

Prognostic significance of cardiac magnetic resonance imaging: Update 2010

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Abstract

Cardiac magnetic resonance imaging (CMR) has become an indispensable imaging technique for the diagnosis and treatment of patients with cardiovascular diseases. Technical advances in the past have rendered CMR unique in the evaluation of cardiovascular anatomy, physiology, and pathophysiology due to its unique ability to produce high resolution tomographic images of the human heart and vessels in any arbitrary orientation, with soft tissue contrast that is superior to competing imaging modalities without the use of ionizing radiation. CMR imaging is the gold standard for assessing left and right ventricular function and for detecting myocardial tissue abnormalities like edema, infarction, or scars. For prognostic reasons abnormal structure and dysfunction of the heart, and the detection of myocardial ischemia and/or myocardial scars are the main targets for CMR imaging. In this review we briefly describe the prognostic significance of several CMR imaging techniques and special CMR parameters in patients with coronary artery disease (CAD), with cardiomyopathies, and with chronic heart failure. Myocardial ischemia proved to be a strong predictor of an adverse outcome in patients with CAD. Microvascular obstruction in acute myocardial infarction is a new and independent parameter of negative left ventricular remodeling and a worse prognosis. Myocardial scars in patients with CAD and unrecognized myocardial infarction heralds a negative outcome. Scar in patients with dilated or hypertrophic cardiomyopathy are a strong predictor of both life-threatening ventricular tachyarrhythmias and prognosis. CMR imaging may improve the assessment of inter- and intraventricular dyssynchrony and provide prognostic information by detecting myocardial scars. (Cardiol J 2010; 17, 6: 549–557)

Key words: cardiac magnetic resonance imaging, late gadolinium enhancement, prognosis, coronary artery disease, dilated cardiomyopathy, hypertrophic cardiomyopathy, chronic heart failure, cardiac resynchronization therapy

Clinical significance of cardiac magnetic resonance imaging

Magnetic resonance imaging (MRI) has become a unique tool for investigating the morphology and function of the cardiovascular system. The strength of cardiac magnetic resonance (CMR) is based on its high spatial and temporal resolution,

the high blood-tissue contrast particularly when using the steady-state free precession (SSFP) techniques, and the capability of three-dimensional (3D) reconstruction of cardiac and vascular structures. In contrast to echocardiography, MRI is largely user independent and does not substantially suffer from variations in image quality and poor patient-related echogenicity. It is independent of slice-orienta-

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tion or acoustic window, and its 3D nature enables direct calculation of ventricular dimensions without any need of geometrical models. Most importantly, MRI enables direct visualization of myocardial perfusion (T1 weighting imaging after gadolinium contrast injection), and the identification of myocardial edema (by native T2 weighted imaging). Equally important appears the capability to detect myocardial tissue abnormalities such as necrosis or scar using the late gadolinium enhancement (LGE) technique. Lastly, this imaging technique completely avoids biological damage to the patient in contrast to radiation exposure from nuclear or x-ray techniques (e.g. computed tomography etc).

For prognosis abnormal structure and dysfunction of the heart are the main targets for CMR imaging. The main parameters identifying ventricular dysfunction are accurate and reproducible measurements of ventricular dimensions, mass and function, as well as the detection and quantitation of areas of LGE.

In the following we will comprehensively review published data on the prognostic significance of several CMR imaging techniques in patients with coronary artery disease (CAD), with cardiomyopathies, and with chronic heart failure (HF).

Chronic stable coronary artery disease

Inducible myocardial ischemia

Dobutamine stress CMR and myocardial first pass perfusion during adenosine stress are the two main CMR techniques widely used to detect myocardial ischemia [1–3]. Imaging of stress-induced myocardial ischemia cannot only be used for detecting or excluding flow limiting coronary artery stenoses, but also for evaluating prognosis.

