 10, 11].

CASE PRESENTATION

A 64-year-old man was referred from the emergency room for orthopnea and arterial hypotension. His medical history included a 3-month history of a late seen and non-revascularized STEMI managed in a non-specialized health center. His others medical history included diabetes, hypertensive and bilateral Peripheral Arterial Disease without stroke.

His clinical presentation included a poor general condition, body mass index BMI: 21.55kg/m², temperature of 38°C, arterial hypotension of 80/50 mmHg, tachycardia, cold extremities, dark urine. Left-sided heart failure symptoms included dyspnea at stage IV of the New York Heart, cough bring up mucus tinged with blood, spread peak shock, fine bilateral and symmetrical crackles at the 2 pulmonary bases and rapid heartbeat. Right-sided heart failure symptoms included spontaneous jugular vein turgor, tender hepatomegaly with a blunt lower edge, hepatojugular reflux, bilateral and soft lower limb edema. Chest X-ray objectified a cardiomegaly with cardiothoracic index at 0.64.

Electrocardiogram (Figure 1) showed Q waves of necrosis in the lower territories, subepicardial ischemia in extensive anterior territories, left anterior hemi-block and right ventricular hypertrophy.

The Transthoracic cardiac doppler ultrasound allows to objectify a dilated left ventricle with LVd/s (60mm/50mm or an indexed LV =34.48 kg/m²), a segmental kinetic disorder such as "Global hypokinesia and septal apical dyskinesia, and a severe impairment of left ventricular systolic function with a Left Ventricular Ejection Fraction (LVEF) to 22%. The filling pressures were high with a moderate mitral regurgitation due to dilation of the mitral annulus. There were a hypokinetic right ventricle of normal size with a TAPSE lowered to 11 mm, dilated inferior vena cava at 24 mm and moderate pulmonary arterial hypertension at 49 mmHg.

In the biological tests, there were moderate renal insufficiency with a creatinine clearance of 33.5 (Urea: 0.95 g/l, Creatinine: 23.1 mg/l), hyperglycemia (Glycemia: 1.29 g/l), malaria access with 3000 trophozoites, polymorphonuclear-predominant (87.7%) hyperleukocytosis (16000.67 white blood cells).

Diagnostic Coronary Angiography revealed triple-vessel disease (TVD) with occlusion of anterior descending artery. No coronary revascularization was performed.

Arteriovenous doppler ultrasound of the lower limbs described a non-aneurysmal abdominal aorta (DAP = 15MM) with moderately atheromatous walls. Moderate atheromatous overload of the iliac arteries with triphasic flow of type N of Saint Bonnet. - At the femoropopliteal level, moderate calcified overload of the left and right arteries of the femoropopliteal axes with triphasic flow of type N of Saint Bonnet. - At the tibial level, on the left: stenosis at the middle 1/3 of the anterior tibial artery. The flow was monophasic dampened at the level of the type D of Saint Bonnet pedigree. At the tibial level, on the right: distal stenosis of the anterior tibial and fibular arteries. The flows were monophasic, dampened at the level of the pedigree and the posterior tibial type D of Saint Bonnet. In conclusion, there was a bilateral Peripheral Arterial Disease (PAD) with distal predominance characterized by stenosis of the anterior tibial arteries and the left fibular artery.

The cardiac MRI (Figure 2) performed to assess myocardial viability on a 1.5 Tesla machine concluded to a myocardial nonviability. Indeed, there was an ischemic heart disease with severe biventricular systolic dysfunction, an aneurysm of the territory of the anterior interventricular complicated by an apical thrombus. A transmural between 75% -100% therefore an absence of myocardial viability. The Cine MRI sequences (SSFP) Short axis, Long axis 2 chambers, 03 chambers and 04 chambers, of average quality, made it possible to evaluate the global functions of the 2 ventricles. There are segmental kinetic disorders such as dyskinesia of segments 2, 3, 7, 8, 9, 13, 14, 15 and 17, i.e. 50 to 55% of the left ventricle. On the mapping, the native values before injection of gadolinium chelate allowed to have normal values in particular in the median septal region at 1027 ms and the T2 value at 46 ms. After injection of gadolinium chelate, there is a subendocardial hypersignal at the transmural level affecting segments 2, 3, 7, 8, 9, 13, 15 and 16, 17 or 50 to 55% of the left ventricle. This transmural tension extends to the tip of the right ventricle. Also note a thrombus concerning the antero-septal apical wall extending to the apex and measuring 37mmx14 mm. Given the contraindication of myocardial revascularization due to the myocardial nonviability, the patient was put on medical treatment based on reduction in sodium and fluid intake, diuretics, vasoactive amines, anti-platelet aggregants, anticoagulants, ACE inhibitors taking into account creatinine clearance, statins, antibiotic and antimalarial treatment. The patient died in the intensive care unit from cardiocirculatory failure.