For instance dobutamine stress CMR (DSMR) has been used [4] for preoperative risk stratification, in a subgroup of patients with intermediate risk prior to a non-cardiac surgical procedure, positive DSMR test was an independent predictor of myocardial infarction, cardiac death, or HF during or after surgery. Jahnke et al. [5] evaluated the prognostic value of DSMR and CMR-first pass perfusion (FPP) in 533 patients with chest pain or dyspnea and suspected CAD. The 3-year event-free survival was 99.2% for patients with normal DSMR and FPP and 83.5% for those with abnormal DSMR and FPP. By multivariate analysis, ischemia on magnetic resonance testing (either DSMR or FPP) was an independent predictor of cardiac events. The cumulative event rate for an abnormal DSMR at 1-, 2-,

and 3-year was 7.3, 10.3, and 18.8, and for an abnormal FPP 6.2, 12.2, and 16.3, respectively. Moreover, an abnormal CMR stress test result had significant incremental value over clinical risk factors and resting wall motion abnormality ($p < 0.001$). In another study by Hundley et al. [6] DSMR has also been used for determining the prognosis of 279 patients with suspected CAD, who could not be studied adequately using transthoracic stress echocardiography. In a multivariate analysis, the presence of inducible myocardial ischemia by DSMR (hazard ratio [HR] 3.3) or an left ventricular ejection fraction (LVEF) $< 40\%$ (HR 4.2) was associated with future myocardial infarction (MI) or cardiac death independent of the presence of risk factors for coronary arteriosclerosis.

Several studies have also reported on the prognostic significance of CMR-FPP. Pilz et al. [7] followed 218 patients with suspected CAD and a negative MR perfusion test. The 12-month rate of major adverse cardiovascular events (MACE) was 2 of 218 patients with no cardiac death or MI, and the negative predictive value of a negative MR first pass perfusion was 99.1%. The authors concluded that a negative first pass perfusion result may reduce the number of redundant coronary angiographies. Jahnke et al. [5] showed that a myocardial perfusion defect on FPP is associated with a HR of 10.6 for the endpoints cardiac death or MI over a follow-up period of 2.3 years. Bodi et al. [8] performed dipyridamole stress CMR and FPP in a series of 420 patients with chest pain and known or suspected CAD. MACE rate after a median follow-up period of 420 days was significantly higher in patients with inducible perfusion defects compared to patients without a perfusion defect (17% vs 5%). This was also the case for presence vs absence of LGE (20% vs 6%). However, in a multivariate analysis adjusted for baseline characteristics, the extent of wall motion abnormalities on dipyridamole stress was independently related to the MACE rate. FPP studies may also be useful in patients with acute chest pain, as demonstrated by Ingkanisorn et al. [9] in 135 troponin-negative patients admitted to the emergency department. Patients with a negative stress test had a 100% event-free survival after 1 year follow-up (no subsequent diagnosis of CAD or an adverse outcome), compared with only 20% with a perfusion defect in FPP. Thus, it may be concluded that whenever myocardial ischemia can be excluded in patients with suspected or known CAD by either DSMR or FPP, these patients will have a favourable outcome prospectively.

Unrecognized myocardial scar

Late gadolinium enhancement CMR (LGE-CMR) has shown excellent sensitivity and specificity for detection of even small myocardial scar. Furthermore, there is some data indicating that LGE imaging can reveal previously unknown MI, and that the presence of LGE has a strong prognostic impact. Unrecognized myocardial scar is more frequent than expected. In a population-based study in Sweden by Barbier et al. [10] including 259 randomly selected individuals aged 70-years, LGE indicating scar was detected in 19.8% of the subjects without a history of MI. The prevalence of unrecognized myocardial scar seems to be increased in patients with higher cardiovascular risk. In a study by Kwong et al. [11] LGE was present in 44 of 195 patients with suspected CAD but without known prior MI. The authors were able to show that the presence of even small amounts of LGE indicates a high risk and provides incremental prognostic value for MACE and cardiac mortality beyond common clinical, angiographic, and functional predictors. In another study the same group found similar results in diabetic patients without known prior infarction [12]. The presence of LGE as a sign of previous coronary events was the strongest predictor for an adverse cardiac outcome. These findings strongly support the general use of LGE imaging in patients with suspected CAD for improving risk assessment.

Acute myocardial infarction

Peri-infarct zone and ventricular arrhythmias

Ventricular arrhythmias and sudden cardiac death (SCD) are a major cause of morbidity and mortality in the post-MI period. The border zone of an infarct is often composed of areas of viable myocardium interspersed with infarcted tissue or scar, which can become a substrate for impaired conduction and re-entrant circuits as a prerequisite of malignant ventricular tachyarrhythmias. It has been postulated that this peri-infarct zone can be visualized by CMR on LGE images as areas along the infarct border of intermediate signal intensity. Schmidt et al. [13] studied 40 patients undergoing an electrophysiology study (EPS) prior to implantable cardioverter-defibrillator (ICD) implantation for primary prevention of SCD and found that the size of the infarct border zone measured by CMR predicted inducibility of monomorphic ventricular tachycardia (VT) on EPS. Yan et al. [14] reported that the size of the peri-infarct zone measured by

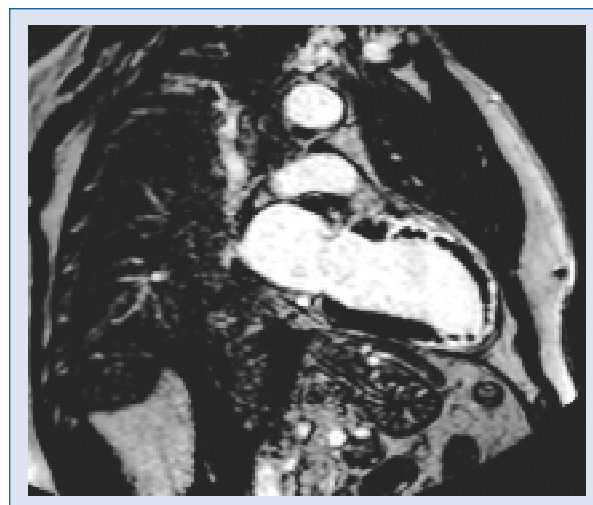


Figure 1. Late gadolinium enhancement image of a patient with acute anteroseptal myocardial infarction in a 2-chamber view. Note the bright areas of contrast accumulation (= infarcted myocardium), and some intramural (intra-infarct) dark areas indicating microvascular obstruction.

CMR was predictive for all cause and cardiac mortality over 2.4 years of follow up. Roes et al. [15] found in a CMR study of 91 post-MI patients scheduled for ICD implantation for either primary or secondary prevention of SCD that the size of the peri-infarct zone was a stronger predictor of appropriate ICD therapy, as a surrogate for SCD as compared to LVEF or total infarct size. Heidary et al. [16] could confirm these results in a study of 70 patients with a history of MI and ischemic cardiomyopathy, who were evaluated for ICD implantation and followed for 18 months. They could show that the size of the peri-infarct zone as well as total infarct size were predictive of cardiac events. Thus, it seems that LGE-based assessment of the structure and extent of the peri-infarct zone may provide a non-invasive parameter of an increased risk of post-MI patients of malignant ventricular arrhythmias and adverse outcome.

Myocardial infarct size

CMR-based assessment of infarct size using LGE is well validated (Fig. 1). Infarct size has been reported to be highly predictive for cardiac events at follow up. Yokota et al. [17] were able to show in 86 patients with prior MI followed for an average of 20 months that infarct size by LGE was a better predictor of cardiac events than LVEF, LV end-systolic volume (LVESV) or LV end-diastolic volume (LVEDV). Similar results were obtained by Cheong