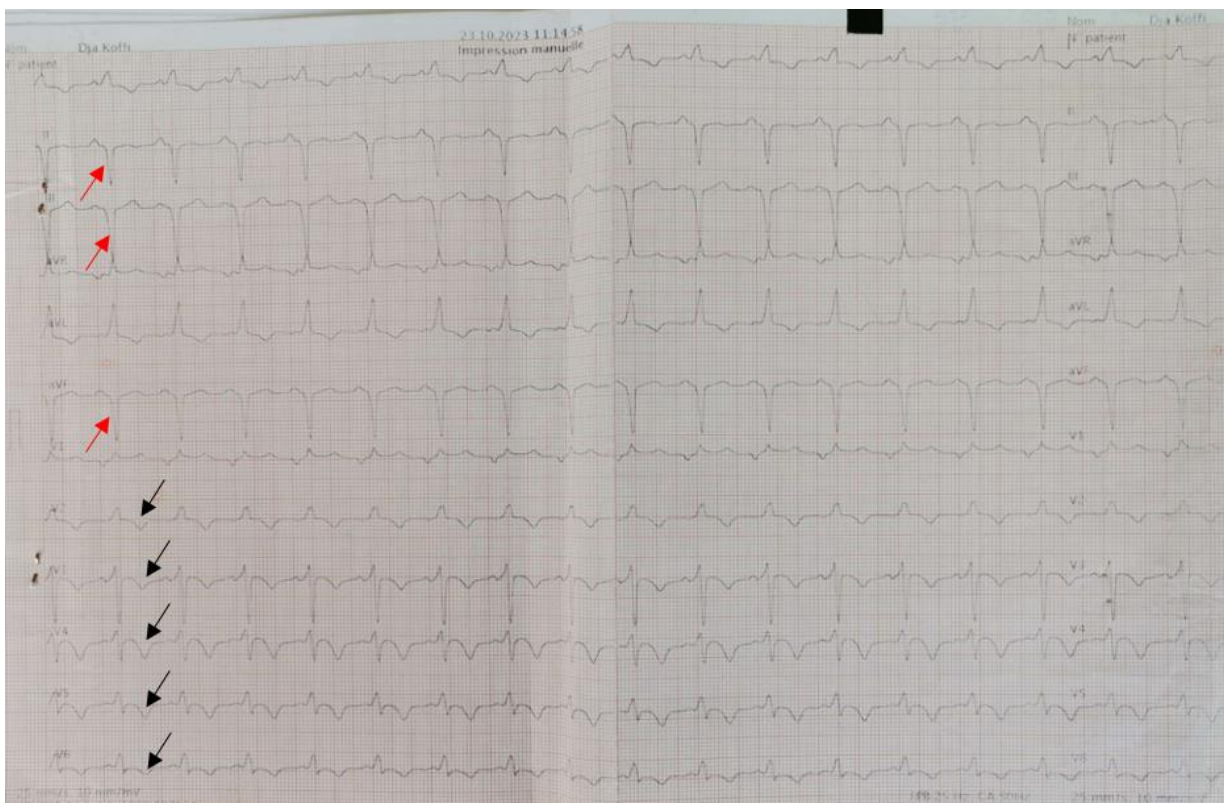


Figure 1: Electrocardiogram (ECG) which showed Q waves (red arrows) of necrosis in the lower territories, subepicardial ischemia in extensive anterior territories (black arrows), left anterior hemi-block and right ventricular hypertrophy.