et al. [18] in a cohort of 857 patients with and without CAD followed for a median of 4.4 years. They demonstrated that presence of LGE, reduced LVEF and HF symptoms were independent predictors of mortality. It was also shown that patients with LGE and an ejection fraction more than 50% had the MACE rates as those with an LVEF less than 50% without presence of LGE. These results were again corroborated by Larose et al. [19] in 103 acute ST elevation MI (STEMI) patients undergoing CMR within 12 hours and 6 months after percutaneous coronary intervention (PCI) and follow-up of an average of 2.4 years. They demonstrated that LGE size measured immediately after revascularization strongly and independently predicted recovery of LVEF after 6 months and long-term major cardiac events better than initial LVEF or clinical parameters. Wu et al. [20] found similar results in 122 STEMI patients who received CMR after revascularization and were followed for 2 years; infarct size was again a stronger indicator of worsened LV systolic function and clinical outcomes at follow up than were baseline measurements of LV systolic function. In addition, Plein et al. [21] were able to show that the ratio of infarct size (by LGE) to the extent of ischemia (visualized by adenosine stress first pass perfusion) was significantly higher in patients with Q-wave STEMI than in those with non-Q-wave STEMI or non-STEMI, while conversely, the amount of ischemia present was higher in NSTEMI patients than in STEMI patients, whereas the total area of ischemia and infarction was not different between the three groups.

Right ventricular infarction

Right ventricular (RV) infarction classically occurs along with an inferior LV infarction due to proximal occlusion of the right coronary artery. RV infarction is usually diagnosed by physical examination, ST elevation on right-sided electrocardiogram leads, or by echocardiographic evidence of RV dysfunction. However, CMR has been demonstrated to be much more sensitive for detecting small or medium RV infarcts than physical exam, ECG or echocardiography [22]. In a study of Jensen et al. [22] on 50 patients LGE indicating RV involvement was detected in 54% of patients (in 14 of 30 patients with inferior STEMI, and in 13 of 20 patients with anterior STEMI) with LGE-CMR. In a multivariate logistic regression model RV involvement was a strong independent predictor (odds ratio 15.8; cardiac index 4–63%) of major cardiac adverse events within a follow-up of 32 ± 8 months, even after normalization for LV infarct size and location [10, 13].

Bueno et al. [23] studied 198 elderly patients with inferior MI, 75% of whom did not receive revascularization. RV infarction in this cohort occurred in 41%, with a mortality of 47%, compared to 10% mortality in those without RV infarction. The incidence of cardiogenic shock, atrioventricular conduction block, and rupture of the interventricular septum was also markedly higher in those with RV involvement. In a more recent study by Giannitsis et al. [24] of 88 inferior MI patients, 87 of whom underwent PCI, RV infarction was present in 31% but did not show any significant short-term prognostic implications. These results were corroborated in a CMR study by Hombach et al. [25] of 110 MI patients followed for 7.5 months, 85% of whom were revascularized by PCI. Late gadolinium enhancement of the RV was seen in 17% of these patients, with RV wall motion abnormalities in 43%. RV infarction in this study was not associated with a worse outcome. On the other hand, RV infarction by LGE after MI was shown to be predictive of RV dilation at 6 month follow up [26]. In conclusion, RV involvement in acute MI can be reliably detected by CMR, but the prognostic significance of LGE of the right ventricle remains unclear based on the present results in the literature.

Microvascular obstruction

In about 20% to 50% of patients with an acute MI a “slow-flow” or “no-reflow” phenomenon in the infarcted myocardium can be observed, despite modern acute mechanical recanalization techniques (i.e. primary PCI and concomitant medical therapy with GP IIb/IIIa). This phenomenon is also called “microvascular obstruction” (MVO) and can be assessed by CMR using two separate techniques. Early MVO is visualized early after first pass perfusion as hypoenhancement persisting longer than 1 to 2 min after contrast injection. The second technique uses LGE images 10 to 15 min after contrast injection, and MVO appears as an area of hypoenhancement encompassed within the core of hyper-enhanced infarcted myocardium, often extending from the subendocardium (Fig. 2). This latter method in the literature is sometimes termed late MO or persistent MO (PMO) [25]. There is still no definite consensus on whether early or late MO should be the preferred technique for evaluating MO. Wu et al. [27] found that the spatial extent of early MO correlates well with histology and myocardial contrast echo. Orn et al. [28] reported that early MO was predictive of LV function and remodeling at one year follow up, and was a better marker of 1 year infarct size than late MO. Wu et al. [29] found that

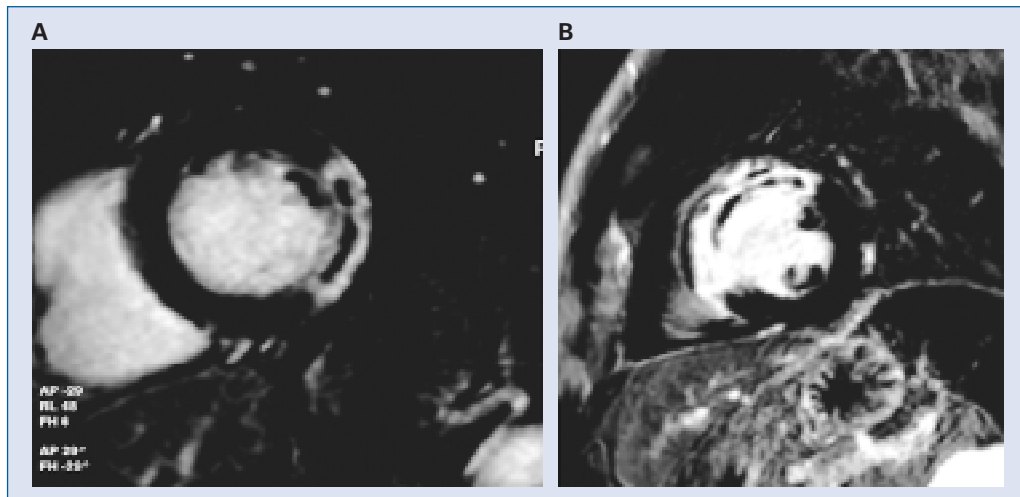


Figure 2. Microvascular obstruction in a patient with posterolateral myocardial infarction from occlusion of the left circumflex artery (A), and in a patient with anteroseptal infarction (B) in the short axis view. Note the area of hypoenhancement encompassed within the core of hyperenhanced infarcted myocardium (A), and the hypoenhanced area of microvascular obstruction extending from the subendocardium (B).

early MO predicts infarct transmuralty, LV function and remodeling after 16 months, and is also a powerful independent prognostic indicator of MACE even after normalization for infarct size. In contrast, Nijveldt et al. [30] found in a study of 60 MI patients undergoing CMR at 5 days and 4 months that both early and late MO correlated with poor ST segment resolution. None of both methods correlated well with angiographic parameters such as angiographic blush grade or TIMI flow grade.

On the other hand late MO (PMO) correlates with invasive coronary flow velocity measures of MVO [31], and several studies have shown that PMO better predicted follow up LV function and remodeling than early MO, and provides additional prognostic information beyond that of infarct size or transmuralty. For example, Nijveldt et al. [32] studied 63 acute MI patients who received PCI and optimal medical management and followed them by CMR with assessment of early, mid, and late MO 4 to 7 days after MI and follow up CMR at 4 months. They reported that PMO was a better predictor of follow up LVEF, LVEDV and LVESV than early MO. In a study of 110 MI patients, Hombach et al. [25] found that PMO was an independent predictor of LV remodeling, and independently indicated an increase in cardiac events at 8 month follow up and gave additional prognostic information than infarct size alone. This was corroborated in a CMR study of Bruder et al. [33] on 67 STEMI patients followed for 14 months, showing that late MO was a better predictor of adverse cardiac events than infarct size or baseline ejection fraction. The timing of the CMR

examination after MI is also important when assessing MO using first pass perfusion. Studies demonstrate that early MO is more often seen when CMR is performed 2 days after MI than on days 7 or 9, but is not predictive of long term functional or clinical outcomes until the later time points [28, 29]. It can be concluded that if MO either early or late is present in a patient in the acute phase of MI this patient has a worse prognosis compared with a patient without MO. Thus, the detection of MO after MI may force to adhere strictly to modern standard medical therapy, and to monitor patients with presence of MO post-MI closer and more strictly than those without MO.