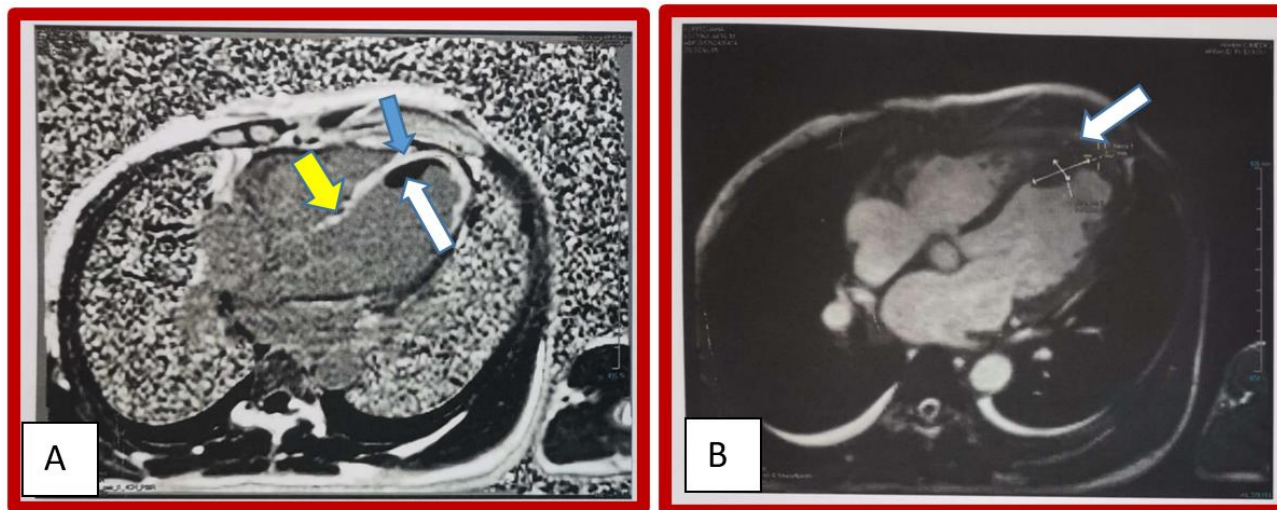


Figure 2: Cardiac magnetic resonance imaging which highlights in picture **A**, an ischemic model with subendocardial contrast uptake with transmural extension (blue arrow) and no-reflow (yellow arrow) and in picture **B**, a thrombus in the septal apical position (white arrows).

DISCUSSION

Viable myocardium and nosology of coronary artery diseases

The viability of the cardiomyocyte in the post-infarction refers to living cells, not irreversibly altered [5, 20]. For many years, a segmental asynergy post-infarction was considered as a definitive and irreversible process [10,

20]. And only in the years 1978-1980 [10, 11, 25], Diamond and Forrester put forward the theory of the concept of myocardial viability with the possibility of myocardial recovery. The nosological classification of ischemic heart disease is divided into 3 major categories [6, 10, 11, 20]: 1- infarction corresponding to myocardial necrosis and fibrosis; 2- myocardial sideration [26] also called stunning corresponds to a reversible dysfunction of

the myocardium in the post-infarction period with an ad-integrum restitution of myocardial contractile function. It was described by Kloner and Braunwald and 3-myocardial hibernation described by Rahimtoola [27] in 1985, is a state of segmental hypocontractility due to a decrease in perfusion and therefore coronary flow [20, 27, 28]. However, it is important to specify that in the presence of a very extensive zone of akinesia, these 03 nosological anomalies can coexist [10].

Myocardial viability in MRI and clinical and therapeutic implications

Assessment of myocardial viability in patients with coronary artery disease is crucial to distinguish reversible myocardial lesions or irreversible myocardial alterations [29]. The functional recovery of myocardial cells in the post-infarction depends on the state of viability or non-viability [1, 30, 31].

According to the ESC 2018 [33], it is strongly recommended with level of evidence A (class IA), the revascularization of bi or tri-vessel patients with systolic dysfunction of the left ventricle and a radiological proof of viability. In case of viable myocardium, revascularized patients improve their survival and reduce cardiovascular morbidity and mortality [10, 6, 20, 34] by reducing by 3 the risk of occurrence of a coronary event in the three years post infarction [10, 35]. However, the regions of the myocardium largely infarcted are not all likely to benefit from revascularization and this revascularization is necessary only when it can bring a benefit to the patients because the non-viable myocardium will not recover its left ventricular systolic function [1, 6, 11, 20, 34, 36]. MRI because of its high spatial resolution, explores cellular metabolism and the integrity of the cellular limb [10, 37]. In late enhancement, the gadolinium chelate penetrates the altered cellular limb of the myocytes with diffusion of gadolinium into the intracellular medium, which accumulates in the necrotic areas leading to an increase in the volume of distribution and a slowdown in the elimination kinetics (Wash out) [20, 38] According to the literature, there is a correlation between the viability and transmural of subendocardial hypersignals on the LGE enhancement sequence [2, 6, 20]. A transmural >50% is synonymous with the absence of myocardial viability [2, 36]. Indeed, several studies [39-41] have confirmed that patients with transmural hypersignals of 25% and more than 50% would recover, respectively, 80% and 10% of their function. The problem arises for patients with an intermediate transmural between 25% and 50%, whose chances of recovery would be 50:50 [39]. In clinical practice, visual quantification of late enhancement or automatic quantification with a threshold of 50% can be performed [39, 42, 43].

Other methods for exploring myocardial viability [10, 42, 44].

Non-invasive methods for assessing myocardial viability are stress echocardiography under dobutamine, myocardial tomoscintigraphy and cardiac MRI, positron emission tomography, single-photon emission computed tomography, post-extrasystolic potentiation (left ventricular cine-angiography), electromechanical

mapping, 201Tl scintigraphy, positron emission tomography, stress echocardiography. These explorations globally interpret myocardial viability. The gold standard in the exploration of myocardial perfusion thanks to gadolinium chelate but also the contractile reserve under dobutamine (stress MRI) remains Cardiac MRI.

CONCLUSION

Atherosclerosis and its risk factors such as hypertension and diabetes expose patients to serious and multisegmental damage. MRI is the gold standard examination to assess cardiac anatomy, segmental and global function, myocardial perfusion and tissue characterization [43]. The clinical and therapeutic implications of myocardial viability influence 1- the decision of revascularization, 2- the prognosis of patients, 3- the management of heart failure and 4- the risk assessment.

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