Ischemic cardiomyopathy

Patients with ischemic cardiomyopathy (ICM) have a limited prognosis, and in addition to depressed LV function and electrocardiographic predictors of potentially life-threatening ventricular tachyarrhythmias or SCD CMR may provide independent prognostic information in this patient group. Yokota et al. [17] studied 86 patients with ICM with a mean LVEF of $26 \pm 12\%$ using delayed-enhancement CMR with a follow-up of 20 ± 16 months. Patients with cardiovascular events had significantly larger scar volume and scar percentage of the myocardium than patients without events. Quantification of scar volume was superior to LVEDV, LVESV, and LVEF in predicting future cardiovascular events in ICM patients. Similar results were reported by Kwon et al. [34] in 349 patients with ICM and severely reduced LVEF. Mean scar per-

centage and transmural score were significantly higher in patients with events *vs* those without events. Kim et al. [35] using CMR imaging with delayed enhancement (LGE) were able to show in a systematic study in 185 patients with suspected CAD without clinical or electrocardiography (ECG) evidence of prior infarction that unrecognized non-Q-wave MI by LGE was associated with an 11-fold higher mortality risk independent of LVEF.

Non-ischemic cardiomyopathies

Idiopathic dilated cardiomyopathy (IDC)

CMR may also provide additional prognostic information in patients with dilated cardiomyopathy and/or HF by both left and right ventricular function and LGE. Nazarian et al. [36] reported in 26 patients with non-ICM predominance of scar localization and distribution, that a transmural extent of LGE of 26% to 75% of wall thickness was significantly predictive of inducible VT on EPS and remained predictive after adjustment for LVEF (odds ratio 9.125, $p = 0.020$). Thus, scar distribution may identify the substrate for inducible VT and may identify high-risk patients with non-ICM. Similar results were reported by Yokokawa et al. [37] in 47 patients with IDC and advanced HF.

In a total of 101 consecutive patients with non-ICM and HF Asomull et al. [38] found midwall fibrosis by LGE-CMR in 35%, which was associated with a higher rate of the predefined combined endpoint (all-cause death and hospitalization for a cardiovascular event, HR 3.4, $p = 0.01$) and for prediction of secondary endpoint (SCD and/or sustained VT, HR 5.2, $p = 0.03$). Wu et al. [39] found LGE in 27 (42%) patients in a series of 65 patients with non-ICM and ejection fraction $\leq 35\%$ planned for an ICD implantation, and 12 of LGE positive patients (44%) had an index composite outcome event (cardiac death, appropriate ICD shock, and hospitalization for decompensated HF) compared with only 8% of LGE negative patients ($p < 0.001$). After adjustment for LV volume index and functional class, patients with LGE had an 8-fold higher risk of experiencing the primary endpoint (HR 8.2, $p = 0.002$).

Hombach et al. [40] studied 141 consecutive IDC patients by clinical, conventional ECG, CMR functional parameters, and LGE-CMR. In 63 patients myocardial biopsy was performed, and chronic inflammation was excluded by molecular analysis. Non-CAD type mid-wall or subepicardial LGE was present in 36 (26%) patients. After median follow-up of 1339 days including 483 patient years 25 (18%) patients experienced the primary endpoint

(cardiac death 16, sudden death 3 plus 6 with an ICD shock). Kaplan-Meier analysis showed QRS duration > 110 ms ($p = 0.010$), presence of LGE ($p = 0.037$), and diabetes mellitus ($p < 0.001$) as significant predictors of a worse outcome. Multivariable analysis revealed cardiac index ($p < 0.001$) and increased RVEDVI ($p = 0.006$) derived from CMR, the presence of diabetes mellitus ($p = 0.006$), and QRS > 110 ms ($p = 0.0045$) as significant predictors of the primary endpoint. In conclusion, it was stressed that prognostically relevant hemodynamic (cardiac index, RVEDVI) and ECG parameters (QRS duration) can be derived from two non-invasive techniques with highest precision and without complicated analysis techniques.

Hypertrophic cardiomyopathy

Recent publications have shown that in patients with hypertrophic cardiomyopathy (HCM) the extent of fibrosis or scar as documented by LGE (Fig. 3) is associated with the prevalence of ventricular (tachy-) arrhythmias and with a worse prognosis. Adabag et al. [41] reported in a cohort of 177 HCM patients that patients with LGE had a greater number of PVCs, couplets, and non-sustained VT (nsTV) episodes than those without LGE; LGE was an independent predictor of nsVT. Suk et al. [42] reported in 25 HCM patients that VT occurred in 32% of patients and this was associated with both increased scar mass (by LGE) and wall thickness, compared with non-VT patients. Fluechter et al. [43] found in a retrospective study on 76 consecutive HCM patients that 12 of 38 high-risk patients (defined by 1 or more of the conventional risk factors) had inducible VT on EPS, and the relative LV mass with LGE was significantly higher in patients with inducible VT compared with those without (22% *vs* 10%, $p = 0.03$). Leonardi et al. [44] reported similar results. In a cohort of 108 consecutive HCM patients they found that compared to patients without arrhythmias patients with VT/VF ($n = 33$) had a higher LGE score, and the LGE score was the only independent predictor of VT/VF in multivariable analysis; LGE score, maximal LV wall thickness, and LV mass index were significantly greater among patients at risk for SCD ($n = 51$) compared with the remaining 57 patients at low risk. The authors concluded that in HCM patients several CMR parameters are associated with risk for SCD, and a semi-quantitative LGE score is a significant predictor of both clinical VT/VF and of risk for SCD, and may contribute to risk assessment in borderline or controversial cases.

O'Hanlon et al. [45] assessed the presence and amount of myocardial fibrosis by LGE in 217 con-

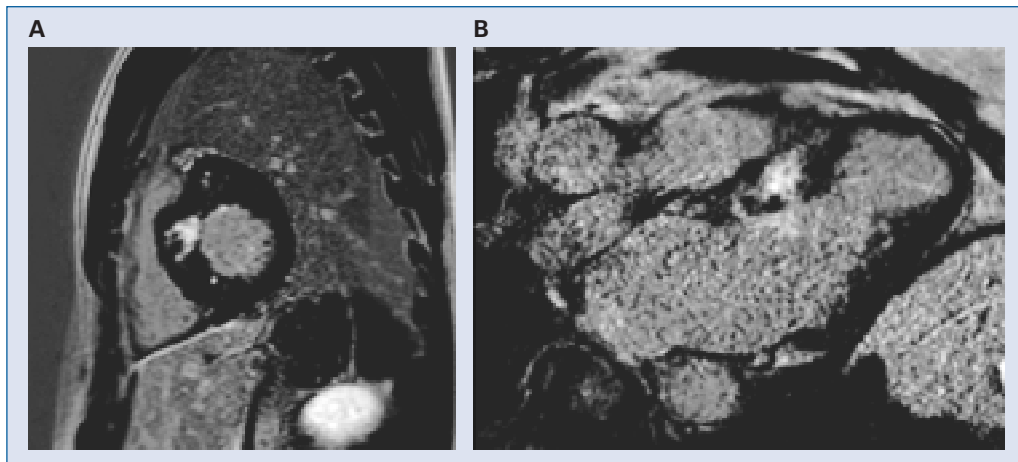


Figure 3. Myocardial fibrosis (scar) in a patient with hypertrophic cardiomyopathy in a short axis (A) and four-chamber view (B). Note the hyperenhanced area within the interventricular septum in the region of greatest thickness of the septal wall.

secutive HCM patients and prospectively followed them over 3.1 ± 1.7 years. Thirty-four of the 136 patients (25%) in the fibrosis group but only 6 of 81 without fibrosis reached the combined primary endpoint of cardiovascular death, unexplained cardiovascular admission, sustained VT or VF, or appropriate ICD shock (HR 3.4, $p = 0.006$). The extent of fibrosis and nonsustained VT were univariable predictors of arrhythmic endpoints (sustained VT or VF), appropriate ICD discharge, or SCD (HR 1.3, $p = 0.04$). In multivariable analysis only nonsustained VT remained an independent predictor of arrhythmic endpoints, whereas the extent of fibrosis did not. Bruder et al. [46] enrolled 243 HCM patients, who were investigated by a comprehensive CMR protocol including LGE-CMR, and follow-up was completed in 220 patients after mean 1,090 days for all-cause and cardiac mortality. The presence of scar visualized by CMR yielded an odds ratio of 5.47 for all-cause mortality and 8.01 for cardiac mortality. This seemed to be superior to classical clinical risk factors, because in their dataset the presence of 2 risk factors yielded an odds ratio of 3.86 for all-cause mortality and of 2.20 for cardiac mortality. Multivariable analysis also revealed the presence of LGE as a good independent predictor of death in HCM patients. Thus, it may be concluded that the presence and extent of myocardial fibrosis as detected by LGE on CMR imaging heralds an adverse outcome in HCM patients and may be superior to the known classical clinical risk factors. CMR imaging may offer new access to a better risk stratification not only in patients with advanced, but also with borderline disease states of HCM.

Patients with chronic heart failure

Cardiac magnetic resonance derived dyssynchrony in heart failure

CMR imaging is useful in patients with chronic HF not only for assessing ventricular dysfunction, myocardial scar, and prognosis, but also for detecting and quantifying the amount of inter- and particularly intraventricular dyssynchrony and its prognostic impact. Chalil et al. [47] were able to show that the CMR derived tissue synchronization index (TSI) as a parameter of LV dyssynchrony had prognostic impact in 77 patients with HF undergoing cardiac resynchronization therapy (CRT). Kaplan-Meier curves showed that patients with a TSI ≥ 110 ms had a worse outcome regarding survival from hospitalization for MACE, hospitalization for HF, survival from death from any cause, and survival from cardiovascular death. These results are puzzling as on one hand LV dyssynchrony is a predictor of increased mortality in patients with HF, on the other hand CRT should improve outcome and clinical symptoms in patients with HF. This discrepancy may be explained by the study of Leyva et al. [48], who developed a triple index to predict survival after CRT. Out of 16 prognostic risk factors of HF patients, Cox proportional hazards analyses revealed dyssynchrony, postero-lateral scar and elevated creatinine as independent predictors of cardiovascular mortality (DSC index). Compared to patients with a DSC < 3 , cardiovascular mortality in patients in the intermediate DSC index (3–5; HR 11.1, $p = 0.0003$) and high DSC index group (≥ 5 ; HR 30.5, $p < 0.0001$) was significantly higher. In

this study hazard ratio of postero-lateral scar location was 12.2 compared to CMR-TSI with a hazard ratio of 1.01 and creatinine with a hazard ratio of 1.01, and thus, postero-lateral scar was the most powerful prognostic parameter within this triple DSC index. In conclusion, CMR assessment of intraventricular dyssynchrony and detection of myocardial scar by CMR imaging appear to be promising new parameters of risk stratification in patients with chronic HF and significant dyssynchrony. However, further prognostic studies have to be performed to validate these preliminary results.

Conclusions

CMR is an essential technique for characterization of patients with cardiovascular disease. It allows accurate assessment and quantification of many parameters such as ventricular function/dysfunction, myocardial ischemia, and myocardial damage/scar with highest precision and without radiation exposure. Several CMR-derived parameters provide significant prognostic information in patients with CAD, with cardiomyopathies, and with chronic HF. Myocardial ischemia, either detected by dobutamine stress CMR or by CMR first pass perfusion, is a strong predictor of an adverse outcome in patients with suspected or known CAD. Microvascular obstruction in patients with acute MI is a new and independent parameter of negative LV remodeling and a worse prognosis. Moreover, demonstration of myocardial scar in patients with known or suspected CAD and unrecognized MI by delayed enhancement-CMR (LGE) heralds a negative outcome. Fibrosis or scar in patients with dilated as well as with hypertrophic cardiomyopathy is a strong predictor of both life-threatening ventricular tachyarrhythmias and prognosis. Preliminary data indicate that CMR imaging may not only improve the assessment of inter- and particularly intraventricular dyssynchrony in patients with severe LV dysfunction to be selected for CRT, but also may aid in selecting CRT responders and provide prognostic information by detecting myocardial scars using the LGE technique.

Acknowledgements

The authors do not report any conflict of interest regarding this work.

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